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HOST ADAPTATION IN *PHYTOPHTHORA INFESTANS*: A REVIEW FROM A POPULATION BIOLOGY PERSPECTIVE

ABSTRACT

Phytophthora infestans behaves in natural and agricultural ecosystems as a biotrophic pathogen, although it can be cultured on artificial media. Pathogenicity and host adaptation are therefore essential traits to understand its biology and to come up with durable, efficient management of late blight. The present review focuses on adaptation to host species and host cultivars, and to both qualitative and quantitative types of resistance. It also discusses some of the patterns and population mechanisms involved in this adaptation, such as selection, genetic drift and migration. This highlights the need for an in–depth analysis of each local situation to accurately describe and understand the mechanisms responsible for observed population displacements.

Key words: aggressiveness, host resistance, late blight, non–host resistance, specificity, virulence

INTRODUCTION

Phytophthora infestans, which causes late blight of Solanaceae, is often described as a highly variable pathogen. When applied to pathogenicity traits, this variability is regarded as the major cause for the breakdown of cultivar resistances used to control the disease. Consequently, over the last seventy years, a large corpus of research has dealt with the characterization of pathogenicity in *P. infestans* isolates or populations world–wide. Its primary aim has been to make a better use of the resistance sources available to potato breeders and growers, by choosing them according to the pathogenicity features prevalent in local *P. infestans* populations.

Different types of pathogenicity features must be considered in species which, like *P. infestans*, can interact with their hosts at various levels of specificity. Host specificity at the genus/species or at the cultivar

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level allow to define the host range and physiological races of the patho–gen, while the quantitative assessment of the disease induced in sus–ceptible hosts is a major, but completely different component of pathogenicity (see for a discussion Andrivon 1993). We therefore inves–tigated the extent of pathogenic variation present in "old" and "new" populations of *P. infestans* (*sensu* Spielman *et al.* 1991) at the different specificity levels, through a review of some of the published data.

The introduction, beginning in the mid–to late 1970s, of a number of pathogen genotypes into Europe, and subsequently to most potato crop–ping areas in the world (Spielman *et al.* 1991, Fry *et al.* 1992, 1993, Goodwin *et al.* 1994), led to a rapid displacement of local isolates (called "old populations" by Spielman *et al.* 1991) by newly introduced ones. The extreme speed of this population shifts, and their world–wide occur–rence despite the variation in the characteristics of immigrant geno–types, led to further concern about the extent and sources of variability present in *P. infestans*. Three main factors can be advocated to account for the evolutionary success of the immigrant strains over previously established populations: increased pathogenicity, in terms of host range and of aggressiveness to each major host (Fry *et al.* 1992, Day and Shattock 1997, Kato *et al.* 1997, Miller *et al.* 1998); increased fitness, through higher adaptability (Fry *et al.* 1992), a wider range of ecological competence (e.g. Mizubuti and Fry 1998), or better survival as a conse–quence of the possible occurrence of the sexual stage (Fry *et al.* 1989, 1992, Andrivon 1995); and higher gene flow, either because of migra–tions or of sexual reproduction (Goodwin *et al.* 1998).

Although much work has been dedicated to the identification of popu–lation structures of *P. infestans* in North America (e.g. Fry *et al.* 1992, Goodwin *et al.* 1992, 1994, 1998), Europe (e.g. Fry *et al.* 1991, Andrivon *et al.* 1994, Drenth *et al.* 1994, Day and Shattock 1997, Lebreton *et al.* 1998, Carlisle *et al.* 2001, Cooke *et al.* 2003) and other parts of the world (e.g. Goodwin *et al.* 1994, Koh *et al.* 1994, Forbes *et al.* 1997, Reis *et al.* 2003), we are still a long way from a firm assessment of the implications of the three above–mentioned mechanisms in the current set–up and recent evolution of populations of the fungus in the different geograph–ical and ecological situations where the pathogen prospers. One of the major reasons for this is the fact that most recent population research has focused on selectively neutral markers, but much less on pathoge–nicity features considered from a population biology perspective. Fur–thermore, these studies have shown that the characteristics of the groups of genotypes of the pathogen present in the various parts of the world, while different from those of the original set of clones (US–1 and related sub–clones; Goodwin *et al.* 1994, 1998), are also markedly dis–tinct from one another. Populations present in North America are usu–ally quite simple, with one genotype dominating at each location in a given year (Goodwin *et al.* 1998), whereas the setup of European pop–ulations is often much more complex, with a number of genotypes coex–

isting at any single time (Drenth *et al.* 1993, Lebreton and Andrivon 1998, Lebreton *et al.* 1998, Cooke *et al.* 2003). Finally, while late blight resurgence is a fairly recent phenomenon in North America (Fry and Goodwin 1997), the disease has been prevalent in Europe and in several other parts of the world for the past 150 years, with years without severe outbreaks the exception more than the rule (e.g. Large 1953, 1956, Schöber 1987). This much greater prevalence of the disease in Europe is directly translated in terms of pesticides used to control late blight. While growers in the Columbia basin of Washington and Oregon would spray an average of 2.5 times against *P. infestans* in a year of low incidence of the disease (such as 1994) and up to 10 times in a severe blight year, such as 1995 (Johnson *et al.* 1997), potato crops in western Europe would typically be sprayed an average of 6–8 times against blight, and up to 15–20 times in severe blight years and susceptible cultivars (Schepers 2003). All together, the large discrepancies in population composition and epidemic potential make generalizations from one population to others problematic.

The aims of this paper are to review the information currently available about patterns of adaptation to hosts in populations of *P. infestans*, and about the evolutionary mechanisms related to these patterns. The goal is to provide keys to explain – and, if possible, predict – future changes in pathogenicity features at the population level, which are needed for a better and more durable management of genetic resources for resistance to late blight. Because most of the data available concern either North American or European populations, the review is focused on these two groups of populations.

ADAPTATION TO HOST GENUS/SPECIES

P. infestans is known to be pathogenic to at least forty species of *Solanaceae* (Turkensteen 1978). Host specificity is of pathological, but also of evolutionary significance, because the possibility for infecting more than one host determines to a large extent the availability of "green bridges" during the pathogen's life cycle. These are critical in maximizing survival opportunities in species with very low saprophytic abilities, such as *P. infestans* (see Andrivon 1995 for a review), and probably condition the extent of gene flow between isolates. Host specificity may also have led to a speciation event between *P. infestans* and *P. mirabilis*, two species giving rise to fertile hybrids (Goodwin and Fry 1994), morphologically indistinguishable from one another (Galindo and Hohl 1985), but with mutually exclusive host ranges. This separation of host ranges explains the reproductive isolation of *P. infestans* and *P. mirabilis* in nature, which in turn probably led to the accumulation of genetic differences detectable in current collections (Goodwin, personal communication). *P. mirabilis* was thus considered either as a variety (Servin 1958) or a *forma specialis* of *P. infestans* (Möller *et al.* 1993), or

regarded as a valid species (Galindo and Hohl 1985, Goodwin and Fry 1994, Goodwin and others, personal communication). Similar speciation patterns have been described in recent years in South America (Adler *et al.* 2002). They involve sympatric wild and/or cultivated hosts, which points to a selective advantage to host specialisation in habitats where a number of potential hosts are present (Lapchin 2002). However, this general trend towards specialisation (i.e. restriction of host range) is sometimes reverted, as shown by the recent discovery in the Netherlands of isolates overcoming the resistance of *Solanum nigrum*, until then regarded as a non–host for *P. infestans* (Flier *et al.* 2003a).

The specialisation process does not always lead to species individualisation. Many authors observed isolates more specifically adapted to either potato (*Solanum tuberosum*) or tomato (*Lycopersicum esculentum*), both in "old" (e.g. Berg 1926, Small 1938) and in "new" populations (Legard *et al.* 1995, Lebreton *et al.* 1998, 1999, Oyarzun *et al.* 1998, Reis *et al.* 2003), but specificity was never restrictive enough to warrant the "forma specialis" denomination. The initial adaptation of any isolate can be reverted by repeated passages through the other host. Although this process is unlikely to be of significance in agricultural practice, since isolates initially adapted to one host would be out–competed on the other host, it shows a high level of genetic plasticity in *P. infestans* regarding pathogenicity determinants.

Two isolates of *P. infestans* collected from tomato in southwestern Europe and characterized by isozyme alleles and mitochondrial haplotypes as "old" populations showed different genetic fingerprints (Lebreton and Andrivon 1998), which suggests that the higher genetic diversity of the pathogen observed today on tomato in this area might have existed before the introduction of "new" genotypes. In many "new" populations, differences in the frequency distribution of genetic features other than pathogenicity (such as mating types, mitochondrial DNA haplotypes, isozyme alleles, or nuclear DNA fingerprints) between collections of isolates made on potato and on tomato is the rule rather than the exception (Lebreton and Andrivon 1998, Oyarzun *et al.* 1998). Interestingly, isolates present on tomato generally belong to simpler races than those collected on potato (Deahl *et al.* 1993, Lebreton and Andrivon 1998). However, there is no consistent association on a world–wide basis between genetic markers and adaptation to one or the other host. For instance, A2 isolates are more frequently found on tomato than on potato in France (Lebreton and Andrivon 1998), but are restricted to potato in several South American countries, such as Brazil (Brommonschenkel 1988, Reis *et al.* 2003).

ADAPTATION TO HOST CULTIVARS

Virulence to *R*–genes

The existence of race–specific resistance genes in *Solanum tuberosum* and of matching physiological races in *P. infestans* have been recognised since the 1940s and extensively investigated since the early 1950s (see Wastie 1991 for a review). The initial 11 *R*–genes described originate from the Mexican species *Solanum demissum*, but similar genes exist in many other tuber–bearing *Solanum* species (Hawkes 1958, Rivera–Peña 1990, Tooley 1990). Some of these species, including *S. bulbocastanum*, have been considered as promising sources of highly efficient *R*–genes, and the corresponding *R*–genes have been mapped and/or cloned (Song *et al.* 2003, Van der Vossen *et al.* 2003). However, both the RB / *Rpi–blb1* genes recently cloned from *S. bulbocastanum* and *R1* from *S. demissum* (Ballvora *et al.* 2002) are genetically similar to many known – and defeated – *R*–genes which belong to the NBS–LRR (nucleotide binding site–leucine rich repeat) class. This suggests that all can be defeated rapidly by new races of *P. infestans* if deployed in commercial cultivars.

Because deployment strategies of *R*–genes are an obvious factor shaping the changes in race frequencies, complex races were selected in "old" populations wherever popular potato cultivars carried combinations of *R*–genes, such as in Great Britain (Malcolmson 1969, Shattock *et al.* 1977). In some instances, the introduction of "new" populations led to a marked increase in the complexity of races (Deahl *et al.* 1993, Drenth *et al.* 1994). However, a comparative analysis of race structure characteristics in "old" and "new" populations of *P. infestans* collected world–wide and surveyed with the same set of differential clones showed no consistent trend towards an increase in virulence complexity or virulence diversity in the most recent populations (Andrivon 1994a).

Part of the discrepancy between regions might be due to the fact that most of the virulences present in the isolates now recovered outside Mexico do not reflect local selection, but selection in Mexico prior to migration. A number of virulences present nowadays in Europe (such as virulences to *R7*, which is very common, and virulences to *R5*, *R6* and *R8*, which are less frequent) match resistance genes which have never been used in European commercial cultivars. These virulences can be regarded as "fossil" traits, once selected for in central Mexico where the matching *R*–genes exist in nature, and maintained in isolates after their migration, as no genetic mechanism existed to remove them. Accumulation of "fossil virulences" has been postulated in rust pathogens of cereals, although in this case the original selection was exerted locally (Andrivon and de Vallavieille–Pope 1995). From an evolutionary perspective, this implies the absence of a fitness penalty associated with unnecessary virulences, making most virulences behave as selectively neutral markers when resistance genes are not used, which is the case

now in several parts of Europe (Andrivon *et al.* 1994) and in North America (Fry and Goodwin 1997). Indeed, there seems to be no correlation between fitness and race complexity in "new" European populations (Schöber and Turkensteen 1992, Pilet 2003). It is therefore difficult to attribute the success of immigrant genotypes in displacing former clones to their higher virulence complexity, because (i) not all migrant genotypes belonged to races more complex than the clones they displaced, and (ii) no immediately identifiable fitness benefit can be associated with this increase, when it occurred.

Aggressiveness

P. infestans isolates have been shown to vary largely in their aggressiveness towards potato cultivars. This variation is not related to physiological races (e.g. Jeffrey *et al.* 1962, Denward 1967, Caten 1974), and can be detected both in controlled conditions and in the field (e.g. Tooley and Fry 1985, Tooley *et al.* 1986, Day and Shattock 1997). Aggressiveness can decrease during repeated subculturing on artificial media, but it can be restored *via* inoculation of living plant material (Jeffrey *et al.* 1962, Jinks and Grindle 1963). In several experiments, aggressiveness remained stable over successive transfers to potato plants (Caten 1974). Furthermore, specific components of aggressiveness have been detected repeatedly (e.g. de Bruyn 1947, Jeffrey *et al.* 1962, Jinks and Grindle 1963, Caten 1974, Carlisle *et al.* 2002, Corbière *et al.* 2002), reflecting the fact that each isolate usually grows better on the variety it was recovered from than on other varieties with the same *R*–genes. However, recent work with populations sampled at the same locations on cultivars free of *R*–genes, but expressing different levels of partial resistance, consistently revealed higher average agressiveness towards both susceptible and partially resistant cultivars in populations sampled from susceptible rather than from partially resistant hosts (Pilet 2003).

Evidence is accumulating to show a higher aggressiveness in isolates belonging to "new" populations than in their "older" counterparts (Day and Shattock 1997, Kato *et al.* 1997, Flier and Turkensteen 1999). However, because of methodological limitations, these data should be interpreted with caution. Aggressiveness among isolates is a composite of many traits, and is thus difficult to measure accurately. Variation is characteristic of most of aggressiveness components (e.g. latent period, infection efficiency, sporulation), but is not always directly correlated with disease progress in the field (Spielman *et al.* 1992). Therefore, comparisons made on single components might not accurately describe actual differences in global aggressiveness between isolates. Furthermore, "old" and "new" isolates being compared may not have been subjected to the same number of transfers on artificial media. Finally, the extensive variation present among "new" isolates of *P. infestans* sometimes overlaps the range of differences between "old" and "new" isolates (Schepers 1998). Indeed, US–1 remains a very destructive pathogen in

some tropical highland areas, such as Ecuador (Oyarzun *et al.* 1998), or in temperate conditions in South Africa (McLeod *et al.* 1998).

While blight has undoubtedly gained importance in North America over the last decade (Fry and Goodwin 1997), no consistent trend towards a worsening of the disease appears from long–term surveys of blight incidence and severity in Europe. This discrepancy might be explained by the fact that, contrary to what happens in most parts of North America, blight is observed every year in western Europe, where the pathogen usually finds very favourable climatic conditions (mild, humid climate). However, it is noteworthy that not all years since the introduction of "new" strains have been severe blight years in Europe. For instance, Hardwick and Turner (1996) reported that blight incidence and severity were low in England and Wales in the early the 1990s, although only "new" genotypes were present (Day and Shattock 1997). A similar situation occurred in France in 1989 and 1990 (Duvauchelle 1993). The use of fungicides for blight control in Europe was 15–40% higher in 1997 (severe blight) than in 1996 (moderate blight) (Schepers 1998), although there was no evidence of major changes in the pathogen population between the two years. Overall, the incidence and severity of blight seems to be much better correlated (at least in Europe) with the climatic conditions prevalent in a given year than with the type of populations present. Indeed, disease forecasting systems, despite being based on experimental data relating to "old" isolates, still work pretty accurately, the major factor conditioning their performance being the quality of input meteorological data (Hansen 1998).

PATHOGENICITY, ADAPTATION, AND FITNESS

Genetic diversity and adaptability

"New" populations of the pathogen are constituted of a larger number of genotypes, and are usually genetically more diverse (at least for neutral markers) than were "old" populations (Goodwin *et al.* 1994). However, it is not known how this higher diversity is translated in terms of adaptability. As discussed above, there is no unequivocal evidence that new populations are always more pathogenic than their former counterparts. While there is some evidence for adaptation to a broader range of climates in "new" lineages (Mizubuti and Fry 1998), these data are not unequivocal. For instance, over a range of temperatures, US–1 sporulated and germinated on average more abundantly than US–7 and US–8 in controlled experiments (Mizubuti and Fry 1998), although these authors concluded that new genotypes were fitter than members of the US–1 lineage from measurements of incubation period and/or lesion size. Flier *et al.* (2003b) also showed that US–1 was more pathogenic than US–8 on some European potato cultivars, and that "new" European isolates exhibited a wide range of variability concerning the

pathogenicity to these cultivars. Finally, recent work done in France and Ecuador (Pilet 2003) has revealed the existence of extensive variability for aggressiveness in isolates belonging to the same AFLP (amplified fragment length polymorphism) pattern.

Survival

The formation of long lasting oospores is an obvious advantage for survival in an organism with saprophytic capacities as low as those of *P. infestans* (Fry *et al.* 1989, Andrivon 1995). This advantage is even greater in parts of the world with extreme climates (either hot summers, such as in Mexico, or very cold winters, such as in Canada, Poland or Scandinavia). Not surprisingly, the most convincing evidence for involvement of sexual spores in the epidemiology of late blight comes just from such areas (Sujkowski *et al.* 1994, Goodwin *et al.* 1995, Andersson *et al.* 1998). In areas with less contrasted seasons, the opportunities for asexual survival of the pathogen in infected tubers (volunteers, refuse piles) are probably high enough to ensure that the largest part of the primary inoculum is of asexual origin, even though oospores might be present. The difficulty is then to detect the fraction of the inoculum derived from oospores in the global populations of isolates. Indeed, refuse piles remain the major primary infection sources for commercial potato crops in Flevoland, the Netherlands, despite the occurrence in the immediate vicinity of allotment gardens where both mating types and oospores are present (Zwankhuizen *et al.* 1998).

A major consequence of the poor survival of *P. infestans* outside its host and of the limited number of situations where oospores play a significant epidemiological part to date is the structural lability noticed in many local populations of the fungus over a series of years (Drenth *et al.* 1993, Andrivon 1994a, Goodwin *et al.* 1998, Lebreton *et al.* 1998, Zwankhuizen *et al.* 1998). This makes impossible to predict the population structure in a given year from the knowledge of its structure in former years (Fry and Goodwin 1997, Goodwin *et al.* 1998, Lebreton *et al.* 1998), at the same time indicating a major role of founder effects in shaping population structures over time (Fry *et al.* 1992, Andrivon 1994a, Fry and Goodwin 1997, Lebreton *et al.* 1998). These observations strongly suggest that pathogenic fitness is one, but probably not the main, of the components of global fitness in *P. infestans* genotypes, and that chance and survival ability are more critical than pathogenicity in long term evolutionary success. In this respect, the observation that in Brittany, late blight epidemics are often started by very simple races, usually found mainly on tomatoes and present late in the season but less pathogenic to potato, and that the major, complex potato races only develop later (Andrivon 1994b), is significant, because it illustrates the fact that the highest pathogenicity is not a prerequisite for persistence over time, and might even hinder it. This resembles wild host–parasite

systems, where balanced pathogenicity commonly develops (Bull 1994, Lapchin 2002).

SOME CONCLUSIONS

This review confirms that extensive variation exists in both "old" and "new" populations of *P. infestans* for all components of pathogenicity, but also outlines the fact that pathogenicity is only one element of the story when attempting to understand population structures in this pathogen. Although the evidence exists that "new" populations might be on average more virulent (i.e. include more complex races) and more aggressive than their former counterparts, this trend does not refer equally to all situations, and exceptions can be easily found in the available data. Furthermore, the large influence of conditions prevalent during epidemic development, but even more during survival, result in strongly pronounced drift effects, which means that the actual fitness of a genotype (i.e. its contribution to the next generation or to the population in the following year), is only partially dependent on the pathogenicity of this genotype. This has two major consequences for explaining and predicting changes in population structures of the late blight pathogen: first, the extent to which the changes of pathogenicity affect population structures cannot be evaluated on a general and uniform basis, but needs to be assessed for a particular situation; second, it is very difficult to predict population changes based only on pathogenicity features. The metapopulation structure of *P. infestans* populations on local and regional scales (Andrivon *et al.* 1994, Lebreton and Andrivon 1998) make this prediction even more difficult, because of the random nature of founding events and of the poor quantitative assessment of migration rates between patches. Until comprehensive models, including random (or more likely frequency–dependent) extinction events during survival stages, possibilities for switching hosts, and pathogenic fitness on each host, are developed, predicting population structures of *P. infestans* will remain largely a guess. Further work is therefore needed to better appreciate and quantify the amount of gene flow, the modalities of extinction and the impact of long–lasting oospores on survival and population structures in the long term, which are the absolute prerequisites for the development of such comprehensive models.

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