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Erosion of quantitative host resistance in the apple × *Venturia inaequalis* pathosystem

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ABSTRACT

Theoretical approaches predict that host quantitative resistance selects for pathogens with a high level of pathogenicity, leading to erosion of the resistance. This process of erosion has, however, rarely been experimentally demonstrated. To investigate the erosion of apple quantitative resistance to scab disease, we surveyed scab incidence over time in a network of three orchards planted with susceptible and quantitatively resistant apple genotypes. We sampled *Venturia inaequalis* isolates from two of these orchards at the beginning of the experiment and we tested their quantitative components of pathogenicity (i.e., global disease severity, lesion density, lesion size, latent period) under controlled conditions. The disease severity produced by the isolates on the quantitatively resistant apple genotypes differed between the sites. Our study showed that quantitative resistance may be subject to erosion and even complete breakdown, depending on the site. We observed this evolution over time for apple genotypes that combine two broad-spectrum scab resistance QTLs, F11 and F17, showing a significant synergic effect of this combination in favour of resistance (i.e., favourable epistatic effect). We showed that isolates sampled in the orchard where the resistance was inefficient presented a similar level of pathogenicity on both apple genotypes with quantitative resistance and susceptible genotypes. As a consequence, our results revealed a case where the use of quantitative resistance may result in the emergence of a generalist pathogen population that has extended its pathogenicity range by performing similarly on susceptible and resistant genotypes. This emphasizes the need to develop quantitative resistances conducive to trade-offs within the pathogen populations concerned.

1. Introduction

Plant genetic resistance to pathogens offers an interesting alternative to disease control methods based on the use of pesticides, but lacks durability due to the rapid evolution of pathogen populations. Pathogen populations are able to adapt, frequently resulting in the breakdown of the resistance in the case of major resistance genes faced with the qualitative component of pathogenicity, i.e., the ability of a pathogen to infect a plant. Quantitative resistance based on quantitative trait loci (QTL) induces a partial reduction of pathogen development. Various mechanisms may underlie quantitative resistance, including basal defence, chemical warfare, defence signal transduction or weaker forms of major resistance genes (Poland et al., 2009), and quantitative resistance may either be specific to some isolates of a pathogen or be broad-spectrum (i.e., efficient against all isolates of a pathogen). Quantitative resistance affects quantitative components of pathogenicity (e.g., rate of infection, latent period, rate of sporulation), which are dependent on the host, the pathogen and the interaction between host and

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pathogen (Lannou, 2012). Quantitative resistance is frequently assumed to be more durable than qualitative resistance (Parlevliet, 2002), which has been experimentally shown, for example, in the case of wheat leaf rust on the cultivar, 'Apache' (Papaix et al., 2011). However, isolates sampled from partial resistant cultivars can induce a higher amount of disease on partial resistant cultivars than isolates sampled on susceptible ones, as observed for *Mycosphaerella graminicola* on wheat (Cowger and Mundt, 2002), *Phytophthora infestans* on potato (Flier et al., 2003; Andrivon et al., 2007; Montarry et al., 2008) and *Plasmopara viticola* on grapevine (Delmotte et al., 2013). Over several generations, quantitative resistance can select for isolates with an increased rate of infection, a shortened latent period or an increased size of lesions, as shown for *Blumeria graminis* f.sp. *hordei* on barley, *Puccinia recondita* f.sp. *tritici* on wheat, and *Cochliobolus heterostrophus* on maize (Kolmer and Leonard, 1986; Newton and McGurk, 1991; Lehman and Shaner, 1997, 2007), respectively. Experiments using isolates inoculated in mixtures on different partially resistant cultivars can result in a modification of the isolate frequencies according to the host (Zhan et al., 2002; Lê Van et al., 2013). Thus, directional selection might also occur in pathogen populations that infect cultivars with quantitative resistance but at a slower rate than in pathogen populations that infect cultivars carrying major resistance genes (Gould et al., 1991; Zhan et al., 2002; Sommerhalder et al., 2011). This is in agreement with simulation models (Gandon and Michalakis, 2000; McDonald and Linde, 2002), suggesting that quantitative resistance may select for isolates with a high level of pathogenicity due to within-host competition, which may lead to erosion of the resistance.

Even if differential efficiencies of quantitative resistance have been shown among sites (Andrivon et al., 2007) and among pathogen isolates (Talukder et al., 2004; Le Guen et al., 2007; Marcel et al., 2008; Delmotte et al., 2013), the erosion of quantitative resistance at one site over time has rarely been demonstrated (Mundt et al., 2002). It is indeed difficult to detect gradual changes in efficiencies of quantitative resistance (McDonald and Linde, 2002), since these efficiencies are influenced by environmental conditions (Young, 1996; Pariaud et al., 2009; Lannou, 2012). As for the management of cultivars in space and time, it is crucial to determine if the process of erosion induces higher, lower or similar levels of pathogenicity on susceptible plants. For wheat leaf rust (Lehman and Shaner, 1997) and potato late blight (Andrivon et al., 2007), isolates that were well adapted to a quantitative resistance presented a reduced level of pathogenicity on susceptible hosts or other quantitatively resistant hosts. In such cases, the pathogen population is composed of several specialised isolates (i.e., 'specialists'). Each specialist has a higher multiplication rate on its host of origin (i.e., specific interactions between host and pathogen). In contrast, in the case of a broad-spectrum quantitative resistance with no specific interaction between host and pathogen, theoretical modelling (Gandon and Michalakis, 2000) predicted that such resistance selects for isolates able to produce a higher level of disease, i.e., the isolates that are well adapted to the susceptible hosts are also well adapted to the quantitatively resistant hosts. In such a case, the pathogen population would consist of a 'generalist' with a high multiplication rate on all hosts, but this has not yet been adequately experimentally documented.

In our study, we investigated the erosion of apple quantitative resistance to scab caused by the fungus *Venturia inaequalis*, which has a high evolutionary potential (Gladieux et al., 2008; Bus et al., 2011; Lê Van et al., 2012). Many quantitative resistance factors to scab have been identified in apple (Durel et al., 2003; Liebhard et al., 2003; Calenge et al., 2004; Soufflet-Freslon et al., 2008), either broad-spectrum or specific. Our aims were (1) to compare quantitative components of pathogenicity of *V. inaequalis* populations towards two broad-spectrum quantitative resistance factors (alone or in combination) at different sites, (2) to test the hypothesis that the isolates with a high level of pathogenicity on quantitative resistance also have a high level of pathogenicity on susceptible hosts, and (3) to compare the erosion of the quantitative resistances in three sites over an 8-year period.

2. Materials and methods

2.1. Orchard network

We used progenies of the cross between cv. Prima and cv. Fiesta (referred to as the "J progeny") produced by the former CPRO-DLO, which is now part of Wageningen-UR Plant Breeding, The Netherlands (Maliepaard et al., 1998). This progeny segregates for the two major genes, *Rvi1* (=*Vg*) and *Rvi6* (=*Vf*), present in cv. Prima at the heterozygous state (Maliepaard et al., 1998; Durel et al., 2000), and for two broad-spectrum QTLs on linkage groups 11 and 17 (referred to as F11 and F17 in the present study) detected in cv. Fiesta both in the greenhouse and in the field (Durel et al., 2003, 2004; Liebhard et al., 2003). Using molecular markers (Maliepaard et al., 1998; Schouten et al., 2012), we selected 16 apple genotypes (Table 1) that carried either no resistant allele for both QTLs (Class0 = both QTLs at the homozygous 'susceptible/susceptible' state), only one resistant allele for QTL F11 (ClassF11 = QTL F11 at the heterozygous 'resistant/susceptible' state while QTL F17 is at the homozygous susceptible state), only one resistant allele for QTL F17 (ClassF17 = QTL F17 at the heterozygous 'resistant/susceptible' state while QTL F11 is at the homozygous susceptible state), or two resistant alleles, one for each of the QTLs, F11 and F17 (ClassF11F17 = both QTLs F11 and F17 at the heterozygous 'resistant/susceptible' state), from the 161 progenies. The selected genotypes did not carry the major resistance genes *Rvi1* or *Rvi6*. For the sake of simplicity, "presence/absence of the favourable allele of a given QTL" is indicated by "presence/absence of the QTL" in the text.

Each genotype was grafted onto the apple rootstock, 'Pajam 2', and planted in an orchard network. This network was composed of three orchards located at three sites at a distance of more than 300 km from each other, representing three different environments and climates (Lanxade in South-Western France, Angers in North-Western France, and Villeneuve d'Ascq in Northern France, referred to as "Villeneuve" in the text). One replicate per genotype was planted in a randomised three-block design in each orchard.

Table 1
Description of the apple genotypes of cv. Prima x cv. Fiesta progeny used in the present study.

Class	Apple genotype	Orchard	Pathogenicity test (1)	Pathogenicity test (2)
Class0	J25	x	x	x
	J32	x		
	J51	x		
	J61	x		
	J108	x		
	J119	x	x	
	J150	x		
ClassF11	J66	x	x	x
	J151	x		
	J153	x	x	
ClassF17	J99	x	x	
	J160	x	x	x
ClassF11F17	J28	x		
	J63	x	x	x
	J80	x	x	
	J115	x		

x used in the orchard or pathogenicity test.