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Quantitative resistance affects the speed of frequency increase but not the diversity of the virulence alleles overcoming a major resistance gene to *Leptosphaeria maculans* in oilseed rape

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ABSTRACT

Quantitative resistance mediated by multiple genetic factors has been shown to increase the potential for durability of major resistance genes. This was demonstrated in the *Leptosphaeria maculans*/*Brassica napus* pathosystem in a 5 year recurrent selection field experiment on lines harboring the qualitative resistance gene *Rlm6* combined or not with quantitative resistance. The quantitative resistance limited the size of the virulent isolate population. In this study we continued this recurrent selection experiment in the same way to examine whether the pathogen population could adapt and render the major gene ineffective in the longer term. The cultivars Eurol, with a susceptible background, and Darmor, with quantitative resistance, were used. We confirmed that the combination of qualitative and quantitative resistance is an effective approach for controlling the pathogen epidemics over time. This combination did not prevent isolates virulent against the major gene from amplifying in the long term but the quantitative resistance significantly delayed for 5 years the loss of effectiveness of the qualitative resistance and disease severity was maintained at a low level on the genotype with both types of resistance after the fungus population had adapted to the major gene. We also showed that diversity of *AvrLm6* virulence alleles was comparable in isolates recovered after the recurrent selection on lines carrying either the major gene alone or in combination with quantitative resistance: a single repeat-induced point mutation and deletion events were observed in both situations. Breeding varieties which combine qualitative and quantitative resistance can effectively contribute to disease control by increasing the potential for durability of major resistance genes.

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1. Introduction

Global food security and sustainable crop production largely depend on the control of pathogen epidemics. Crop protection strategies have the dual aim of achieving effective and stable epidemic control. Their effectiveness – *i.e.* the capacity to produce an effect at one point of time and space – depends on pathogen biology and population size (McDonald and Linde, 2002; Bousset and Chèvre, 2012). The stability of their effect – *i.e.* the persistence of

their effectiveness in time and space – depends on adaptation dynamics in pathogen populations (Bousset and Chèvre, 2013). How epidemics reduce crop yield is influenced by plants, pathogens, environment, and human actions. Genetic resistance is undeniably an effective tactic for crop protection and offers an alternative to pesticide use. Thus, a main objective is to breed cultivars which carry resistance factors conferring the most effective resistance possible while optimizing the stability of this effectiveness.

Durable disease resistance is resistance that remains effective during prolonged and widespread use in an environment favourable to disease (Johnson, 1981, 1984). The durability of a resistant genotype will depend on (i) the evolutionary potential of the pathogen population that can be inferred from the reproduction system, the potential for gene flow (dispersal), the effective population size and mutational rates, (ii) the way the resistance

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is deployed and its consequences on pathogen population adaptation dynamics but also (iii) the nature of the resistance present in the genotype (Barrett et al., 2008; McDonald and Linde, 2002; Parlevliet, 2002). Resistant cultivars can harbor either qualitative resistance mediated by single gene(s) providing complete or high level resistance, or quantitative resistance mediated by many quantitative trait loci – QTL – with each providing a partial increase in resistance, or a combination of both types. A resistant plant genotype imposes a selection pressure on the pathogen populations and reduces its size. The magnitude of these effects depends on the type of resistance carried by the plant. Then, because the nature of the resistance affects the structure and the size of the pathogen populations, plant breeding can contribute to the goal of increasing not only the effectiveness, but also the potential for durability of resistance (Bousset et al., 2011). The knowledge of the relative potential for durability of different genetic constructions i.e. the relative expected duration of the benefit of using a construction rather than another is important to guide breeding. It is especially important to foster breeding efforts that favor some more beneficial constructions.

The relative potential for durability of different resistant genotypes prior to their commercial release can be assessed theoretically or empirically. Only a few theoretical studies have compared potential for durability by modeling the evolution of a pathogen population faced with different genotypes or combinations of genotypes and rarely include quantitative resistance (Fabre et al., 2009; Kiyosawa, 1982; Sapoukhina et al., 2009, 2013). Experimental data comparing the potential for durability of different genetic constructions confronted with the same pathogen population prior to their release are also scarce. One experimental approach that has proved successful is based on annual recurrent selection of pathogen populations on the resistant genotypes to be assessed (Brun et al., 2000). Based on such a recurrent selection, results reported in three different pathosystems involving virus, nematode and fungus showed that the combination of major genes (or major QTL) with other smaller effect QTL is an effective way to increase the potential for durability of the resistance. In these three cases, the reduction of the virulent population size by the quantitative resistance was a major determinant of this durability increase. Nevertheless, partial adaptation to the combination of qualitative and quantitative resistance was reported for the virus and nematode models.

The *pvr2³* gene controlling the resistance to *Potato virus Y* in pepper was defeated at high frequency when introgressed into a susceptible genetic background whereas it was not when combined with partial quantitative resistance (Palloix et al., 2009). Quenouille et al. (2013) recently showed that the quantitative resistance level and its effect on viral accumulation was the major determinant of the resistance breakdown frequency i.e. the frequency of plants where the virus was systemically detected. The fittest variants able to infect the plants with *pvr2³* and quantitative resistance were only recovered at a very low frequency in relation to the number or nature of mutations required to render the resistance ineffective. The quantitative resistance level had a direct effect on the risk of these variants appearing by limiting the size of the virus population (Quenouille et al., 2013). Although the combination of a major gene with quantitative resistance did significantly increase the potential for durability of the resistance, PVY could adapt to the pepper lines combining both resistances either after primary selection for virulence towards the major gene or after the increase in selection pressure and only some of the molecular events allowed to produce variants fittest on the combination of resistances (Palloix et al., 2009; Quenouille et al., 2013).

The plant genetic background was also shown to have an impact on the resistance durability of different potato genotypes carrying the major QTL *GpaV_{vrn}* for resistance to potato cyst nematode (*Globodera pallida*) (Fournet et al., 2013). The genetic background

had an effect on the evolution of the sex ratio within the population, by limiting the number of larvae that developed into females and then on the number of generations of recurrent selection required to get a significant increase in the percentage of females produced. Eight generations of recurrent selection were not sufficient to obtain nematode populations that rendered the resistance of some genotypes completely ineffective and it was hypothesized that additional resistance factors are present in these genotypes (Fournet et al., 2013). However, the percentage of females produced significantly increased on the genotypes which had the least lost their effectiveness, after five or eight generations of recurrent selection depending on the nematode population.

The durability of the qualitative *Rlm6* resistance of oilseed rape to *Leptosphaeria maculans*, a major pathogen of oilseed rape crops (Fitt et al., 2006), was also increased when it was combined with quantitative resistance (Brun et al., 2010). *Rlm6*-mediated resistance in a susceptible background became ineffective after 3 years of recurrent selection but it was still effective after 5 years when combined with quantitative resistance (Brun et al., 2010). Isolates which were virulent against the major gene *Rlm6* were present at a low frequency at the beginning of the experiment and were rapidly amplified after recurrent selection on the genotype carrying the major gene in a susceptible background. When quantitative resistance was combined with the major gene, it had a direct effect on the amplification and thus on the size of the virulent population by limiting disease symptoms and subsequent reproduction (Brun et al., 2010). However, this protection might not be effective enough to prevent the pathogen population adaptation and the loss of effectiveness of the major gene in the longer term. This hypothesis can only be tested by continuing the recurrent selection experiment.

L. maculans, combining large population sizes, a mixed reproduction regime and a high dispersal ability, is a fungus with high evolutionary potential (West et al., 2001; McDonald and Linde, 2002). The molecular diversity of virulence alleles has been investigated in different situations where monogenic resistance genes have been defeated. Three main molecular mechanisms leading to virulence were reported. The *Rlm1* gene in susceptible backgrounds was defeated after 3 years of commercial cultivation (Rouxel et al., 2003). Gout et al. (2007) showed that a unique large deletion of a chromosomal fragment containing *AvrLm1* was responsible for the switch to virulence on *Rlm1* and was largely predominating in the populations a few years after *Rlm1* overcoming. Fudal et al. (2009) investigated the molecular mechanisms at the *AvrLm6* locus responsible for the switch to virulence on *Rlm6* in a set of isolates recovered from different recurrent selection experiments on *Rlm6* in susceptible backgrounds. *AvrLm6* was shown to be absent from 66% of the virulent isolates tested. Sequencing of virulence alleles revealed the presence of repeat-induced point (RIP) mutations leading to the modification of 4–9% of the bases compared to the avirulence allele and generating 2–4 stop codons. Parlange et al. (2009) found that a single base mutation, changing a glycine residue to an arginine residue, was responsible for the loss of recognition of *AvrLm4* by *Rlm4*. Finally, Daverdin et al. (2012) investigated the first events leading to virulence against *Rlm7* after introduction of this specific resistance in commercial varieties. Sequence analysis of the virulence alleles showed that a great diversity of molecular events had occurred with mainly RIP mutations in the early stage of population evolution and deletion events later (Daverdin et al., 2012). Investigating the diversity of molecular events leading to virulence against *Rlm6* in our recurrent selection scheme would then help to understand the changes in the populations.

In this study the recurrent selection experiment on the *L. maculans*/*B. napus* pathosystem was continued on the qualitative resistance gene *Rlm6* in combination or not with quantitative

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