



Pyramiding Quantitative Resistance with a Major Resistance Gene in Apple: From Ephemeral to Enduring Effectiveness in Controlling Scab

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Pauline Lasserre-Zuber, Valérie Caffier, René Stievenard, Arnaud Lemarquand, Bruno Le Cam, et al.. Pyramiding Quantitative Resistance with a Major Resistance Gene in Apple: From Ephemeral to Enduring Effectiveness in Controlling Scab. *Plant Disease*, 2018, 102 (11), pp.2220-2223. 10.1094/PDIS-11-17-1759-RE . hal-02516687

HAL Id: hal-02516687

<https://univ-angers.hal.science/hal-02516687>

Submitted on 26 May 2020

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1 **Pyramiding Quantitative Resistance with a Major Resistance Gene in**
2 **Apple: from Ephemeral to Enduring Effectiveness in Controlling Apple**
3 **Scab**

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Abstract

Genetic resistance is a useful strategy to control plant disease, but its effectiveness may be reduced over time due to the emergence of pathogens able to circumvent the defences of the plant. However, the pyramiding of different resistance factors in the same plant can improve the effectiveness and durability of the resistance. To investigate the potential for this approach in apple to control scab disease we surveyed scab incidence in two experimental orchards located more than 300 km apart planted with apple genotypes carrying quantitative resistance and major gene resistance alone or in combination. Our results showed that the effectiveness of pyramiding in controlling scab was dependent on the site and could not be completely explained by the effectiveness level of the resistances alone.

Key words: apple, *Malus x domestica*, scab, *Venturia inaequalis*, durability, sustainability, Quantitative Trait Locus, major resistance gene, pyramiding

The control of plant diseases by chemical products presents risks to human health and the ecological environment. Furthermore, these treatments can be ineffective if they are not used at the appropriate time or if the pathogens adapt and become resistant. Plant genetic resistance constitutes an environmentally friendly way to control disease and to reduce chemical inputs. Plant resistance can be described by two traits: (1) its phenotype with either a complete or partial level of resistance, and (2) its mode of heredity with either a continuous or a bimodal distribution between resistant and susceptible phenotypes in a progeny (Niks et al. 2015). Plant resistance is generally classified in two extreme categories: qualitative resistance conditioned by a single gene (i.e. major gene) and quantitative resistance conditioned by multiple genes of partial effect (i.e. Quantitative Trait Locus = QTL), with possibilities of intermediate types of resistances between these two categories (Poland et al. 2009; Stuthman et al. 2007). Genetically homogeneous crops with qualitative resistance induce strong directional selection on pathogen populations when they are deployed at a large scale and can result in resistance breakdown (Bus et al. 2011; Peressotti et al. 2010; Rouxel et al. 2003). Quantitative resistance has been frequently considered to be more durable than qualitative resistance (Lindhout 2002; Parlevliet 2002; Stuthman et al. 2007). However, it was postulated that quantitative resistance might also exert selection on pathogen populations (Gandon and Michalakakis 2000; Parlevliet and Zadoks 1977). Indeed, it was shown experimentally in several pathosystems that quantitative resistance can also be eroded, for instance for *Mycosphaerella graminicola* on wheat (Cowger and Mundt 2002), lettuce mosaic virus on lettuce (Pink et al. 1992), *Phytophthora infestans* on potato (Andrison et al. 2007; Montarry et al. 2008), *Plasmopara viticola* on grape (Delmas et al. 2016; Delmotte et al. 2014) and *Venturia inaequalis* on apple (Caffier et al. 2014; Caffier et al. 2016). As a consequence, both types of resistance may be eroded or overcome more or less rapidly according to the evolutionary potential of the pathogen, the host characteristics and the agronomic practices. It is therefore necessary to develop strategies of resistance gene management that can improve the durability of resistance (Fabre et al. 2012; Mundt 2014; Sapoukhina et al. 2009; Sapoukhina et al. 2013).

Pyramiding genes of resistance in one cultivar is one of these strategies and is now feasible by marker-assisted breeding (Pilet-Nayel et al. 2017; Pink 2002). Several studies have demonstrated that combining resistance factors increases resistance effectiveness, either by cumulating major genes (Hittalmani et al. 2000; Tan et al. 2010; Koide et al. 2010), QTL (Castro et al. 2003; Hamon et al. 2010; Laloi et al. 2017; Richardson et al. 2006) or major genes and QTL (Eibach et al. 2007; Tabien et al. 2002). It has been experimentally observed that combinations of major genes and QTL can increase both resistance effectiveness and durability, for instance for *Leptosphaeria maculans* on rapeseed (Delourme et al. 2014), *Microcyclus ulei* on rubber (Le Guen et al. 2011), *Globodera pallida* on potato (Fournet et al. 2013), *Meloidogyne* species on pepper (Barbary et al. 2014) and Potato

1 virus Y on pepper (Palloix et al. 2009 ; Quenouille et al. 2014). Indeed, major genes are totally
 2 effective to control the avirulent fraction of the pathogen population, whereas QTL reduce the
 3 pathogen population size, which limits the selection for virulent isolates and maintains a partial
 4 effectiveness of the resistance towards the virulent isolates (Delourme et al. 2014). Theoretical
 5 studies indicate that durability of cultivars pyramiding resistances will depend on the way that the
 6 resistance genes are employed (Bourget et al. 2013). If each resistance gene is used separately,
 7 concomitantly or before the use of cultivars pyramiding resistances, then the pathogen will be able
 8 to overcome the resistance by incrementally stacking virulences (Ayliffe et al. 2008; Pink 2002;
 9 Stuthman 2002) either by independent successive gain of virulence in the same strain or by sexual
 10 recombination between mono-virulent strains. It should be noted that the durability of resistance
 11 pyramiding may depend on the genetic structure of the pathogen populations and ability of
 12 pathogen populations carrying single mutations to recombine, but this point has not yet been
 13 explored experimentally.

14 Here we investigated the effectiveness and durability of combining one major gene with two
 15 QTL in the apple scab pathosystem. Apple scab, caused by the fungus *Venturia inaequalis*, is one of
 16 the most important diseases in commercial orchards in all temperate countries, because it strongly
 17 reduces fruit quality which is vital for fresh market sales. Disease control today generally depends on
 18 the use of fungicides, spraying up to 20 treatments per year (Sauphanor et al. 2009). The perennial
 19 trait of apple necessitates genotypes that can maintain resistance over many years. Major resistance
 20 genes have been identified in wild or domesticated apple species (Bus et al. 2011; Gessler et al. 2006;
 21 Soufflet-Freslon et al. 2008). The *Rvi6* (= *Vf*) major resistance gene has been widely used in apple
 22 breeding programs during the last 50-60 years (Laurens 1999), but this monogenic resistance has
 23 been overcome by the pathogen (Bus et al. 2011; Lemaire et al. 2016). Many QTL, including specific
 24 and broad-spectrum QTL, have also been identified (Calenge et al. 2004; Durel et al. 2003; Lê Van et
 25 al. 2013 ; Liebhard et al. 2003a; Soufflet-Freslon et al. 2008). The combination of two broad-spectrum
 26 resistance QTL, F11 and F17, has already been shown to be eroded in experimental trials (Caffier et
 27 al. 2014). Thus, our aim was to evaluate the utility of pyramiding *Rvi6* with QTL F11 and F17, in two
 28 unsprayed orchards where resistance effectiveness of these QTL and this major gene has already
 29 been challenged.

31 Materials and Methods

32 **Plant material.** A cross between cv. 'Prima' and cv. 'Fiesta' was carried out by the former
 33 CPRO-DLO, which is now part of Wageningen-UR Plant Breeding, The Netherlands (Maliepaard et al.
 34 1998). These progeny (referred to as the "J progeny") segregate for two major genes, *Rvi1* (= *Vg*) and

1 *Rvi6* (= *Vff*), and two broad-spectrum QTL, since cv. 'Prima' carries *Rvi1* and *Rvi6* (Durel et al. 2000;
 2 Maliepaard et al. 1998) and cv. 'Fiesta' carries two QTL on linkage groups 11 and 17 (referred to as
 3 F11 and F17 in the present study) (Durel et al. 2004; Durel et al. 2003; Liebhard et al. 2003b). Using
 4 available molecular markers for the initial and curated marker positions published in Maliepaard et
 5 al. (1998) and Schouten et al. (2012) respectively, we selected 22 apple genotypes that carried either
 6 no favourable allele for QTL or for *Rvi6* (Class-0), two favorable alleles for both QTL F11 and F17 only
 7 (Class-QTL), favourable allele for *Rvi6* only (Class-R), and favorable alleles for F11, F17 and *Rvi6*
 8 (Class-RQTL), from the 161 available progenies. As *Rvi1* is largely overcome in *V. inaequalis*
 9 populations, we considered that this major gene had no effect, and that the classes with *Rvi1* and
 10 *Rvi6* represented the effect of *Rvi6*. The number of genotypes per combination varied from five to
 11 seven (Table 1).

12 **Orchard network.** During winter 2003-2004, each genotype was planted in two experimental
 13 orchards located more than 300 km apart, representing two different environments and climates
 14 (Angers in north-western France, and Villeneuve d'Ascq in the north of France, later referred to as
 15 "Villeneuve"). In each orchard, three replicates per genotype were planted in a randomised three-
 16 block design after grafting onto the apple rootstock, 'Pajam 2'. The distance between the trees on a
 17 row was 1.5 m in Villeneuve and 1 m in Angers; the distance between the rows was 4 m. Pesticides
 18 were applied when necessary, but no fungicide treatment was applied against scab.

19 **Disease assessment.** Scab incidence was scored on each tree in June after the period of
 20 primary infection, in 2006-2008 at both sites. In Villeneuve, data from 2007 could not be interpreted
 21 because of a very low level of disease on the susceptible trees. In Angers, where the orchard was
 22 maintained after the end of the project, an additional assessment was carried out in 2012. On each
 23 tree, disease incidence was scored using an ordinal scale adapted from Lateur and Populer (1994).
 24 This scale consists of nine levels, described as intervals around the following values: 0%, 1%, 3%,
 25 12.5%, 25%, 37.5%, 50%, 75% and 90% of scabbed leaves.

26 **Statistical analysis.** All statistical analyses were performed on the variable "disease
 27 incidence". Within each site and each year, the four classes of genotypes were compared for their
 28 disease incidence using a linear mixed effect model LME (Pinheiro and Bates 2000). The 'genotype'
 29 was treated as a random factor nested in the class of genotypes, and 'block' as a fixed factor. The
 30 model was fitted by maximizing the log-likelihood and the best model was selected using the
 31 Bayesian Information Criterion after including each factor ('class of genotypes' and 'block') through
 32 an ascendant selection (Pinheiro and Bates 2000). The data were transformed into arcsine square
 33 root to normalise and homogenise the residuals. The resistance of Class-R, Class-QTL or Class-RQTL
 34 was considered as effective if "disease incidence" was significantly different from "disease incidence"

of Class-0. All statistical analyses were performed with R software, version 3.1.2 (R Core Team 2016), using the 'nlme' package (Pinheiro et al. 2016).

Results

In both sites, the level of disease on the control (Class-0) was high with fluctuations from year to year ranging from 51% to 83% scabbed leaves. In Angers, the resistance based on *Rvi6* (Class-R) was completely effective in 2006 with no scabbed leaves (fig. 1a). This resistance was still effective in 2007 but a higher level of disease was observed (22% scabbed leaves). This resistance became ineffective by 2008 (67% scabbed leaves). The QTL combination F11F17 (Class-QTL) significantly reduced the disease each year compared to Class-0, even if the level of control decreased slowly over time (not shown in the present study, see Caffier et al. 2014). In 2006, the pyramiding of *Rvi6* and F11F17 (Class-RQTL) was completely effective with no scabbed leaves, due to the effectiveness of *Rvi6*. In 2007, this pyramiding was significantly more effective (6% scabbed leaves) than the resistance factors *Rvi6* (Class-R with 22% scabbed leaves) or F11F17 (Class-QTL with 55% scabbed leaves) alone. In 2008 and 2012, the resistance of Class-RQTL was still effective with 20% and 64% scabbed leaves respectively, compared to 75 and 83% for Class-0. However, the added value of *Rvi6* was lost from 2008, while the effect of F11F17 remained with no significant difference between Class-RQTL and Class-QTL in 2008 and in 2012. By contrast, in Villeneuve, both the major resistance *Rvi6* (Class-R) and the QTL combination F11F77 (Class-QTL) were ineffective from the beginning of the experiment with more than 40% scabbed leaves. However, the apple genotypes carrying the pyramiding of *Rvi6* and F11F17 (Class-RQTL) were significantly less diseased than Class-0 with less than 20% scabbed leaves for the two observed years 2006 and 2008 (fig. 1b).

Discussion

We evaluated the effectiveness of pyramiding quantitative resistance with a major resistance gene in two contrasting situations: one site (Angers) where the resistance factors alone were progressively overcome during the experiment trial and one site (Villeneuve) where the resistance factors alone were already overcome when the experiment started (Caffier et al. 2014). Our results showed the effectiveness of pyramiding resistances for controlling scab in both sites, but to a different extent depending on the site. In Angers, the added value of pyramiding quantitative resistance with a major gene to control scab was ephemeral and disappeared when the level of disease control based on resistance factors alone decreased, whereas in Villeneuve, the pyramiding (Class-RQTL) remained effective both in 2006 and 2008, even if the resistance factors alone were not effective compared to Class-0.

1 These results agree with other studies suggesting pyramiding QTL with major resistance
2 genes improves effectiveness and increases durability of resistance (Delourme et al. 2014; Le Guen et
3 al. 2011; Quenouille et al. 2014). The effectiveness of pyramiding may be due to the low probability
4 of a pathogen strain to accumulate independent mutations necessary to overcome all resistance
5 genes in the pyramid (Mundt 2014), the reduction in the pathogen size population or the
6 diversification in the selection pressures exerted on the pathogen population (Pilet-Nayel et al.
7 2017). It is surprising however that the added value of pyramiding was maintained in Villeneuve
8 whereas it was lost in Angers. Indeed, at the Villeneuve site, the effectiveness of pyramiding the *Rvi6*
9 gene with QTL was observed although apple genotypes with single non-effective resistance factors
10 (Class-R or Class-QTL) were planted in the same orchard, which putatively facilitates stacking
11 pathogenicity loci by mating. In such a situation, we could have expected disease control associated
12 with pyramiding to decrease or be completely lost over time (Ayliffe et al. 2008; Pink 2002; Stuthman
13 2002), which was not observed. We assume that the effectiveness of resistance observed at this site
14 was probably due to a lack of recombination between pathogen populations growing on class-R and
15 populations growing on class-QTL. Indeed, a strong genetic differentiation was observed in the north-
16 west of France between populations virulent towards *Rvi6* and populations avirulent towards *Rvi6*
17 (Gladieux et al. 2011). At the Angers site, the improved resistance associated with pyramiding instead
18 of single resistance factors was also observed in 2007 at the beginning of *Rvi6* breakdown, but was
19 lost from 2008, with the level of disease control associated with pyramiding QTL and the major gene
20 (Class-RQTL) becoming similar to the level of disease control observed with the QTL (Class-QTL)
21 alone. At this site, Leroy et al. (2016) showed a low genetic differentiation between populations
22 virulent towards *Rvi6* and populations avirulent towards *Rvi6* due to recombination between the two
23 populations. Such a recombination may have resulted in the development of strains that were well
24 adapted to all resistance factors incorporated in the pyramiding. Our results suggest that the
25 effectiveness of pyramiding resistance genes not only depends on the way the resistance genes are
26 used (Ayliffe et al. 2008; Bourget et al. 2013; Pink 2002; Stuthman 2002) but also on the capacity of
27 pathogen populations carrying virulence and aggressiveness traits to recombine. We assume that the
28 more gene flow that occurs between populations able to overcome different resistances the more
29 quickly the resistance of the pyramiding will be eroded.

30 Even in case of ephemeral effectiveness, these results are promising for the development of
31 environmentally friendly strategies to control apple scab and to reduce chemical treatments. Indeed,
32 in a previous study, Didelot et al. (2016) showed that the breakdown of the major resistance *Rvi6* by
33 virulent isolates could be delayed by the removal of leaf litter in autumn associated with spraying of
34 fungicides in case of moderate or high risks of scab infection. We can expect that the association of
35 all these control methods, i.e. pyramiding of quantitative resistance with major resistance genes,

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7

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sanitation and chemical treatment applied only in case of great risk of scab infection, will provide a sustainable management of disease in a low-fungicide input system, which is crucial for a perennial plant.

Acknowledgements

We thank Wageningen-UR Plant Breeding for the availability of the apple progeny of cv. 'Prima' x cv. 'Fiesta' and especially Eric van de Weg for the sharing/use of molecular marker data for this family. We thank Eric van de Weg and Jason Shiller for helpful comments on the manuscript. We thank Mathieu Lascostes from CRRG for his contribution to the planting of the experimental orchard at Villeneuve d'Ascq and the phenotyping. We thank Philippe Billy, Christophe Brouard, Serge Brunet, Christophe Vilfroy from INRA Experimental Unit UEH for participating to the phenotyping and taking care of the experimental orchard located in Angers. We thank Abou Kouassi and Amidou N'Diaye for their contribution in the phenotyping during their post-doc stay at INRA-Angers. We also thank Fabienne Mathis and Caroline Denancé from IRHS for complementary genotyping. This work was funded by the CTPS programme "Durabilité des résistances variétales à des bioagresseurs 2006-2008" and the ANR grant, "ANR-05-PADD-05, CEDRE".

Literature cited

- Andrison, D., Pilet F., Montarry J., Hafidi M., Corbière R., Achbani E.H., Pellé R., Ellissèche D., 2007. Adaptation of *Phytophthora infestans* to partial resistance in potato: Evidence from French and Moroccan populations. *Phytopathology* 97:338-343.
- Ayliffe, M., Singh R., Lagudah E., 2008. Durable resistance to wheat stem rust needed. *Curr. Opin. Plant Biol.* 11:187-192.
- Barbary, A., Palloix A., Fazari A., Marteu N., Castagnone-Sereno P., Djian-Caporalino C., 2014. The plant genetic background affects the efficiency of the pepper major nematode resistance genes *Me1* and *Me3*. *Theor. Appl. Genet.* 127:499-507.
- Bourget, R., Chaumont L., Sapoukhina N., 2013. Timing of pathogen adaptation to a multicomponent treatment. *PLoS ONE* 8.

- 1 Bus, V.G.M., Rikkerink E.H.A., Caffier V., Durel C.E., Plummer K.M., 2011. Revision of the
2 nomenclature of the differential host-pathogen interactions of *Venturia inaequalis* and *Malus*. Annu.
3 Rev. Phytopathol. 49:391-413.
4
- 5 Caffier, V., Lasserre-Zuber P., Giraud M., Lascostes M., Stievenard R., Lemarquand A., van de Weg E.,
6 Expert P., Denancé C., Didelot F., Le Cam B., Durel C., 2014. Erosion of quantitative host resistance in
7 the apple - *Venturia inaequalis* pathosystem. Infect. Genet. Evol. 27:481-489.
8
- 9 Caffier, V., Le Cam B., Al Rifaï M., Bellanger M.N., Comby M., Denance C., Didelot F., Expert P.,
10 Kerdraon T., Lemarquand A., Ravon E., Durel C.E., 2016. Slow erosion of a quantitative apple
11 resistance to *Venturia inaequalis* based on an isolate-specific Quantitative Trait Locus. Infect. Genet.
12 Evol. 44:541-548.
13
- 14 Calenge, F., Faure A., Goerre M., Gebhardt C., van de Weg W.E., Parisi L., Durel C.E., 2004.
15 Quantitative trait loci (QTL) analysis reveals both broad-spectrum and isolate-specific QTL for scab
16 resistance in an apple progeny challenged with eight isolates of *Venturia inaequalis*. Phytopathology
17 94:370-379.
18
- 19 Castro, A.J., Chen X., Hayes P.M., Johnston M., 2003. Pyramiding quantitative trait locus (QTL) alleles
20 determining resistance to barley stripe rust: Effects on resistance at the seedling stage. Crop Sci.
21 43:651-659.
22
- 23 Cowger, C., Mundt C.C., 2002. Aggressiveness of *Mycosphaerella graminicola* isolates from
24 susceptible and partially resistant wheat cultivars. Phytopathology 92:624-630.
25
- 26 Delmas, C.E.L., Fabre F., Jolivet J., Mazet I.D., Richart Cervera S., Delière L., Delmotte F., 2016.
27 Adaptation of a plant pathogen to partial host resistance: selection for greater aggressiveness in
28 grapevine downy mildew. Evol. Appl. 9:709-725.
29
- 30 Delmotte, F., Mestre P., Schneider C., Kassemeyer H.H., Kozma P., Richart-Cervera S., Rouxel M.,
31 Delière L., 2014. Rapid and multiregional adaptation to host partial resistance in a plant pathogenic
32 oomycete: evidence from European populations of *Plasmopara viticola*, the causal agent of
33 grapevine downy mildew. Infect. Genet. Evol. 27:500-508.
34

- 1 Delourme, R., Bousset L., Ermel M., Duffé P., Besnard A.L., Marquer B., Fudal I., Linglin J., Chadoeuf J.,
2 Brun H., 2014. Quantitative resistance affects the speed of frequency increase but not the diversity of
3 the virulence alleles overcoming a major resistance gene to *Leptosphaeria maculans* in oilseed rape.
4 Infect. Genet. Evol. 27:490-499.
5
- 6 Didelot, F., Caffier V., Orain G., Lemarquand A., Parisi L., 2016. Sustainable management of scab
7 control through the integration of apple resistant cultivars in a low-fungicide input system. Agric.
8 Ecosyst. Environ. 217:41-48.
9
- 10 Durel, C.E., Calenge F., Parisi L., van de Weg W.E., Kodde L.P., Liebhard R., Gessler C., Thiermann M.,
11 Dunemann F., Gennari F., Tartarini S., Lespinasse Y., 2004. An overview of the position and
12 robustness of scab resistance QTLs and major genes by aligning genetic maps of five apple progenies.
13 Acta Hortic. 663:135-140.
14
- 15 Durel, C.E., Parisi L., Laurens F., van de Weg W.E., Liebhard R., Jourjon M.F., 2003. Genetic dissection
16 of partial resistance to race 6 of *Venturia inaequalis* in apple. Genome 46:224-234.
17
- 18 Durel, C.E., van de Weg W.E., Venisse J.S., Parisi L., 2000. Localisation of a major gene for apple scab
19 resistance on the European genetic map of the Prima x Fiesta cross. IOBC wprs Bulletin 23:245-248.
20
- 21 Eibach, R., Zyprian E., Welter L., Topfer R., 2007. The use of molecular markers for pyramiding
22 resistance genes in grapevine breeding. Vitis 46:120-124.
23
- 24 Fabre, F., Rousseau E., Mailleret L., Moury B., 2012. Durable strategies to deploy plant resistance in
25 agricultural landscapes. New Phytol. 193:1064-1075.
26
- 27 Fournet, S., Kerlan M.C., Renault L., Dantec J.P., Rouaux C., Montarry J., 2013. Selection of
28 nematodes by resistant plants has implications for local adaptation and cross-virulence. Plant Pathol.
29 62:184-193.
30
- 31 Gandon, S., Michalakakis Y., 2000. Evolution of parasite virulence against qualitative or quantitative
32 host resistance. Proc. R. Soc. Lond. B 267:985-990.
33
- 34 Gessler, C., Patocchi A., Sansavini S., Tartarini S., Gianfranceschi L., 2006. *Venturia inaequalis*
35 resistance in apple. Crit. Rev. Plant Sci. 25:473-503.

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- 1
- 2 Gladieux, P., Guerin F., Giraud T., Caffier V., Lemaire C., Parisi L., Didelot F., Le Cam B., 2011.
- 3 Emergence of novel fungal pathogens by ecological speciation: importance of the reduced viability of
- 4 immigrants. *Mol. Ecol.* 20:4521-4532.
- 5
- 6 Hamon, C., Baranger A., Miteul H., Lecointe R., Le Goff I., Deniot G., Onfroy C., Moussart A., Prosperi
- 7 J.M., Tivoli B., Delourme R., Pilet-Nayel M.L., 2010. A complex genetic network involving a broad-
- 8 spectrum locus and strain-specific loci controls resistance to different pathotypes of *Aphanomyces*
- 9 *euteiches* in *Medicago truncatula*. *Theor. Appl. Genet.* 120:955-970.
- 10
- 11 Hittalmani, S., Parco A., Mew T.V., Zeigler R.S., Huang N., 2000. Fine mapping and DNA marker-
- 12 assisted pyramiding of the three major genes for blast resistance in rice. *Theor. Appl. Genet.*
- 13 100:1121-1128.
- 14
- 15 Koide, Y., Kawasaki A., Telebanco-Yanoria M.J., Hairmansis A., Nguyet N.T.M., Bigirimana J., Fujita D.,
- 16 Kobayashi N., Fukuta Y., 2010. Development of pyramided lines with two resistance genes, Pish and
- 17 Pib, for blast disease (*Magnaporthe oryzae* B. Couch) in rice (*Oryza sativa* L.). *Plant Breed.* 129:670-
- 18 675.
- 19
- 20 Laloi, G., Vergne E., Durel C.-E., Le Cam B., Caffier V., 2017. Efficiency of pyramiding of three
- 21 quantitative resistance loci to apple scab. *Plant Pathol.* 66:412–422.
- 22
- 23 Lateur, M., Populer C., 1994. Screening fruit tree genetic resources in Belgium for disease resistance
- 24 and other desirable characters. *Euphytica* 77:147-153.
- 25
- 26 Laurens, F., 1999. Review of the current apple breeding programmes in the world: objectives for
- 27 scion cultivar improvement. *Acta Hortic.* 484:163-170.
- 28
- 29 Le Guen, V., Garcia D., Doaré F., Mattos C.R.R., Condina V., Couturier C., Chambon A., Weber C.,
- 30 Espéout S., Seguin M., 2011. A rubber tree's durable resistance to *Microcyclus ulei* is conferred by a
- 31 qualitative gene and a major quantitative resistance factor. *Tree Genet. Genomes* 7:877-889.
- 32

- 1 Lê Van, A., Caffier V., Lasserre-Zuber P., Chauveau A., Brunel D., Le Cam B., Durel C., 2013.
- 2 Differential selection pressures exerted by host resistance quantitative trait loci on a pathogen
- 3 population: a case study in an apple x *Venturia inaequalis* pathosystem. New Phytol. 197:899-908.
- 4
- 5 Lemaire, C., De Gracia M., Leroy T., Michalecka M., Lindhard-Pedersen H., Guerin F., Gladieux P., Le
- 6 Cam B., 2016. Emergence of new virulent populations of apple scab from nonagricultural disease
- 7 reservoirs. New Phytol. 209:1220-9.
- 8
- 9 Leroy, T., Caffier V., Celton J.M., Anger N., Durel C.E., Lemaire C., Le Cam B., 2016. When virulence
- 10 originates from nonagricultural hosts: evolutionary and epidemiological consequences of
- 11 introgressions following secondary contacts in *Venturia inaequalis* New Phytol. 210:1443-1452.
- 12
- 13 Liebhard, R., Kellerhals M., Pfammatter W., Jertmini M., Gessler C., 2003a. Mapping quantitative
- 14 physiological traits in apple (*Malus x domestica* Borkh.). Plant Mol. Biol. 52:511-526.
- 15
- 16 Liebhard, R., Koller B., Patocchi A., Kellerhals M., Pfammatter W., Jermini M., Gessler C., 2003b.
- 17 Mapping quantitative field resistance against apple scab in a 'Fiesta' x 'Discovery' progeny.
- 18 Phytopathology 93:493-501.
- 19
- 20 Lindhout, P., 2002. The perspectives of polygenic resistance in breeding for durable disease
- 21 resistance. Euphytica 124:217-226.
- 22
- 23 Maliepaard, C., Alston F.H., Van Arkel G., Brown L.M., Chevreau E., Dunemann F., Evans K.M.,
- 24 Gardiner S., Guilford P., Van Heusden A.W., Janse J., Laurens F., Lynn J.R., Manganaris A.G., den Nijs
- 25 A.P.M., Periam N., Rikkerink E., Roche P., Ryder C., Sansavini S., Schmidt H., Tartarini S., Verhaegh J.J.,
- 26 Vrielink-Van Ginkel M., King G.J., 1998. Aligning male and female linkage maps of apple (*Malus*
- 27 *pumila* Mill.) using multi-allelic markers. Theor. Appl. Genet. 97:60-73.
- 28
- 29 Montarry, J., Glais I., Corbiere R., Andrivon D., 2008. Adaptation to the most abundant host genotype
- 30 in an agricultural plant-pathogen system - potato late blight. J. Evolut. Biol. 21:1397-1407.
- 31
- 32 Mundt, C.C., 2014. Durable resistance: a key to sustainable management of pathogens and pests.
- 33 Infect. Genet. Evol. 27:446-55.
- 34

- 1 Niks, R.E., Qi X.Q., Marcel T.C., 2015. Quantitative resistance to biotrophic filamentous plant
2 pathogens: concepts, misconceptions, and mechanisms. *Annu. Rev. Phytopathol.* 53:445-470.
3
- 4 Palloix, A., Ayme V., Moury B., 2009. Durability of plant major resistance genes to pathogens depends
5 on the genetic background, experimental evidence and consequences for breeding strategies. *New*
6 *Phytol.* 183:190-199.
7
- 8 Parlevliet, J.E., 2002. Durability of resistance against fungal, bacterial and viral pathogens; present
9 situation. *Euphytica* 124:147-156.
10
- 11 Parlevliet, J.E., Zadoks J.C., 1977. The integrated concept of disease resistance: a new view including
12 horizontal and vertical resistance in plants. *Euphytica* 26:5-21.
13
- 14 Peressotti, E., Wiedemann-Merdinoglu S., Delmotte F., Bellin D., Di Gaspero G., Testolin R.,
15 Merdinoglu D., Mestre P., 2010. Breakdown of resistance to grapevine downy mildew upon limited
16 deployment of a resistant variety. *BMC Plant Biol.* 10:147.
17
- 18 Pilet-Nayel, M.L., Moury B., Caffier V., Montarry J., Kerlan M.C., Fournet S., Durel C.E., Delourme R.,
19 2017. Quantitative resistance to plant pathogens in pyramiding strategies for durable crop
20 protection. *Front. Plant Sci.* 8:1838.
21
- 22 Pinheiro, J., Bates D., Debroy S., Sarkar D., R Core Team, 2016. *Nlme: linear and nonlinear mixed*
23 *effects models. R package version 3.1-128:*<http://CRAN.R-project.org/package=nlme>.
24 Pinheiro, J.C., Bates D.M., 2000. *Mixed-effects models in S and S-PLUS*. New York, USA: Springer-
25 Verlag.
26
- 27 Pink, D.A.C., 2002. Strategies using genes for non-durable disease resistance. *Euphytica* 124:227-236.
28 Pink, D.A.C., Lot H., Johnson R., 1992. Novel pathotypes of lettuce mosaic virus - breakdown of a
29 durable resistance? *Euphytica* 63:169-174.
30
- 31 Poland, J.A., Balint-Kurti P.J., Wisser R.J., Pratt R.C., Nelson R.J., 2009. Shades of gray: the world of
32 quantitative disease resistance. *Trends Plant Sci.* 14:21-29.
33

- 1 Quenouille, J., Paulhiac E., Moury B., Palloix A., 2014. Quantitative trait loci from the host genetic
2 background modulate the durability of a resistance gene: a rational basis for sustainable resistance
3 breeding in plants. *Heredity* 112:579-587.
- 4
- 5 R Core Team, 2016. *R: A language and environment for statistical computing*. Vienna, Austria: R
6 Foundation for Statistical Computing [<http://www.R-project.org>].
- 7
- 8 Richardson, K.L., Vales M.I., Kling J.G., Mundt C.C., Hayes P.M., 2006. Pyramiding and dissecting
9 disease resistance QTL to barley stripe rust. *Theor. Appl. Genet.* 113:485-495.
- 10
- 11 Rouxel, T., Penaud A., Pinochet X., Brun H., Gout L., Delourme R., Schmit J., Balesdent M.H., 2003. A
12 10-year survey of populations of *Leptosphaeria maculans* in France indicates a rapid a
13 daptation towards the *Rlm1* resistance gene of oilseed rape. *Eur. J. Plant Pathol.* 109:871-881.
- 14
- 15 Sapoukhina, N., Durel C.E., Le Cam B., 2009. Spatial deployment of gene-for-gene resistance governs
16 evolution and spread of pathogen populations. *Theor. Ecol.* 2:229-238.
- 17
- 18 Sapoukhina, N., Paillard S., Dedryver F., de Vallavieille-Pope C., 2013. Quantitative plant resistance in
19 cultivar mixtures: wheat yellow rust as a modeling case study. *New Phytol.* 200:888-897.
- 20
- 21 Sauphanor, B., Dirwimmer, C., Boutin, S., Chaussabel, A.L., Dupont, N., Fauriel, J., Gallia, V., Lambert,
22 N., Navarro, E., Parisi, L., Plenet, D., Ricaud, V., Sagnes, J.L., Sauvaitre, D., Simon, S., Speich, P.,
23 Zavagli, F., 2009. Analyse comparative de différents systèmes en arboriculture fruitière. In: Volay, T.
24 (Ed.), *Ecophyto R&D: vers des systèmes de culture économes en produits phytosanitaires*, rapport
25 d'expertise collective. INRA, France.
- 26
- 27 Schouten, H.J., van de Weg W.E., Carling J., Khan S.A., Mckay S.J., Van Kaauwen M.P.W., Wittenberg
28 A.H.J., Koehorst-Van Putten H.J.J., Noordijk Y., Gao Z.S., Rees D.J.G., Van Dyk M.M., Jaccoud D.,
29 Considine M.J., Kilian A., 2012. Diversity arrays technology (DArT) markers in apple for genetic linkage
30 maps. *Mol. Breed.* 29:645-660.
- 31
- 32 Soufflet-Freslon, V., Gianfranceschi L., Patocchi A., Durel C.E., 2008. Inheritance studies of apple scab
33 resistance and identification of *Rvi14*, a new major gene that acts together with other broad-
34 spectrum QTL. *Genome* 51:657-667.
- 35

1 Stuthman, D.D., 2002. Contribution of durable disease resistance to sustainable agriculture.
2 Euphytica 124:253-258.
3
4 Stuthman, D.D., Leonard K.J., Miller-Garvin J., 2007. Breeding crops for durable resistance to disease.
5 Adv. Agron. 95:319-367.
6
7 Tabien, R.E., Li Z., Paterson A.H., Marchetti M.A., Stansel J.W., Pinson S.R.M., 2002. Mapping QTLs for
8 field resistance to the rice blast pathogen and evaluating their individual and combined utility in
9 improved varieties. Theor. Appl. Genet. 105:313-324.
10
11 Tan, M.Y.A., Hutten R.C.B., Visser R.G.F., Van Eck H.J., 2010. The effect of pyramiding *Phytophthora*
12 *infestans* resistance genes R (Pi-mcd1) and R (Pi-ber) in potato. Theor. Appl. Genet.
13

Figure Legend

Figure 1. Bar plots of apple scab incidence in two French orchards situated in: 1) Angers and 2) Villeneuve d'Ascq, for four classes of apple genotypes of the cv. 'Prima' x cv. 'Fiesta' progeny: Class-0 with no resistance factor, Class-R carrying the major resistance gene *Rvi6*, Class-QTL carrying the resistance QTL F11 and F17 and Class-RQTL carrying the pyramiding of *Rvi6* with F11 and F17. Different letters indicate significant differences between disease incidence of the four classes for each year and each site with linear mixed-effect modelling ($p < 0.05$).

1 Table 1. Description of the apple genotypes of cv. ‘Prima’ x cv. ‘Fiesta’ progeny used in the
2 present study.
3

Resistance factors	Class-0	Class-R	Class-QTL	Class-RQTL
<i>Rvi6</i>	- ^a	+ ^b	-	+
F11	-	-	+	+
F17	-	-	+	+
Genotypes ^c	J25	J3	J28	J46
	J32	J43	J63	J52
	J51	J83	J80	J104
	J61	J101	J90	J122
	J108	J103	J115	J158
	J119			
	J150			

4
5 ^a Absence of the resistance factor
6 ^b Presence of the resistance factor
7 ^c Apple progenies of the cross between cv. ‘Prima’ and cv. ‘Fiesta’
8

