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The genetic background modulates the intensity of Rpv3-dependent downy mildew resistance in grapevine

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Abstract

Grape varieties with resistance to downy mildew (DM) carry alien chromosome segments in *Vitis vinifera* backgrounds. We previously showed that the largest descent group shares a non-*vinifera* haplotype at the locus *Rpv3*. Here, we performed a common garden experiment with 76 varieties to evaluate the level of field resistance across four years. All varieties exhibited effector-triggered immunity (ETI)-associated necrosis. On a scale of 1–9, the median OIV452 value for foliar resistance was 7.1 in the resistant lineage vs. 3.2 in *vinifera* controls. Genotype, year and their interaction significantly affected the level of resistance. Some resistant genotypes showed high mean values of OIV452 and low variance among years. Other resistant genotypes showed lower mean OIV452 and higher variance. They were capable of activating ETI, but the intensity was inadequate to restrict pathogen growth under highly conducive conditions. *Rpv3*-dependent responses were stronger in highly native genetic backgrounds and tended to attenuate in late backcross generations. Genetic backgrounds donated by European winegrapes of the convarietas *occidentalis* provided on average higher levels of *Rpv3* resistance than backgrounds of *orientalis* table grapes.

KEYWORDS
breeding, disease, *Plasmopara viticola*, *Vitis vinifera*

1 | INTRODUCTION

Crop wild relatives (CWR) native to North America were widely used in planned hybridizations with the European bunch grape (*Vitis vinifera*). CWR genes improved significantly the plant immune system in new grape varieties (Eibach, Zyprian, Welter, & Töpfer, 2007; Sánchez-Mora et al., 2017; Schwander et al., 2012; Venuti et al., 2013). CWR genes in the *Rpv3* locus confer resistance to *Plasmopara viticola*, the causal agent of grape downy mildew. The *Rpv3* locus is located on chromosome 18 (Bellin et al., 2009; Welter et al., 2007). Since the late 1800s, grape breeders have used the introgression line 'Seibel 4614' to generate a lineage of DM-resistant varieties that share the same *Rpv3* resistance haplotype (Di Gaspero et al., 2012). During this process, the resistance haplotype, initially donated by an unknown North American species, has been transferred into the genetic background of cultivated varieties of *V. vinifera*, including European winegrapes, Caucasian winegrapes and table grapes from the Near East. In segregating populations, the resistance haplotype is necessary and sufficient to trigger a hypersensitive response (HR) in grapevine leaves infected by specific strains of *P. viticola* (Bellin et al., 2009; Zyprian et al., 2016). This resistance haplotype encodes NB-LRR and LRR-kinase receptors (Foria, 2015). According to the ETI model, R gene products act as pathogen sensors and activate signal transduction pathways (Cui, Tsuda, & Parker, 2015). In grapevine, *Rpv3*-dependent resistance follows this model of gene-for-gene interaction (Casagrande, Falginella, Castellarin, Testolin, & Di Gaspero, 2011). In model species, appropriate transducers, receptors, transcription factors, DNA and protein targets, and miRNA-directed phased siRNAs are required for activation of gene expression, ubiquitin-associated protein degradation, and hormone regulation that collectively bring about resistance. We hypothesized that the pace and magnitude of *Rpv3*-dependent defence in grapevine vary with the genetic

background of each introgression line, resulting into different levels of field resistance. In this study, we scored DM foliar resistance, under vineyard conditions, across four consecutive years, in 69 varieties of the 'Seibel 4614' lineage. These varieties have all inherited the same *Rpv3* resistance haplotype from their common ancestor. In spite of this commonality, different numbers of generations separate them from the wild grapevine that originally donated the resistance haplotype, and they are the result of backcrosses to genetically diverse cultivars, including representatives of the convarietas *occidentalis*, *orientalis* and *pontica* (Negrul, 1946). These convarietas evolved separately in Western Europe, in the Caucasus and in the Near East, developing considerable ecogeographical and genetic differentiation. We exploited the existing variation in genetic backgrounds in which the same resistance haplotype has been introgressed, which is the legacy of more than a century of breeding, to produce the first report in a perennial species on the variable efficacy of a conserved R gene in such a wide range of accessions. We also used phenotypic controls from six resistant lineages with North American ancestry different from 'Seibel 4614' (Table 1). An analysis of multilocus haplotype frequencies with *Rpv3*-linked markers has prefigured the existence of additional CWR alleles and their association with DM resistance in those lineages (Di Gaspero et al., 2012). One of these resistance haplotypes—conserved in the 'Munson' lineage—has been recently confirmed by QTL mapping (Zyprian et al., 2016).

2 | MATERIALS AND METHODS

2.1 | Plant material

Grape varieties were sorted according to their genotype at the *Rpv3* locus, following the nomenclature used by Di Gaspero et al. (2012). Haplotypes were named after the size of two hypervariable microsatellites in the locus (Table 1). The set of varieties under investigation comprised 76 genotypes of the 'Seibel 4614' family that share the resistance haplotype $Rpv3^{299\text{-}279}$, 69 of which carry $Rpv3^{299\text{-}279}$ in combination with a *vinifera* haplotype, hereafter referred to as carrying genotype $Rpv3^{299\text{-}279}/Rpv3^{vin}$. The 'Seibel 4614' family tree is illustrated in Di Gaspero et al. (2012). Background information on (i) the country of selection, (ii) the number of generations each variety is removed from 'Seibel 4614' and (iii) the type of *V. vinifera* parental variety used in the last cross-combination is given in Table S1. The set of varieties also included 29 controls, represented by 26 introgression lines with North American ancestry, belonging to resistant lineages different from 'Seibel 4614' and carrying North American haplotypes different from $Rpv3^{299\text{-}279}$, and three varieties of *V. vinifera* (Table S1). The sample of *V. vinifera* included the extremely sensitive 'Kishmish rozovyi', and two varieties with attenuated DM symptoms: 'Mgaloblishvili' (Toffolatti et al., 2016) and 'Marandi Shemakhinskii'.

TABLE 1 Haplotypes and genotypes at the *Rpv3* locus (*n*, number of accessions analysed in this paper). Superscript numbers indicate CWR haplotypes based on the allele sizes of two microsatellites in the locus. "Null" stands for non-amplifying alleles, "NA" indicates collectively conserved haplotypes from North American *Vitis* sp., "vin" indicates collectively *V. vinifera* haplotypes, "unknown" indicates other haplotypes, whose origin could not be predicted from pedigrees, names in brackets indicate CWR *Rpv3* alleles confirmed by QTL mapping, according to the VIVC nomenclature[4]

CWR *Rpv3* haplotype	Founder of descent group	Evidence of DM resistance	*n*
$Rpv3^{299\text{-}279}$ (Rpv3-1)	'Seibel 4614'	QTL mapping[1] and association analysis[2]	76
$Rpv3^{null\text{-}297}$ (Rpv3-2)	'Munson'	Association analysis[2] and QTL mapping[3]	13
$Rpv3^{null\text{-}271}$ (Rpv3-3)	'Noah'	Association analysis[2] and QTL mapping[4]	8
$Rpv3^{321\text{-}312}$	'Noah'	Association analysis[2]	8
$Rpv3^{361\text{-}299}$	'Ganzin'	Association analysis[2]	4
$Rpv3^{null\text{-}287}$	'Bayard'	Association analysis[2]	4
***Rpv3* genotype**	**Grouping in this paper**	**Evidence of DM resistance**	***n***
$Rpv3^{299\text{-}279}/Rpv3^{vin}$	'Seibel 4614' lineage	[5]	69
$Rpv3^{299\text{-}279}/Rpv3^{NA}$	'Seibel 4614' lineage and other NA lineages	[5]	7
$Rpv3^{NA}/Rpv3^{NA}$	Resistant control	[5]	4
$Rpv3^{null\text{-}297}/Rpv3^{vin\ or\ unknown}$	Resistant control	[5]	7
$Rpv3^{321\text{-}312}/Rpv3^{vin\ or\ unknown}$	Resistant control	[5]	6
$Rpv3^{null\text{-}271}/Rpv3^{vin\ or\ unknown}$	Resistant control	[5]	7
$Rpv3^{null\text{-}287}/Rpv3^{vin\ or\ unknown}$	Resistant control	[5]	2
$Rpv3^{vin}/Rpv3^{vin}$	Susceptible control	[5]	3

[1]Welter et al. (2007); Bellin et al. (2009); van Heerden et al. (2014); [2]Di Gaspero et al. (2012); [3]Zyprian et al. (2016); [4]Vitis International Variety Catalogue (VIVC), Table of Loci for Traits in Grapevine, http://www.vivc.de; [5]data from this paper.

2.2 | Field scoring of DM resistance

Phenotypic observations were carried out from 2013 to 2016 in a germplasm repository located in Udine, Italy. The vines were 3 years old at the beginning of this study. The scions were grafted on Kober 5BB and were trellised to a Guyot system. Field assessment of DM resistance was performed in unsprayed plots. Vines were unprotected by agrochemicals at the beginning of every vegetative season from bud burst to the occurrence of the first severe outbreak of DM. As the primary site of DM infection is the leaf lamina, DM resistance was scored as foliar resistance using the OIV452 parameter (IPGRI 1997). Visual scoring of OIV452 is a reliable method for predicting the field level of DM resistance (Calonnec et al., 2013; Vezzulli, Vecchione, Stefanini, & Zulini, 2017; Zyprian et al., 2016). OIV452 scores were taken every year in the latter half of June. Each accession was grown in a plot consisting of five vegetatively propagated vines in a row. For each accession, two evaluators visually inspected all vines and identified the two most heavily infected vines, based on the overall impression of each plant as described by Zyprian et al. (2016). Then, OIV452 scores were assigned, independently by each evaluator, to the most heavily infected leaf for a given plant, following the sampling procedure used by Cadle-Davidson (2008). All observations were made on leaves of primary shoots. Summer laterals from axillary buds had not developed fully expanded leaves by the time of the scoring. OIV452 rating per vine was given as mean value of the evaluators' scores. OIV452 rating per accession was given as mean values of two vines. Then, plots were sprayed until the end of each season to sanitize the vines. The hypothesis of spatial autocorrelation for 4-year mean OIV452 ratings among accessions in the repository vineyard was tested using Moran's *I* statistic and rejected based on *p*-value = .38. A factorial ANOVA was performed with R for the factors genotype, year and their interaction. Statistically significant difference between distributions was assessed using a Wilcoxon test. Climate data were recorded by a weather station located 350 m from the vineyard site.

3 | RESULTS

3.1 | Year-to-year variation in disease pressure

Figure 1 shows seasonal trends of climate parameters. In 2013, May was characterized by a combination of low temperatures and many rain days. June was extremely dry due to an anticipated heat wave. DM oil spots appeared on June 6. DM infection progressed slowly due to high temperatures and low relative humidity (RH). DM scores were taken on June 27, soon after a precipitation that cooled temperatures down for a few days. In 2014, temperatures showed a regular trend. May and June were marked by sparse precipitation. Oil spots were first observed on May 16. Sporulation did not start until June 7 and progressed slowly. The repository vineyard was hit by a hailstorm on June 24, which caused leaf wounding. The latest scoring of DM before the adverse weather event was taken on June 22 in conditions of low disease pressure. The 2015 season was warmer and drier than average. Precipitation was moderate and concentrated in two periods of consecutive rain days. Conditions were conducive to sporulation at the end of June. DM scores were taken on June 29. In 2016, May and June showed a regular trend of increasing temperatures, accompanied by intense precipitation. Oil spots appeared on June 3. The first decade of June was characterized by high RH and heavy rain, conducive to sporulation. DM scores were taken on June 15. Disease pressure varied substantially among the four years. The mean OIV452 value in the entire set of 105 tested varieties, including resistant benchmarks and sensitive controls, was 6.4 in 2013, 7.7 in 2014, 7.1 in 2015 and 6.5 in 2016 (Table S1). The distributions of OIV452 values were significantly different in pairwise comparisons of all years (*p*-value $< .01$ for Wilcoxon test), except for 2013 vs. 2016 (*p*-value = .72).

3.2 | Range of variation of DM resistance in different lineages

All 76 varieties that share the *Rpv3*$^{299\text{-}279}$ resistance haplotype exhibited necrosis underneath foliar lesions, in response to natural infections of *P. viticola*. The median OIV452 value in this lineage was 7.1. The 4-year mean OIV452 value ranged among varieties from 4.7 to 8.6 (Figure 2). Other resistant varieties, unrelated to the 'Seibel 4614' ancestry tree, defined the benchmark of resistance, which ranged from 5.2 to 9.0. The median OIV452 value in these lineages was 8.4. At the same stage of disease progression, varieties of *V. vinifera* showed unrestricted sporulation and no necrosis. In these varieties, the OIV452 interval of susceptibility ranged from 1.6 to 4.4. 'Mgaloblishvili' was the accession of *vinifera* least affected by DM damage.

3.3 | Genotype and year effects

The genotype and the year significantly affected the level of foliar resistance in a subset of 69 varieties that carried the *Rpv3*$^{299\text{-}279}$ resistance haplotype in combination with a *vinifera* haplotype (Tables 2 and S1). Considering that all these accessions share the same R gene and activate an HR, we argue that the source of variation "genotype" likely lies in different genetic factors contributed by the various backgrounds. The effect of the year on the distribution of OIV452 values is shown in Figure 3. For comparison, year-to-year variation in resistance benchmarks and sensitive controls is shown in Figure 4. Factorial ANOVA also detected a significant effect of the genotype x year interaction (Table 2). Varieties with 4-year mean OIV452 values higher than 7.5 were associated with low among years variance (Figure 5). The level of resistance in the varieties was nearly insensitive to year-to-year variation in disease pressure. Varieties with 4-year mean OIV452 scores lower than 6.5 showed higher variance among years.

3.4 | Factors associated with different levels of DM resistance within the 'Seibel 4614' lineage

The highest levels of DM resistance were observed in early backcross generations. We did not detect significant differences in the

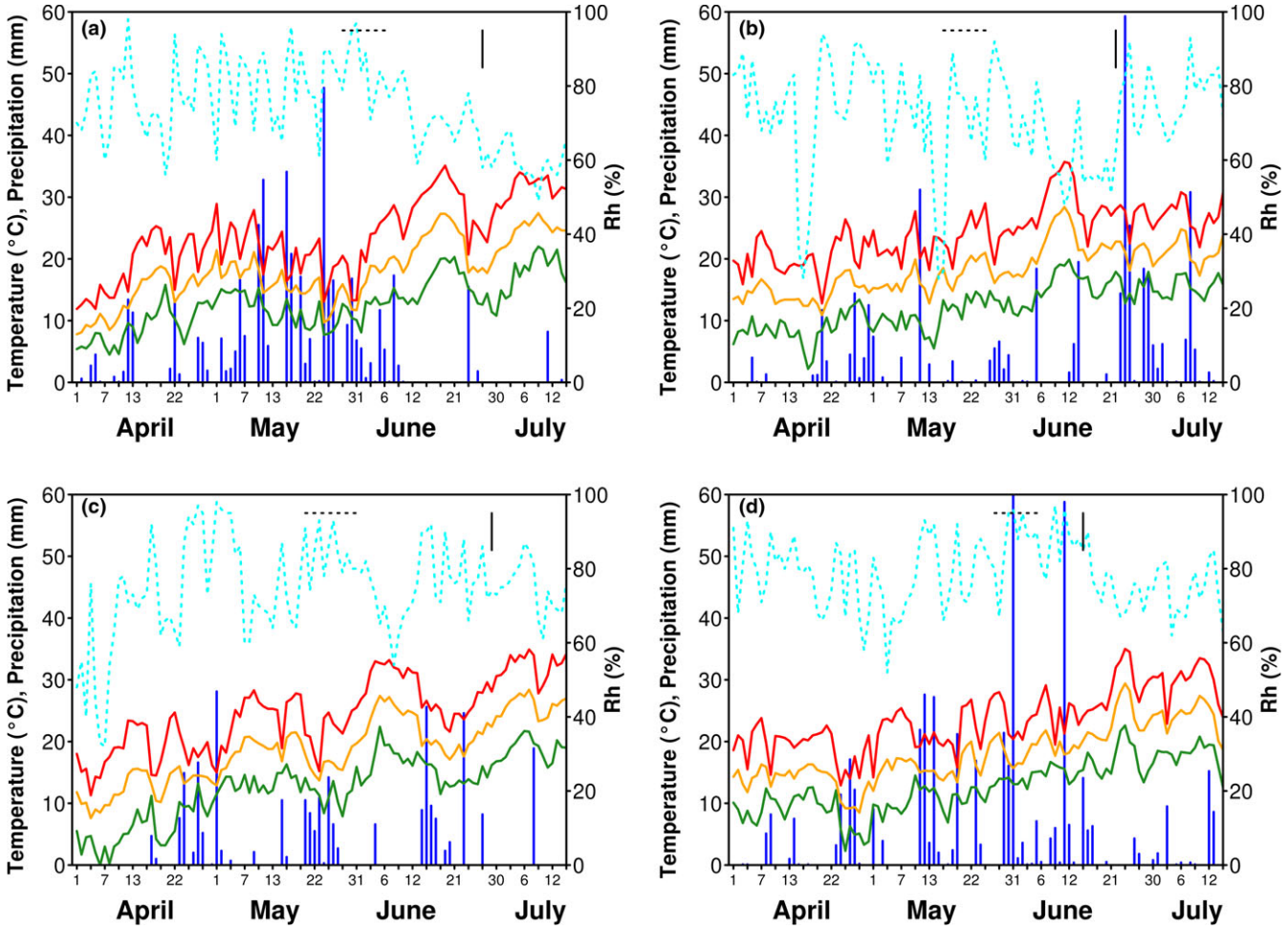

FIGURE 1 Daily rainfall (blue histogram), minimum (green line), average (orange line) and maximum (red line) air temperature, and relative humidity (dashed cyan line) from April 1 through July 15, in 2013 (a), 2014 (b), 2015 (c), 2016 (d). Horizontal dashed lines indicate the interval of first bloom dates. Vertical black bars indicate the day of scoring of foliar resistance [Colour figure can be viewed at wileyonlinelibrary.com]

distributions of OIV452 values in the first three backcross generations obtained from 'Seibel 4614'. A significant drop in OIV452 values was observed between the third and the fourth backcross generations (p-value for Wilcoxon test $2.2e^{-05}$). This stage corresponds to the use of 'Villard Blanc' and its full siblings in cross-combination with many *V. vinifera*, which was a common practice during the 1960s in Austria, Czech Republic, Germany, Hungary, Moldova and Ukraine. In genotypes of the same backcross generation, that is, in progeny of 'Villard Blanc' and 'Eger2' (derived from self-pollination of 'Villard Blanc'), the OIV452 values ranged from 4.7 to 8.1, with some varieties retaining as high resistance as their ancestors. From the fourth to the sixth generation of backcross, we did not detect significant differences in the distributions of OIV452 values (p-values > .05 for Wilcoxon test).

We sorted resistant varieties obtained with three to six backcross generations from 'Seibel 4614' into five categories, based on the type of *V. vinifera* parent used in the last cross-combination. Figure 6 shows the distribution of OIV452 values in progeny that inherited a *V. vinifera* haploid genome from Western Europe, Eastern Europe or Caucasian winegrapes, or from table grapes, or from dual-use grapes. The cross-combinations between resistant introgression lines and Western Europe winegrapes showed a distribution of OIV452 values skewed towards higher resistance than cross-combinations with table grapes (p-value for Wilcoxon test < .01). The cross-combinations between resistant introgression lines and Eastern Europe winegrapes also showed higher OIV452 values than cross-combinations with table grapes with a lower level of significance (p-value for Wilcoxon test < .05). We did not detect significant differences in pairwise comparisons between other parental categories of *V. vinifera*.

4 | DISCUSSION

We showed the conditional effect of the genotype, in the presence of the same R gene, on the intensity of expression of the resistance phenotype in a perennial crop. Among the grape varieties that mount an *Rpv3*-dependent ETI, some genotypes exhibited high resistance under all conditions, while others performed well under low disease pressure but suffered substantial damage with higher disease

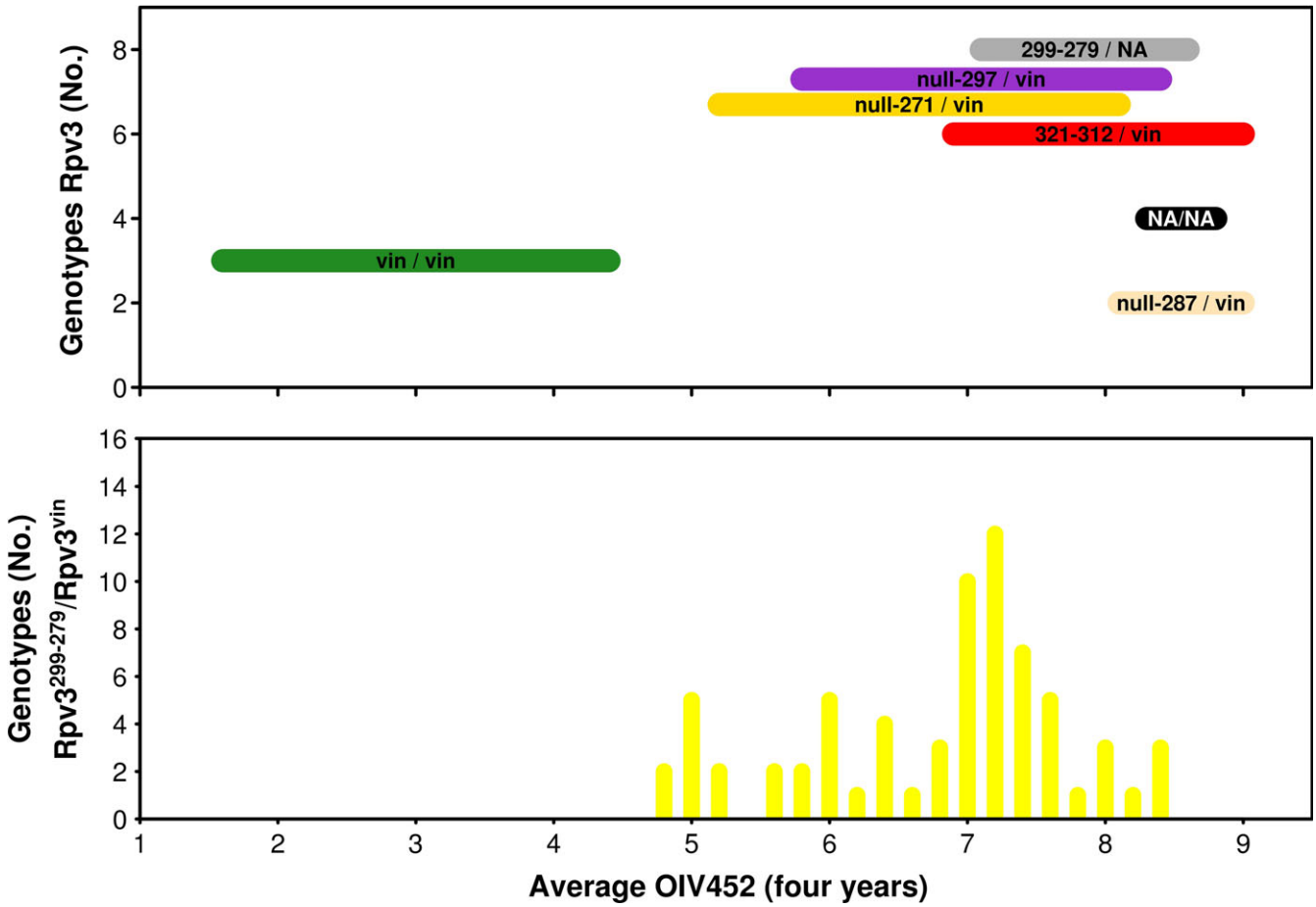

FIGURE 2 Distribution of foliar resistance (average OIV452 values over 4 years) in 105 varieties. In the lower panel, vertical yellow bars indicate OIV452 scores in 69 varieties carrying genotype *Rpv3*$^{299\text{-}279}$/*Rpv3*^{vin}. In the upper panel, horizontal bars show the range of variation for OIV452 scores in other descent groups of resistant varieties and *vinifera* controls. Violet bar indicates varieties sharing the North American haplotype *Rpv3*$^{null\text{-}297}$ donated by 'Munson'. Moccasin bar indicates varieties sharing the North American haplotype *Rpv3*$^{null\text{-}287}$ donated by 'Bayard'. Red or gold bars indicate varieties sharing one of the North American haplotypes donated by 'Noah', either *Rpv3*$^{321\text{-}312}$ (red) or *Rpv3*$^{null\text{-}271}$ (gold). Black bar indicates varieties that carry combinations of conserved North American haplotypes. Grey bar indicates varieties that carry combinations of conserved North American haplotypes, one of which is *Rpv3*$^{299\text{-}279}$. Green bar indicates varieties of *V. vinifera* [Colour figure can be viewed at wileyonlinelibrary.com]

TABLE 2 Analysis of variance for genotype and year effects on foliar resistance in 69 varieties carrying genotype *Rpv3*$^{299\text{-}279}$/*Rpv3*^{vin}

Source of variation	*df*	SS	MS	*F* value	Pr(>*F*)
Year	3	161.3	53.77	213.57	<2e-16 ***
Genotype	68	468.0	6.88	27.34	<2e-16 ***
Genotype × Year	204	310.7	1.52	6.05	<2e-16 ***
Residuals	276	69.5	0.25		

pressure. While maintaining their ability to sense the pathogen and to activate HR, some resistant varieties showed a defence response that was less effective in containing the endophytic growth of the mycelium. A situation of high DM pressure is characterized by high concentration of inoculum, high air temperature and high relative humidity, which are all factors that act in favour of pathogenicity and to the disadvantage of the host defence. In fact, Alonso-Villaverde, Viret, and Gindro (2011) showed that substomatal invasion rates increase with inoculum concentration, possibly due to pathogen-induced deregulation of guard cell functioning, increasing the density of infection sites in the mesophyll. Gessler, Pertot, and Perazzolli (2011) reviewed a large body of evidence in which high temperatures and high relative humidity shortened the incubation time by promoting faster hyphal colonization of the mesophyll. The combination of these factors, leading to more infection sites and faster hyphal growth under highly conducive conditions, may explain lower OIV452 values in resistant varieties with weakened or delayed HR.

We argued that the combination between an R gene for the perception of the pathogen and an appropriate genetic background for the efficacy of the response is critical to obtain high levels of field resistance. This situation may occur in crops more frequently than previously assumed in model species, especially when an R gene is sourced from a distant CWR and transferred into crop germplasm with high genetic differentiation, which is the case study of grapevine. The functionality of *Rpv3* evolved in the cellular context of an

	2013	2014	2015	2016
2013	*	*1,7E-09*	0,055	0,696
2014		*	*4,6E-06*	*5,4E-12*
2015			*	*0,0047*
2016				*

FIGURE 3 Variation of foliar resistance across 4 years in 69 varieties carrying genotype $Rpv3^{299\text{-}279}/Rpv3^{vin}$. Box plot distribution of OIV452 values (panel a) and *p*-values of a Wilcoxon test (panel b) for the distributions shown in panel a

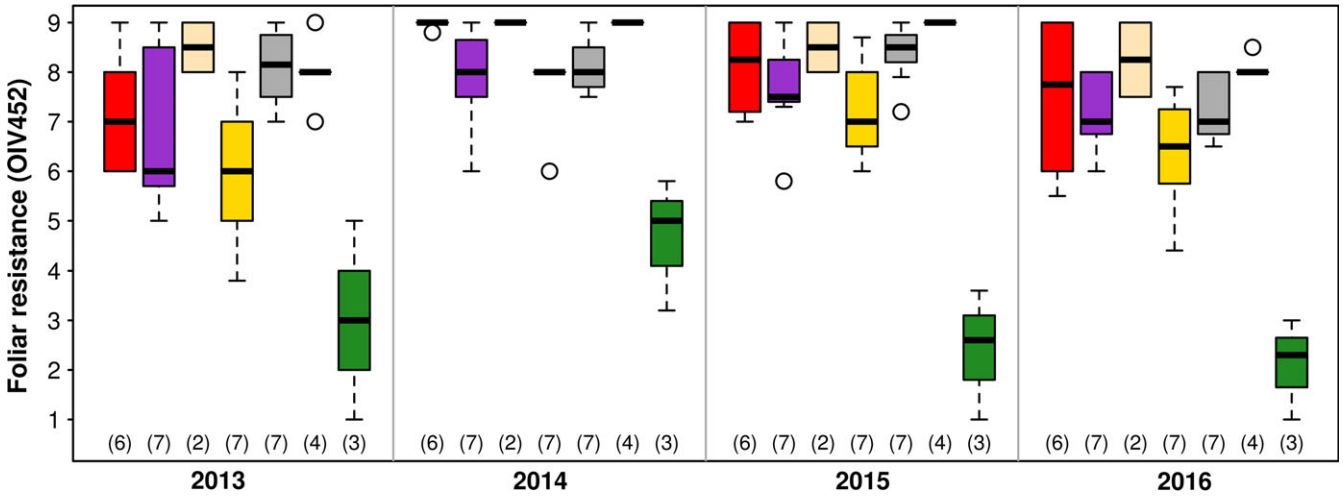

FIGURE 4 Box plot distribution of foliar resistance in different descent groups. Shown are in lane 1 varieties carrying the $Rpv3^{321\text{-}312}$ haplotype from 'Noah'; in lane 2, varieties carrying $Rpv3^{null\text{-}297}$ from 'Munson'; in lane 3, varieties carrying $Rpv3^{null\text{-}287}$ from 'Bayard'; in lane 4, varieties carrying $Rpv3^{null\text{-}271}$ from 'Noah'; in lane 5, varieties carrying $Rpv3^{299\text{-}279}$ in combination with another North American haplotype; in lane 6, varieties carrying combinations of North American haplotypes different from $Rpv3^{299\text{-}279}$; in lane 7, varieties of *V. vinifera*. Numbers in brackets indicate the number of accessions in each descent group [Colour figure can be viewed at wileyonlinelibrary.com]

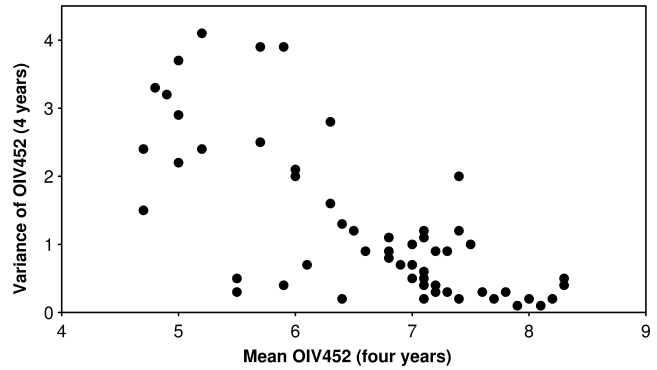

FIGURE 5 Mean–variance relationship for foliar resistance over 4 years in 69 varieties carrying genotype $Rpv3^{299\text{-}279}/Rpv3^{vin}$

unidentified North American species and was introgressed into the European cultivated species, which has diverged from American species for an estimated 11 million years (95% Highest Posterior Density interval 16.58–6.59 million years, Wan et al., 2013). We showed that intensities of *Rpv3*-dependent resistance are higher in wild-type genetic backgrounds and decrease as the number of backcross generations increases. ETI has long been considered a monogenic trait in model species, a notion recently challenged by Iakovidis et al. (2016). These authors showed that cell death in 98 accessions of *Arabidopsis* in the response to a *Pseudomonas syringae* effector has a quantitative nature, exhibiting variation in timing of appearance and strength of the response. Three loci that encode ETI receptors contributed additively to the timing of cell death, and the strong response observed in the accession Bur-0 was attenuated in progeny of Bur-0 with dysfunctional genes for ETI components. Our data suggest that, during the introgression of grape DM resistance in the 'Seibel 4614' lineage, *Rpv3* has been retained as the main initiator of HR, while minor additive ETI loci contributed by the native genetic background and/or wild-type alleles for ETI modulators might have been replaced by less effective alleles. We cannot rule out the possibility that additional factors—contributing to quantitative resistance and not fitting into the ETI model—were initially present in the genetic background of

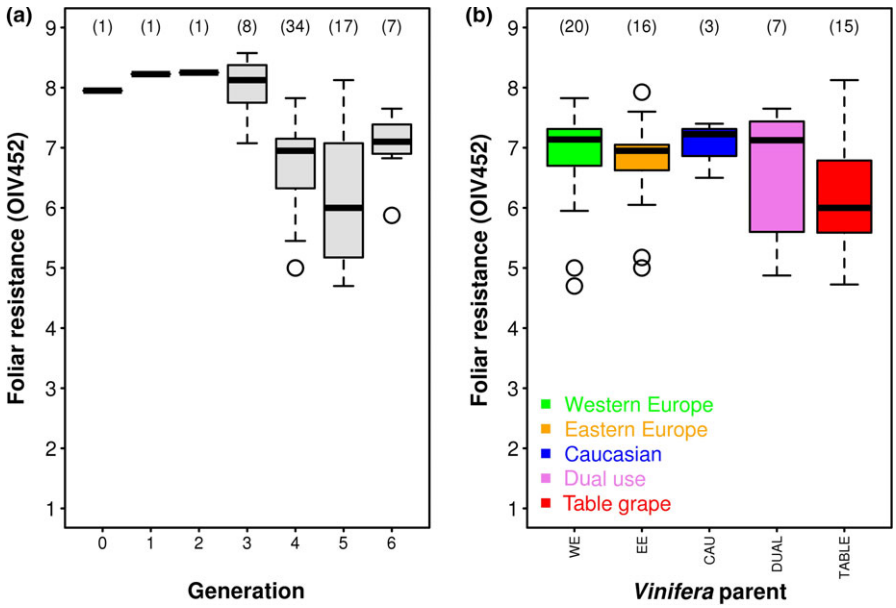

FIGURE 6 Box plot distribution of foliar resistance in the descent group of 'Seibel 4614'. Varieties were sorted by the number of generations that separate them from 'Seibel 4614' (panel a) or by the type of *V. vinifera* parent used in the latest generation of backcross (panel b). Western Europe, Eastern Europe and Caucasian groups include winegrapes sorted by their prevalent area of cultivation. The category "dual use" includes varieties used for both fresh consumption and wine-making. The category "table grape" includes varieties taxonomically classified in the convarietas *orientalis* (Negrul, 1946). Numbers in brackets indicate the number of accessions in each box plot

donor species, that is, *V. riparia* (Marguerit et al., 2009; Moreira et al., 2011) and *V. cinerea* (Ochssner, Hausmann, & Töpfer, 2016), and were progressively lost in the introgression lines.

In grapevine, QTL mapping experiments proved that major R genes activate ETI, explaining from 49% to 78.9% of the observed phenotypic variance. The same experiments did not detect stable QTLs for the remaining genetic component of the phenotypic variance observed in resistant progeny (Bellin et al., 2009; van Heerden, Burger, Vermeulen, & Prins, 2014; Revers et al., 2014; Schwander et al., 2012; Venuti et al., 2013; Welter et al., 2007). We hypothesize that full-sib progeny provide a too narrow range of variation in genetic backgrounds to unleash statistically significant effects and explain the apparent variation in ETI efficacy. Using a wider range of genetic backgrounds, we provided evidence that winegrapes, in particular those belonging to the European convarietas *occidentalis*, donate a genetic background that facilitates the function of *Rpv3* more than table grapes of the convarietas *orientalis*, generating more frequently new varieties with higher resistance.

We provide here a ranked list of introgression lines useful for resistance breeding (Table S1). High OIV452 values indicate parental lines that are more likely to donate appropriate factors for expressing high levels of *Rpv3*-dependent DM resistance in further backcross generations or in intercrosses between different resistant lineages. We speculate that allelic variation for components and modulators of the ETI response, which are associated in other plants with altered expression of thousands of host genes (Lewis et al., 2015), are conditional for the full expression of *Rpv3*-dependent ETI. Similar observations in other crops lend support to this hypothesis. In wheat, the introgression of *Lr22a*-dependent ETI from *Triticum tauschii* to the hexaploid common wheat resulted into lower levels of leaf rust resistance (Pretorius, Rijkenberg, & Wilcoxon, 1990). In rice, a *japonica* background promoted the activation of bacterial-blight ETI more strongly and rapidly than an *indica* background (Zhou et al., 2009). In viticulture, two observations have raised concerns about the deployment of monogenic resistances. First, *Rpv3*-dependent ETI was ineffective in recognizing some pathovariants that have occasionally emerged in European populations of *P. viticola* over the past decades (Peressotti et al., 2010). Second, *P. viticola* collected from partially sporulating ETI lesions had higher aggressiveness, in particular a shorter latency period, than isolates sporulating on leaves of susceptible varieties (Delmas et al., 2016). Enhancing the efficacy of ETI by the introgression of an R gene into an optimal background is important to reduce pathogen propagation from resistant varieties, in order to have greater durability and lower likelihood for the pathogen to adapt. In other crops, monogenic resistances were overcome by local variants of the pathogen more rarely in cultivars that supported stronger ETI expression (Quenouille, Paulhiac, Moury, & Palloix, 2014).

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SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.

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