Polygenic Inheritance of Partial Resistance to *Fusarium oxysporum* f. sp. *melonis* Race 1.2 in Melon

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ABSTRACT

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Fusarium oxysporum f. sp. *melonis* is responsible for Fusarium wilt of melon. Race 1.2 strains overcome two dominant resistance genes (*Fom-1* and *Fom-2*) and are further divided into two types depending on the symptoms they cause, yellowing or wilting. Partial resistance to *F. oxysporum* f. sp. *melonis* race 1.2 was studied by using a recombinant inbred line (RIL) population that was developed by single seed descent from an F₁ hybrid between 'Isabelle', a partially resistant line, and a susceptible line, 'Védraçais'. Artificial inoculations were performed with a yellowing strain (TST) and a wilting strain (D'Oléon 8) and replicated in six

locations. Disease reactions of the parental lines, controls, and RILs were scored using a 1-to-5 scale and by using the area under the disease progress curve (AUDPC). Phenotypic correlations were highly significant between the different locations and experiments. The heritability of the resistance was high, from 0.72 to 0.96, and 4 to 14 genetic factors were estimated to confer resistance to *F. oxysporum* f. sp. *melonis* race 1.2. Thirteen other strains were tested with an RILs subset. Some small strain-specific effects may be involved. These results contribute to a better understanding of the polygenic inheritance of the partial resistance to *F. oxysporum* f. sp. *melonis* race 1.2.

Additional keywords: *Cucumis melo*, disease resistance, soilborne pathogen.

Vascular wilt of melon (*Cucumis melo* L.) caused by *Fusarium oxysporum* Schlechtend. Fr. f. sp. *melonis* W.C. Snyder & H.N. Hans. is an economically important disease worldwide. The characteristic wilt symptoms are provoked by the fungus after it enters the plant's vascular system and rapidly colonizes the host. It is very difficult to control Fusarium wilt because the fungus survives in the soil as chlamydospores during extended periods even in the absence of the host roots. Although long crop rotations are used, *F. oxysporum* f. sp. *melonis* colonizes roots of a broad taxonomic range of plants. *F. oxysporum* f. sp. *melonis* colonizes but does not cause disease on some nonhost crops (11). Some wilt-suppressive soils have been described in France, and the role of nonpathogenic *Fusarium* spp., such as strain Fo47, has been demonstrated (1). Chemicals, such as methyl bromide, are effective in controlling Fusarium wilt but their use will be soon phased out. Acetochlor treatment or dinitroaniline herbicides can reduce disease incidence and this phenomenon may be related to the increase in free amino acid levels in the plant (5,22). More recently, Dong and Cohen (7) showed that dry mycelium of *Penicillium chrysogenum* is a good inducer of resistance against root diseases such as *F. oxysporum* f. sp. *melonis*. Alternative methods to control disease such as grafting onto interspecific hybrids *C. maxima* × *C. moschata*, which are resistant to *F. oxysporum* f. sp. *melonis*, can enhance productivity without negative effects on fruit quality (14). Because this method is expensive and time consuming, it is used only for greenhouse production, but not under field conditions.

Risser et al. (19) defined four races of *F. oxysporum* f. sp. *melonis* (0, 1, 2, and 1.2) based on the interactions between major host resistance genes and variants of the pathogen. Two dominant

resistance genes, *Fom-1* and *Fom-2*, provide resistance to races 0 and 2 and to races 0 and 1, respectively. Race 1.2 overcomes these two resistance genes and is further divided into 1.2y, which induces yellowing symptoms before the death of the plants, and 1.2w, which produces wilting and death without yellowing symptoms. Yellowing symptoms caused by race 1.2y are more commonly observed in France.

Partial resistance to race 1.2 has been found in several Far-Eastern accessions, for instance 'Ogon 9' and 'Kogane Nashi Makuwa' (20). These genotypes allowed breeding of partially resistant lines to race 1.2, such as 'Isabelle', and more recently two doubled-haploid lines, Nad-1 and Nad-2, which show higher resistance than other genotypes (8). This partial resistance is effective on all strains with low aggressiveness whatever their virulence. It is effective against race 1.2 strains, which are not usually very aggressive.

Doubled haploid and recombinant inbred lines (RILs), which are largely homozygous lines and can therefore be immortalized, allow the study of complex resistance because they make it possible to conduct replicated experiments in time and space and use of different strains of the pathogen.

Polygenic resistance to race 2 of Fusarium wilt, *F. oxysporum* f. sp. *niveum*, has been shown in F₁, F₂, and backcross progenies of watermelon (16). Partial and polygenic resistance to basal rot, *F. oxysporum*, has also been observed in four flower bulbs crops, *Lilium*, *Narcissus*, *Gladiolus*, and *Tulipa* (23). Cross et al. (6) studied the inheritance of resistance to race 4 of Fusarium wilt, *F. oxysporum* f. sp. *phaseoli*, in germ plasm of races Durango and Mesoamerica of common bean and showed that this resistance was under polygenic control. Resistance to race 1.2 of *F. oxysporum* f. sp. *melonis* seems to be quantitative but no study about the genetic control of this resistance has been previously published.

The present study reports on the mode of inheritance of resistance to *F. oxysporum* f. sp. *melonis* race 1.2 and estimates

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the number of genes conferring resistance in a segregating RIL population.

MATERIALS AND METHODS

Fungal strains. After a first screening of 15 strains of *F. oxysporum* f. sp. *melonis* race 1.2, strain TST (yellowing) and strain D'Oléon 8 (wilting) were used for the phenotypic evaluations of the RIL population. The 13 other race 1.2 strains were used for inoculations of a subset of melon lines. They were increased from single-spored cultures and stored in sterile agar medium tubes at room temperature. Active cultures were obtained by placing a small aliquot of agar culture in a flask containing 100 ml of nutrient broth (15) and incubating the flask on a rotary shaker for 1 week at 25°C.

Plant material. The breeding line 'Isabelle' was obtained by G. Risser (1978) at the Institut National de la Recherche Agonomique (INRA), Avignon. It was derived from the Chinese accession 'Ogon 9', supplied by M. Iizuka (1963), which is resistant to races 0, 1, and 1.2 of *F. oxysporum* f. sp. *melonis*. The F_1 ('Ogon 9' $\times$ 'Charentais T') was selfed to obtain the F_2 , and then the F_3 generation which was crossed by cv. Doublon (provided by the INRA, resistant to races 0 and 2). 'Isabelle' was obtained after three intercrosses followed by six generations of selfing.

The population used in our study consisted of 120 RILs (F_6 , F_7 , and F_8) derived by single seed descent from a cross between cv. Védrantais (provided by Vilmorin, *Fom-1*) and 'Isabelle' (*Fom-1*, *Fom-2*, and partial resistance to race 1.2). Two commercial F_1 hybrid cultivars partially resistant to *F. oxysporum* f. sp. *melonis* race 1.2 and resistant to races 0, 1, and 2, cv. Manta (provided by Clause-Tezier) and cv. Lunasol (provided by Amélioration des Semences Legumieres [ASL]-Nunhems), were used as controls in the experiments.

Disease evaluations. The 120 RILs, the parental lines (cvs. Védrantais and Isabelle), 'Ogon 9', cv. Manta, and cv. Lunasol were evaluated for resistance to *F. oxysporum* f. sp. *melonis* race 1.2 after artificial inoculation.

They were evaluated independently by five seed companies and the INRA, which were named locations. The D'Oléon 8 strain was used in two locations (A and B) and the TST strain in four locations (C, D, E, and F). In each location, two to six independent experiments were carried out. In each experiment, 10 or 20 plants of each RIL were evaluated in a complete randomized design with one block or two blocks, respectively.

Seedlings were grown in sterilized peat soil mixture in greenhouse conditions (16°C night, 24°C day) for 7 to 10 days until the first true leaves began to emerge. Trays, each containing 50 plants, were dipped into 700-ml suspensions of microconidia (2×10^6 per ml) and were placed in a growth chamber (18°C night, 24°C day, 12-h photoperiod).

The first symptoms appeared between 10 and 14 days after inoculation. As soon as symptoms appeared on a single plant (RIL or control), the severity of symptoms was assessed on infected leaves using a semiquantitative rating scale from 1 to 5. The symptom scale defined for the TST strain was 1 = no symptoms, 2 = yellowing of the cotyledons or the first leaf, 3 = yellowing of two leaves, 4 = yellowing of three or more leaves, and 5 = death of plants. The scale established for the D'Oléon 8 strain was 1 = no symptoms, 2 = wilting of the cotyledons or the first leaf, 3 = wilting of two leaves, 4 = wilting of three or more leaves and browning of the stem, and 5 = death of plants. Plants were examined at 3- to 4-day intervals for 3 weeks after appearance of symptoms.

A subset of 25 RILs, ranked from high susceptibility to partial resistance, were inoculated with 13 race 1.2 strains. For each strain, 30 plants of each RIL were evaluated in a complete randomized design with three blocks. Severity of symptoms was as-

sessed with the same rating scale described previously for 3 weeks. Two or three strains were tested in each location.

Statistical analyses. Four variables were analyzed: the disease scores at the second scoring date (T2), at an intermediate scoring date (TI), and at the final scoring date (TF), and the area under the disease progress curve (AUDPC) was calculated for each RIL using the following formula: $AUDPC = \sum_i [(x_i + x_{i+1})/2](t_{i+1} - t_i)$, with $i = 1$ to 5 or 1 to 6 scorings, x_i = mean disease score of each plant at date i , x_{i+1} = mean disease score of each plant at date $i + 1$, and $t_{i+1} - t_i$ = number of days between scoring date i and scoring date $i + 1$.

Data were analyzed by location ($j = 1$ to 6), by strain (W or Y for the D'Oléon 8 strain or the TST strain, respectively), and for the two strains (WY). Adjusted means of disease scores (lsmeans) of RILs on experiments and blocks (T2j, T2W, T2Y, T2WY, TIj, TIW, TIY, TIWY, TFj, TFW, TFY, and TFWY) and adjusted values of AUDPC of RILs (AUDPCj, AUDPCW, AUDPCY, and AUDPCWY) were estimated by analysis of variance (ANOVA). Phenotypic correlations between the locations, the experiments, and the two strains were calculated. Broad-sense heritabilities (h^2) were estimated for each location and for W, Y, and WY from the mean square (MS) of ANOVA using the formula adapted from Gallais (9). For instance, the formula for location A was $h^2 = \sigma_g^2 / (\sigma_g^2 + (\sigma_{gt}^2/t) + (\sigma_e^2/nt))$, where σ_g^2 is the genetic variance ($MSg - MSgt/nt$), σ_{gt}^2 is the genotype-experiment interaction ($MSgt - MSe/n$), σ_e^2 is the environmental variance (MSe), n is the number of plants, and t is the number of experiments.

The number of segregating genes was estimated by the formula of Snape et al. (21): $k = [(RIL.S - RIL.R)/2]^2 / \sigma_a^2$, where RIL.S is the mean score of the most susceptible line, RIL.R is the mean score of the most resistant line, and σ_a^2 is the additive variance. In other cases, the number of segregating genes was estimated by the formula of Choo and Reinbergs (4): $k' = (\mu - RILmax)^2 / \sigma_a^2$, where RILmax is the mean score of the most extreme (resistant or susceptible) line, μ is the mean of the RIL population, and σ_a^2 is the additive variance.

In an RIL population, dominance variance is absent. This results in a more precise estimate of additive variance by the genetic variance.

Data analyses were performed with Statistical Analysis System (SAS) software (SAS Institute, Cary, NC). Variance analysis of disease scores and AUDPC was performed using PROC GLM of SAS with nested and randomized effects.

RESULTS

Development of symptoms and behavior of the RILs. Distributions of the RILs in classes of disease scores at different dates (T2, TI, and TF) and in classes of AUDPC are shown for only two of the six locations (A and C for the D'Oléon 8 and the TST strains, respectively) (Fig. 1). The parental lines 'Védrantais' and 'Isabelle' were stable, as expected, in susceptibility and resistance, respectively. The controls, cvs. Manta, Lunasol, and Ogon 9, behaved also as expected, among intermediate lines. At T2, most of the lines appeared resistant (location A) or had intermediate reactions shifted toward resistance (location C), but the proportion of lines with intermediate or susceptible behavior increased at TI. At the end of the infection (TF), most of the RILs appeared susceptible (mean disease scores of 4.27 and 3.93 for locations A and C, respectively). With AUDPC, most of the RILs had intermediate reactions shifted toward susceptibility. Similar results were observed in the other four locations.

According to all the locations, one to five RILs seemed to be more resistant than the resistant parental line, 'Isabelle', but only one RIL was significantly different. One to five RILs were significantly more susceptible than cv. Védrantais, showing transgressive segregation.

Correlation between the disease scores and AUDPC, the locations, and the strains. The different disease scores and AUDPC for each location were significantly correlated (Table 1). The correlation was highly significant, from 0.58 to 0.87, between

the six locations not only for AUDPC (Table 2) but also for T2, TI, and TF (data not shown). Diseases scores (T2, TI, and TF) and AUDPC were highly correlated between the two strains TST and D'Oléon 8, ranging from 0.79 to 0.82.

Fig. 1. Distribution of recombinant inbred lines (RILs), from the cross between 'Védrantais' × 'Isabelle' under artificial infection by race 1.2 of *Fusarium oxysporum* f. sp. *melonis*, according to disease scores (T2, TI, and TF) and area under disease progress curve (AUDPC) for locations A and C (D'Oléon 8 and TST strains tested, respectively). The disease scores T2, TI, and TF indicate the disease score at the second scoring date, at the intermediate scoring date, and at the final scoring date, respectively. The mean of the RIL population is m . The genotypes Manta, Ogon9, and Lunasol are the controls.

Genetic analysis of resistance. AUDPC integrates both the intensity of symptoms and time between inoculation and expression of symptoms. Thus, we used AUDPC to explain genetic analysis of resistance.

The distribution of *F. oxysporum* f. sp. *melonis* race 1.2 resistance variables was tested for normality. Distribution of the residuals of AUDPC on bart charts seemed to be normal, but normality tests of Kolmogorov-Smirnov, Cramer-von Mises, and Anderson-Darling were not significant.

Differences in AUDPC among RILs were highly significant (Table 3). The experiment effect was also highly significant. The locations and strain effects were not significant. There were also significant RIL–experiment, RIL–location, and RIL–strain interactions, but their *F* values were much lower than experiment and RILs values. Broad-sense heritabilities calculated from the different locations (AUDPCY, AUDPCW, and AUDPCWY) were between 0.72 and 0.96 (Table 4). The number of genetic factors involved in the RIL population was estimated for AUDPC from 4 to 11 and from 5 to 14 for *k* and *k'*, respectively.

Disease assessment of an RIL subset with other strains. The RIL effect was highly significant (*F* values from 6.5 to 112.17; *P* < 0.0001). The RIL–strain interaction was also significant, with *F* values ranging from 2.10 to 36.33 (*P* < 0.005) according to the locations. The strain effect was also highly significant (*F* = 38.93; *P* < 0.0001) for all the locations. The correlation coefficients were significant between the 15 strains, except for strains 2, 76, and 12449 (Table 5). The *F* values of the RIL effect were significant but low for strains 2 and 76 (*F* values of 6.5 and 8.9, respectively), indicating small differences between the RILs. Thus, these strains were not significantly correlated with the other strains. The RIL effect was high (*F* = 21.2) for strain 12449, but the strain was not significantly correlated with all of the other strains.

DISCUSSION

We aimed to understand the inheritance of resistance to *F. oxysporum* f. sp. *melonis* race 1.2. From our study, we confirm that the *F. oxysporum* f. sp. *melonis* race 1.2 resistance is under polygenic control.

The commercial hybrids, cvs. Manta and Lunasol, are partially resistant to race 1.2 of *F. oxysporum* f. sp. *melonis* in field conditions. These controls behaved as expected in our study, demon-

strating the reliability of the phenotypic evaluations under artificial inoculation. Moreover, indirect evaluations in a growth chamber allowed better control of the disease development and more accurate disease assessment (10).

Disease assessments that were performed with 15 strains showed large differences in aggressiveness. Some strains, for instance strain 71, were so aggressive that neither the resistant RILs nor the parental lines and controls were partially resistant. On the contrary, some strains, for instance strains 2 and 76, were not very aggressive and almost all of the RIL subset and the controls were partially resistant. The most and the least aggressive strains did not allow us to distinguish partially resistant lines and susceptible lines. Among the strains with intermediate aggressiveness, including the TST strain and the D’Oléon 8 strain, the controls behaved as expected and susceptible and resistant RILs could be easily differentiated. These strains are therefore useful to study the inheritance of partial resistance to *F. oxysporum* f. sp. *melonis* race 1.2.

The highly significant correlations between the locations and between the experiments validated the reproducibility of the evaluations. The homogeneous behavior of plants from an RIL and the reproducibility of the evaluations conferred a high herit-

TABLE 3. Variance analysis (ANOVA) of the area under the disease progress curve of race 1.2 of *Fusarium oxysporum* f. sp. *melonis* in 120 recombinant inbred lines derived from the cross between Védrañtais × Isabelle^a

Location	Effect	df	Mean square	F value	P value
A	RIL	119	7,981.90	9.79	<0.0001
	Experiment	5	31,556	38.86	<0.0001
	RIL–experiment	595	818.65	3.95	<0.0001
B	RIL	119	3,152.04	3.60	<0.0001
	Experiment	1	102,210	117.43	<0.0001
	Block(experiment)	2	6,890.03	32.90	<0.0001
	RIL–experiment	119	875.25	4.18	<0.0001
C	RIL	119	10,577	22.56	<0.0001
	Experiment	2	109,921	235.99	<0.0001
	Block(experiment)	3	5,846.18	40.24	<0.0001
	RIL–experiment	238	470.31	3.24	<0.0001
D	RIL	119	4,437.88	4.42	<0.0001
	Experiment	1	240,406	243.25	<0.0001
	Block(experiment)	2	7,701.94	29.43	<0.0001
	RIL–experiment	119	1,005.01	3.84	<0.0001
E	RIL	119	7,316.62	6.10	<0.0001
	Experiment	1	45,703	38.26	<0.0001
	Block(experiment)	2	1,083.00	3.12	0.0442
	RIL–experiment	119	1,198.99	3.45	<0.0001
F	RIL	119	7,457.13	8.29	<0.0001
	Experiment	1	110.87	0.12	0.7258
	Block(experiment)	2	1,195.91	4.56	0.0105
	RIL–experiment	119	899.03	3.43	<0.0001
W	RIL	119	8,266.47	5.85	<0.0001
	Location	1	38,668	4.82	0.0680
	Experiment(location)	6	191,384	162.82	<0.0001
	RIL–location	119	1,413.67	5.95	<0.0001
Y	RIL	119	22,235	18.51	<0.0001
	Location	3	40,725	0.94	0.4520
	Experiment(location)	12	42,860	157.98	<0.0001
	RIL–location	357	1,205.65	4.44	<0.0001
WY	RIL	119	16,133	6.21	<0.0001
	Strain	1	21,405	0.36	0.5605
	Location(strain)	2	124,773	2.13	0.1607
	Experiment(location strain)	12	58,174	257.35	<0.0001
	RIL–strain	119	2,598.01	2.31	<0.0001
	RIL–location(strain)	238	1,127.68	4.99	<0.0001

^a Locations A and B have evaluated the D’Oléon 8 strain. Locations C to F have tested the TST strain. W is the estimate on the two locations in which the D’Oléon8 strain was tested. Y is the estimate on the four locations in which the TST strain was tested. WY is the estimate for the six locations (A to F).

TABLE 1. Correlation coefficients between the adjusted means of the three disease scores (T2, T1, and TF) and the area under disease progress curve (AUDPC) of race 1.2 of *Fusarium oxysporum* f. sp. *melonis* in 120 recombinant inbred lines derived from the cross between Védrañtais × Isabelle^a

Variables	T2	T1	TF
TI	0.97	...	...
TF	0.84	0.91	...
AUDPC	0.92	0.99	0.97

^a The three disease scores T2, T1, and TF are the disease score at the second scoring date, at the intermediate scoring date and at the final scoring date, respectively. All the correlation coefficients are highly significant (*P* < 0.001).

TABLE 2. Correlation coefficients between the adjusted means of the area under disease progress curve of race 1.2 of *Fusarium oxysporum* f. sp. *melonis* in the 120 recombinant inbred lines derived from the cross between Védrañtais × Isabelle in six locations (locations A to F)^a

Locations	A	B	C	D	E
B	0.76	...	...	...	...
C	0.68	0.70	...	...	...
D	0.63	0.61	0.87	...	...
E	0.60	0.58	0.83	0.79	...
F	0.66	0.62	0.87	0.82	0.76

^a All the correlation coefficients are highly significant (*P* < 0.001).

ability to AUDPC and disease scores (data not shown). These high heritability estimates also indicated that the variations observed are mostly genetically controlled and that the estimation of resistance by phenotypic evaluations is reliable. Interactions between genotype and environment can reduce the reliability of estimates of genetic variance and its components and can reduce the accuracy of selecting genotypes based on their phenotypes (12). In our study, residual effects and variation coefficients showed good estimation of the genotype by the phenotypic evaluations. The distribution of the resistance of the RILs to *F. oxysporum* f. sp. *melonis* race 1.2 is continuous between the two parents, which suggests resistance is under polygenic control.

Quantitative resistance is usually controlled by several genes. The segregation of quantitative resistance to *F. oxysporum* f. sp. *melonis* race 1.2 in our RIL population enabled us to estimate the number of genes involved in the resistance. Between 4 and 14 genes (4 to 11 and 5 to 14 for k and k' , respectively) control resistance to *F. oxysporum* f. sp. *melonis* race 1.2. For one location, more factors (from 9 to 14 for k and k' , respectively) are involved in the resistance because of the severity of the disease assessment, the low variability among the RILs, and consequently the low additive variance. The more severe the disease assessments are, the more genetic factors are estimated. The methods of Choo and Reinbergs (4) and Snape et al. (21) assume that gene action is additive, that linkage and epistasis are absent, that gene effect is equal at each locus, and that one or both extreme lines in the population is present. Deviations from these assumptions lead to an underestimation of the number of genetic factors. An RIL population is an adequate additive genetical model. The biggest limitation in the accuracy of estimation is the use of a finite sample that may not contain the extreme genotypes (21). Choo and Reinbergs (4), by using the deviation of the most extreme line from the mean of the population, improved the probability that one of these lines is the most extreme genotype. The sample size in this study is 120 RILs, which gives 98% probability that the best recombinant is included in the population when the resistance is controlled by six or fewer genes (4). Because few transgressions of RILs toward susceptibility are observed, the estimation

is made more accurate by using the deviation between the two extreme lines (k estimates). However, underestimation of the true number of genes remains a strong possibility. Epistatic effects would inflate the additive variance, and consequently k and k' would be underestimated. The hypothesis of equality of gene effects at individual loci is also probably false. Another assumption of these models is that all the alleles for resistance come from the resistant parent. The observation of transgressions implies that the susceptible parent, cv. Védraçais, may also have alleles for resistance. Minor effects could not be detected by these models and the number of genes is probably underestimated.

Quantitative, polygenic resistance is usually thought to be race nonspecific. However, polygenic resistance to specialized pathogens often shows small race-specific effects. In host-pathogen systems like potato/*Phytophthora infestans* (13), pepper/*Potyvirus* (3), barley/*Puccinia hordei* (18), and barley/*Pyrenophora graminea* (2) clear isolate specificity of quantitative trait loci (QTL) has been observed. These isolate-specific QTL support the assumption of Parlevliet and Zadoks (17) that partial resistance may be due to minor gene-for-gene interactions. However, race-specific effects are often so small that only in more refined experiments may one hope to discern them from the experimental error (17). The number of WY genetic factors (11 to 13) is higher than for W (7 to 10) or Y (6 to 7), thus some strain-specific effects may be estimated for WY. It was also noticed that the correlations between locations (Table 2) using the same strain (A and B or C, D, E, and F) (0.76 to 0.87) are higher than correlations between locations using the two different strains (0.58 to 0.70). Moreover, the AUDPC of the subset of 25 RILs evaluated with 15 strains showed significant RIL-strain interaction. Thus, some strain-specific effects may be involved in the partial resistance to *F. oxysporum* f. sp. *melonis* race 1.2. However, it seems there is no symptom-associated (wilting versus yellowing) effect.

In order to design efficient breeding strategies for increasing the level of resistance in melon cultivars, the genetic variance, the heritability, and the estimated number of genes involved in the resistance to *F. oxysporum* f. sp. *melonis* race 1.2 were investigated in this study. Phenotypic selection is possible but will be

TABLE 4. Genetic analysis of the resistance to race 1.2 of *Fusarium oxysporum* f. sp. *melonis* in the recombinant inbred line progeny^a

Parameters	A	B	C	D	E	F	W	Y	WY
h^2	0.90	0.72	0.96	0.88	0.84	0.77	0.83	0.94	0.89
k	5.72	9.09	4.20	4.73	5.94	7.01	6.46	5.57	10.83
k'	7.69	14.31	5.01	6.75	8.00	8.51	9.84	7.14	12.99

^a Heritability and estimated number of genetic factors were calculated for the area under disease progress curve (AUDPC) of the six locations conducted with *F. oxysporum* f. sp. *melonis* race 1.2 strains, TST (Y for locations C, D, E, and F) and D'Oléon 8 (W for locations A and B), and for AUDPCW, AUDPCY, and AUDPCWY. h^2 is the broad-sense heritability, k and k' are the estimated number of segregating factors according to Snape et al. (21) and Choo and Reinbergs (4), respectively.

TABLE 5. Correlation coefficients between the adjusted means of the area under disease progress curve (AUDPC) of *Fusarium oxysporum* f. sp. *melonis* race 1.2 strains in a subset of 25 recombinant inbred lines derived from the cross between Védraçais × Isabelle^a

Strains	71 w	D'Oléon 8 w	2 w	76 w	88 y	2002 y	2001 y	107010 y	876 y	58 y	TST y	45 y	42 y	98008 y
D'Oléon 8 w	0.76**	...	...	...	...	...	...	...	...	...	...	...	...	...
2 w	-0.15	-0.17	...	...	...	...	...	...	...	...	...	...	...	...
76 w	0.31*	0.40**	0.33**	...	...	...	...	...	...	...	...	...	...	...
88 y	0.78**	0.80**	-0.09	0.39**	...	...	...	...	...	...	...	...	...	...
2002 y	0.50**	0.80**	-0.35	0.48**	0.63**	...	...	...	...	...	...	...	...	...
2001 y	0.44**	0.71**	-0.31	0.49**	0.59**	0.92**	...	...	...	...	...	...	...	...
107010 y	0.44**	0.72**	-0.11	0.42**	0.75**	0.78**	0.83**	...	...	...	...	...	...	...
876 y	0.41**	0.65**	-0.31	0.40**	0.63**	0.76**	0.84**	0.82**	...	...	...	...	...	...
58 y	0.46**	0.74**	-0.28	0.40**	0.79**	0.84**	0.85**	0.91**	0.86**	...	...	...	...	...
TST y	0.52**	0.84**	-0.27	0.45**	0.81**	0.91**	0.89**	0.92**	0.85**	0.95**	...	...	...	...
45 y	0.49**	0.75**	-0.31	0.30*	0.75**	0.77**	0.80**	0.82**	0.83**	0.88**	0.88**	...	...	...
42 y	0.55**	0.80**	-0.28	0.17	0.64**	0.68**	0.66**	0.67**	0.69**	0.78**	0.77**	0.82**	...	...
98008 y	0.51**	0.79**	-0.14	0.64**	0.70**	0.90**	0.90**	0.87**	0.86**	0.89**	0.92**	0.80**	0.63**	...
12449 y	0.27*	0.30*	0.12	0.41**	0.48**	0.2	0.29*	0.38**	0.49**	0.30*	0.34**	0.32**	0.08	0.46**

^a Wilting strains are indicated by w and yellowing strains are indicated by y. All the correlation coefficients that are significant are indicated by ** ($P < 0.001$) and * ($P < 0.01$).

difficult at the individual plant level in segregating progenies such as F₂. Research to find molecular markers linked to *F. oxysporum* f. sp. *melonis* race 1.2 is now being undertaken in our laboratory and will allow breeders to create resistant cultivars more efficiently through marker-assisted selection.

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