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A new view on aphid resistance in melon: the role of *Aphis gossypii* variability

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Abstract

Resistance to *Aphis gossypii* in *Cucumis melo* has been largely studied but *A. gossypii* variability has never been considered. Resistance to colonization by *A. gossypii*, clone NM1, and to non persistent virus transmission by this clone is conferred by a NBS-LRR gene called *Vat*, isolated in the PI 161375 accession. We investigated resistance to *A. gossypii* with four clones of *A. gossypii* that belong to two very distinct genotypes, NM1 and C9. The *Vat* gene conferred a high level of resistance to a NM1 clone and partial resistance to a C9 clone. Four additive QTL and two pairs of epistatic QTL were detected in a recombinant inbred line population derived from the cross 'Védrentais' x PI 161375. Half of them clearly have a specific effect on the acceptance by NM1 or C9 genotypes. We observed transgressive lines more resistant to NM1 or C9 in our RIL populations than PI 161375. Moreover, we studied resistance to *A. gossypii* and to virus transmission by *A. gossypii* in a set of 21 *C. melo* accessions. All the accessions resistant to virus transmission by C9 *A. gossypii* were resistant to virus transmission by NM1 genotype, and most of them were resistant to acceptance and colonization by the NM1 genotype. This is the phenotype of PI 161375, and we hypothesized that these phenotypes were controlled by the *Vat* gene. Other phenotypes were observed: resistance to NM1 *A. gossypii* (aphids and virus transmission) and susceptibility to C9 *A. gossypii* (aphids and virus transmission), independence between aphid resistance and resistance to virus transmission when using the same clone of *A. gossypii*. These phenotypes may be conferred by the locus *Vat* (different alleles) or by other locus of aphid resistance.

INTRODUCTION

Colonization of Cucurbits by aphids causes stunting and severe leaf curling and can result in plant death. Aphids may also excrete honeydew on the leaves and the fruits, which serves as a growth medium for sooty mould. Moreover, they are efficient virus vectors and thus contribute to spread of diseases. *A. gossypii* Glover is the only species of aphid colonizing melon. Intensive use of insecticides to control aphids in Cucurbits culture has led to emergence of resistant clones of *A. gossypii* (Delorme et al. 1997). Development and cultivation of aphid resistant varieties should be one of the principal means of non-chemical control of pests in melon.

A. gossypii resistant melon accessions have been largely described since the 1970s, particularly the Indian and Korean accessions PI 414723 and PI 161375

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(Kishaba et al. 1971; Bohn et al. 1972; Lecoq et al. 1979; Pitrat and Lecoq 1980). On both accessions, aphids quickly escape the plants, have a low biotic potential and do not transmit non-persistent viruses. This phenotype is controlled by a gene called *Vat* (Pitrat and Lecoq 1982). This gene belongs to the NBS-LRR gene family (Pauquet et al. 2004). Even though several other resistant accessions have been described (Bohn et al. 1973; Pitrat and Lecoq 1980; Pitrat et al. 1988; Bohn et al. 1996; Soria et al. 2000), inheritance of resistance has not been established for all the sources and up to now, no resistance locus other than *Vat* has been clearly identified.

Since the end of the 1990s, molecular markers have been developed to characterized *A. gossypii* strains (Vanlerberghe-Masutti et al. 1999). They have allowed the description of a host races organization of the species (Vanlerberghe-Masutti and Chavigny 1998; Charaabi et al. 2008). Eight major genotypes are known on Cucurbits, the genotype C9 is found worldwide, as the genotype NM1 is restricted to the Southeast of France (Vanlerberghe-Masutti, comm. pers.). As early as 1971, Kishaba et al. (1971) pointed out that the resistance in melon to the US southeastern biotype of aphids was inefficient against the southwestern biotype. MacCarter and Habeck (1974) observed the opposite case. In the same way, Soria et al. (2000) observed low resistance levels to *A. gossypii* from Spain in accessions that presented a high level of resistance to French *A. gossypii*.

In this study, we looked at aphid resistance in melon with regard to *A. gossypii* variability. We used clones of NM1 and C9 genotypes, on one hand to study quantitative resistance in a recombinant inbred line population derived from a 'Védrantais' x PI 161375 cross, and on the other hand, to study qualitative resistance in a set of 21 accessions.

MATERIALS AND METHODS

Synchronous mass rearings of *A. gossypii* were conducted on melon 'Védrantais' at 24: 18°C under a 16h: 8h photoperiod. Four clones (collected on Cucurbits) were used: NM1-lab and 4-106, both from Southeast of France having a NM1 genotype, and Sudan (originating from Sudan) and 4-104 (originating from the Southeast of France) having a C9 genotype. Five-seven days-old aphids were used to infest plantlets at two-leaf stage for resistance biotests conducted at 24:18°C under a 16h: 8h photoperiod.

Characterization of resistance in a recombinant inbred line population.

One hundred thirty-eight recombinant inbred lines (RILs), derived from a 'Védrantais' x PI 161375 cross, were assessed for the acceptance and the biotic potential of *A. gossypii*. Acceptance was estimated by the number of aphids remaining on a plantlet 48h after inoculation by 10 aphids (n=8-15 per RIL). Biotic potential was estimated by recording 2 life-history parameters (n=4-17 per RIL): the duration of pre-reproductive period and the number of progenies produced during a period as long as the pre-reproductive period. A few adults were caged for nymph production on a leaf on day x . The following day, these adults were removed and a few nymphs, deposited by these adults, were kept in the cage. These nymphs reached the adult stage at the day $x+d$, when they produced nymphs. At this time, one newly emerged adult was kept in the cage and its progeny was counted during d days. The duration of the pre-reproductive period, d , and the progeny produced by one female

during d , P , allowed estimation of the intrinsic rate of increase according to Wyatt and White (1977): $r_m = 0.738 \ln(P/d)$.

Characterization of resistance in accessions

Twenty-two accessions were assessed for their resistance to *A. gossypii* and to virus transmission by *A. gossypii*.

To assess resistance to *A. gossypii*, 10 adults were deposited on a plantlet. Seventy-two days later, the number of aphids remaining on the plantlet was recorded as Acceptance parameter. Seven days after inoculation, the adults were counted and the density of larvae was estimated with a 0-6 scale. Colonization at 7 days was calculated as Colonization = density of larvae + $\ln(\text{number of adults} + 0.001)$. The Acceptance and Colonization parameters were collected on 8-30 plantlets of each accession. The susceptible controls consisted of 8 to 10 plantlets of 'Védrantais' and the resistant control consisted of 8 to 10 plantlets of 'Margot' in each test (Charentais type line with aphid resistance introgressed from PI 161375). Kruskal and Wallis non parametric tests and multiple comparisons, described by Siegel and Castellan (1998), were applied to each accession and both controls and allowed to class each accession as Accession identical to 'Margot', Accession identical to 'Védrantais' or Accession intermediate between 'Margot' and 'Védrantais'.

To assess resistance to virus transmission by *A. gossypii*, aphids from mass rearings were transferred to CMV (isolate I17F) -infected leaves of zucchini 'Diamant' for 10 min virus acquisition. Batches of 10 aphids were deposited on plantlets for inoculation. After 15 min, the aphids were removed, and the plants treated with pyrimicarb (NM1 genotypes) or endosulfan (C9 genotypes) and placed into an insect proof glasshouse. The occurrence of transmission was determined 20 days after inoculation by visual assessment of symptoms. The susceptible control consisted of 8 to 10 plantlets of 'Védrantais' and the resistant control consisted of 8 to 10 plantlets of 'Margot' in each test. Nine to 40 plantlets were tested for each accession. The proportion of infected and symptomless plantlets of each accession was compared to the proportion of infected and symptomless plantlets of susceptible and resistant controls using Fisher's exact test. Because there were two comparisons per accession, p was fixed at 0.025 for significant differences. Then each accession was classed as Accession identical to 'Margot', Accession identical to 'Védrantais' or Accession intermediate between 'Margot' and 'Védrantais'.

Molecular characteristics of 'Védrantais' x PI 161375 map

The genetic map used in this study was built using the map produced by Périn et al. 2002 as a base with the addition of SSR markers. One hundred and ninety recombinant inbred lines were genotyped with 165 SSR markers, 99 AFLP markers, 13 InterSSR, 4 phenotypic markers, 2 PCR specific markers, 1 RFLP and 1 RAPD markers. The map consisted in 12 linkage groups and covered 1326 cM according to the 285 markers used. The median distance between markers was 3.8 cM (2.3 for the first quartile and 6.3 for the third quartile).

The additive QTL were detected using QTL cartographer software (Basten et al. 1997) with the composite interval mapping procedure using 5 cofactors. The thresholds of significant LOD scores ($p=0.05$) were fixed after 5000 permutations. The epistatic QTL were detected using the ANOVA procedure of S-Plus software.

The thresholds of significant p was fixed at 5 % corrected for the Bonferroni effect of multiple analyses, $p = 0.05 / (285 * 284) / 2 = 1.2 \cdot 10^{-6}$.

RESULTS

QTL of resistance to *A. gossypii* in a recombinant inbred line population

The acceptance by NM1lab clone (NM1 genotype) and 4-104 clone (C9 genotype) was observed on 'Védrantais', PI 161375, the F₁ and 138 RILs derived from the cross 'Védrantais' x PI 161375 (Tab. 1). The acceptance by NM1 was higher than the acceptance by C9 on 'Védrantais' (Mann-Whitney, $p=0.047$). In contrast, the acceptance by C9 was higher than the acceptance by NM1 on PI 161375 (Mann-Whitney, $p<0.01$). The acceptance was intermediate between the parents by both clones of *A. gossypii* on the F₁ and the range of the RIL population went beyond the parents. The biotic potential parameters were only observed with the NM1-lab clone on 'Védrantais', PI 161375, the F₁ and 138 RIL derived from the cross 'Védrantais' x PI 161375 (Tab. 1). The biotic parameters of *A. gossypii* observed on the F₁ were similar to those observed on the resistant parent. The range of biotic parameters in the RIL population went beyond the parents parameters.

Table 1. Phenotype of 'Védrantais', PI 161375, the F₁ (mean \pm CI 95 %) and the 138 RIL for the acceptance by *A. gossypii* NM1 and 4-104 (aphids remaining 48h after infestation by 10 aphids) and the biotic potential of NM1 (*d*: duration of pre-reproductive period, *P*: progenies produced by one female during *d*, *r_m*: intrinsic rate of increase).

	Acceptance		Biotic parameters of NM1-lab		
	NM1-lab	4-104	<i>d</i>	<i>P</i>	<i>r_m</i>
PI 161375	2.6 \pm 1.0	4.9 \pm 0.8	6.7 \pm 0.2	19.7 \pm 11.9	0.33
Védrantais	8.3 \pm 1.0	7.1 \pm 0.4	5.8 \pm 0.2	40.3 \pm 12.7	0.47
F ₁	5.4 \pm 1.7	5.8 \pm 0.7	6.7 \pm 1.4	8.4 \pm 7.6	0.23
Range of 138 RIL	2.3 - 8.5	1.8 - 9.6	5.2 - 8.4	3.8 - 68.6	0.13 - 0.57

One major QTL and two minor QTL had additive effect on the acceptance (Tab. 2). The major QTL colocalized with the *Vat* locus. Its effect on the acceptance was stronger for the NM1 clone ($r^2=75\%$) than for the C9 clone ($r^2=61\%$). Both minor QTL only had significant effect on the acceptance by C9 clone. One major QTL and two minor QTL had additive effect on biotic potential (Tab. 2). The major QTL colocalized with the *Vat* locus. Its effect on biotic potential was 65 % on the pre-reproductive period *d*, 49 % on the progenies produced by one female during *d*, *P* and 58 % on the intrinsic rate of increase, *r_m*. The effects of the *Vat* locus were weaker on the biotic potential than on the acceptance by the NM1 clone. One minor QTL, located on the linkage group IV increased *d*. The second minor QTL, located on the linkage group VIII decreased *P* and *r_m*. Minor QTL had their resistance alleles originating either from PI 161375 or from 'Védrantais'. Two pairs of epistatic QTL were detected, one reducing the acceptance by the NM1 clone, the other one reducing the biotic potential of NM1 clone. This last one also had a less significant effect on the acceptance by both clones.

Table 2. QTLs reducing acceptance and biotic potential of *A. gossypii* NM1-lab and 4-104 with i) additive effect significant at $p < 0.05$ (Composite interval mapping) ii) epistatic effect significant at $p < 0.05$ corrected for Bonferroni effect (ANOVA).

	Linkage group and localization ^z	Allele of resistance	Effect (r^2) ^y on		
			Acceptance		Biotic potential
			NM1-lab	4-104	NM1-lab
Additive	V – 81-87 cM	PI ^w	75 %	61 %	49-65 %
	VI – 9-28 cM	Véd ^w		4 %	
	IX – 33-53 cM	PI ^w	(3 %) ^x	4.5 %	
	IV – 45-51 cM	Véd ^w			6 %
	VIII – 78-88 cM	Véd ^w			6 %
Epistatic	VII (15 cM) / XI (54 cM)	Trans	20 %		
Epistatic	VII (140 cM) / XII (32 cM)	Cis	(22 %) ^x	(19 %) ^x	28 %

^z (IC±1 LOD unit for additive QTL)

^y r^2 after composite interval mapping for additive QTL and after ANOVA for epistatic QTL

^x (%) Significant at $p < 0.1$

^w PI=PI 161375 (resistant), Véd=Védrantais (susceptible)

Characterization of resistance to *A. gossypii* in 21 accessions of *C. melo* (Tab. 3)

Fourteen accessions were infested with *A. gossypii* clones NM1-lab and 4-106, both belonging to the NM1 genotype and with Sudan and 4-104, both belonging to the C9 genotype. Eight more accessions were only infested with the NM1-lab and 4-104 clones. On the susceptible control ‘Védrantais’, no significant clone effect was observed for acceptance (average 7.5) and colonization (average 7.5). ‘Védrantais’ was scored S (susceptible) to all the aphids clones. On the resistant control, ‘Margot’, we observed a significant effect of the genotype of *A. gossypii*. ‘Margot’ was resistant to both NM1 clones, nevertheless the clone 4-106 was more aggressive than the clone NM1-lab (NM1-lab acceptance and colonization were 1.8 and 0; 4-106 acceptance and colonization were 3.5 and 2.2). Then ‘Margot’ was scored R (resistant) to NM1 and IR to 4-106 aphids. ‘Margot’ was partially resistant to both C9 clones. Both C9 clones presented the same aggressiveness (C9 acceptance and colonization were 4.1 and 5.4). Then ‘Margot’ was scored I (intermediate) to 4-104 and Sudan aphids. According to acceptance and colonization parameters recorded, the 21 accessions were compared to ‘Margot’ and ‘Védrantais’ and then scored as R, IR, I, IS and S.

We inoculated 21 accessions with CMV using the clones NM1-lab (NM1 genotype) and 4-104 (C9 genotype). Previously to this study, we checked that all the accessions tested were susceptible to CMV, isolate I17F, when mechanically inoculated. Throughout all the biotests, the transmission rates observed on ‘Védrantais’ (the susceptible control) were 93.7 % with the NM1-lab clone and 81.2 % with the 4-104 clone. ‘Védrantais’ was scored as S to both clones. The transmission rates observed on ‘Margot’ (the resistant control) were 0.5 % with the NM1lab clone and 0 % with the 4-104 clone. ‘Margot’ was scored as R to both clones. Fisher’s exact test allowed comparison of the transmission rate of each accession with the transmission rates of

the susceptible and resistant control ('Védrantais' and 'Margot') and then were scored as R (Resistant), I (Intermediate) or S (Susceptible).

Table 3 Phenotype of 21 accessions infested with 4 clones of *A. gossypii*, 4-106 and NM1-lab with NM1 genotype and Sudan and 4-104 with C9 genotype. The class for aphid resistance was fixed in comparison with susceptible and resistant control (non parametric test, observation 2 and 7 days after infestation by 10 aphids). The class for resistance to non persistent virus transmission was fixed in comparison with susceptible and resistant control (Fisher exact test, inoculation of CMV by 10 aphids, symptoms assessment).

Response to	NM1 genotype			C9 genotype		
	To aphids		To virus transmission	To aphids		To virus transmission
	4-106	NM1-lab	NM1-lab	Sudan	4-104	4-104
Accessions						
Margot ^z	IR	R	R	I	I	R
PI 161375	IR	I	R	I	I	R
Ginsen Makuwa	IR	R	R	I	I	R
PI 266935	IR	R	R	I	I	R
PI 414723	IR	R	R	I	I	R
PI 482420	IR	R	R	I	I	R
Chenggam	IR	R	R	IS	I	R
Durgapura Madhu	IR	R	R	IS	I	R
Miel Blanc	IR	R	R	S	IS	R
Kanro Makuwa		R	R		IR	R
K5442		R	R		I	R
PI 255478		R	I		I	R
Meloncillo		IR	R		IS	R
PI 164320		R	I		IS	I
PI 164323		S	R		IS	I
90625	IR	R	R	IS	S	S
Anso 77	IR	R	R	IS	I	S
PI 164723	IR	R	R	IS	S	S
PI 224770	IR	R	R	I	S	S
Fegouss 1	I	R	R	IS	S	S
PI 282448	IR	IR	S	IR	IR	S
Escrito 8429		IR	S		IS	S
Védrantais ^y	S	S	S	S	S	S

^zResistant control, ^ySusceptible control

Twenty accessions were resistant (partially or highly) to acceptance and colonization by the NM1-lab aphids. Among those, 14 were also infested with the clone 4-106 (NM1 genotype) and all exhibited resistance to the 4-106 clone, but as with 'Margot', the clone 4-106 generally appeared more aggressive than the clone NM1. Among the 21 accessions resistant to acceptance and colonization by both NM1 clones 16 were also resistant to CMV transmission by NM1-lab clone, PI 255478 and PI 164320, exhibiting only partial resistance to virus transmission and

‘Escrito 8429’ and PI 282448 were susceptible to CMV after inoculation by the NM1-lab clone. PI 164323 was susceptible to acceptance and colonization by the NM1-lab clone, but exhibited resistance to CMV transmission by this clone.

Ten accessions were as resistant as ‘Margot’ to acceptance and colonization by the 4-104 (C9 genotype). Eight among these 10 accessions were also inoculated by the Sudan clone; they exhibited partial resistance or slight susceptibility to this clone. Only two accessions (‘Kanro makuwa’ and PI 282448) exhibited higher resistance to acceptance and colonization by the C9 clones than ‘Margot’. PI 282448 was also resistant to the Sudan clone (genotype C9). Among these 12 accessions we observed either virus transmission resistance (10 accessions) or virus transmission susceptibility (two accessions). Nine accessions were susceptible to acceptance and colonization by the C9 clones, among them, two were resistant to virus transmission by the clone 4-104, two were partially resistant to virus transmission by the clone 4-104 and five were susceptible to virus transmission by the clone 4-104.

DISCUSSION

The *Vat* gene has been mainly studied using the clone NM1 of *A. gossypii*. We observed that C9 clones also have reduced acceptance on plant with the allele of resistance at *Vat* locus. However, acceptance is reduced less for C9 than for NM1. Moreover, the *Vat* gene might not reduce the biotic potential of C9 (Thomas et al. 2008). QTL with minor effects were detected in the recombinant line populations derived from ‘Védrantais’ x PI 161375. Those QTL had additive and epistatic effect and half of them clearly have a specific effect on the acceptance by NM1 or C9 genotypes of *A. gossypii*. We detected QTL that reduced the biotic potential of NM1 but we did not look for QTL that reduced C9 biotic potential. Nevertheless QTL affecting biotic potential of other clones that NM1 might exist as Kishaba et al. (1976) suggested that minor genetic factors affected biotic potential of *A. gossypii* (biotype D collected in California) in PI 371795 (the parent of PI 414723). Because we detected some QTL having their allele of resistance in the susceptible parent (‘Védrantais’), or QTL with trans epistatic effect, lines with higher resistance than PI 161375 might be obtained. Those lines, more resistant to NM1 or C9, existed in our RIL populations and one line was more resistant to NM1 and C9 than PI 161375 for acceptance.

All the accessions resistant to virus transmission by the 4-104 clone (C9 genotype) were resistant to virus transmission by the NM1-lab clone (NM1 genotype). These accessions were also resistant to acceptance and colonization by the NM1-lab clone except PI 164323. PI 161375, in which the *Vat* gene was isolated, belongs to this group. According to allelism tests conducted by Pitrat et al. (1988), Klingler et al. (2001) and Soria et al. (2003), resistance of three genotypes having those phenotypes (PI 161375, PI 417723, PI 482480=TGR1551) is conferred by the same locus *Vat*. Some accessions were resistant to NM1 *A. gossypii* (aphids and virus transmission) and susceptible to C9 genotype (aphids and virus transmission). For ‘Anso 77’, Pitrat et al. (1988) showed that aphid resistance to NM1 was conferred by the locus *Vat*. Therefore we hypothesized existence of different alleles at the *Vat* locus that conferred resistance to NM1 clones and susceptibility to C9 clones.

Four accessions presented a complete independency between aphid resistance (acceptance and colonization) and resistance to virus transmission when using the same clone of *A. gossypii*. ‘Escrito 8429’ and PI 282448 were resistant to the NM1-lab clone and susceptible to CMV after inoculation by this clone. In contrast, PI 164323 was susceptible to the NM1-lab clone, resistant to CMV after inoculation by this clone. PI 282448 was resistant to the 4-104 clone (C9 genotype) and susceptible to CMV after inoculation by this clone. ‘Miel blanc’ was susceptible to the C9 clones, and resistant to CMV after inoculation by this clone. Those phenotypes may be conferred by the locus *Vat* (different alleles) or other locus for aphid resistance.

Some accessions exhibited new phenotypes of resistance to *A. gossypii*; they are under study to identify new alleles/genes of resistance (Dogimont et al. 2008).

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