

A mutation in the *Rme1* tomato locus reduces *Mi-1.2*-mediated resistance to whitefly *Bemisia tabaci*

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Abstract: The tomato gene *Mi-1.2* is responsible for resistance to three species of root-knot nematodes (*Meloidogyne* spp.), to the potato aphid (*Macrosiphum euphorbiae*) and to the sweetpotato whitefly (*Bemisia tabaci*). Working with a fast-neutron irradiated population of tomato cv. Motelle (*Mi-1/Mi-1*), we identified a mutant (*rme1*) that was defective in a locus other than *Mi-1*. We did this on account of its susceptibility to *M. javanica* and to potato aphid, which indicated that *Rme1* is required for *Mi-1*-mediated resistance. Two experimental assays were carried out in order to evaluate the influence of the *Rme1* locus on *Mi-1*-mediated resistance to the B-biotype of *B. tabaci*. In a greenhouse free-choice assay, 10 two-month-old *rme1* mutant plants, and the same number of Motelle and Moneymaker (near isogenic susceptible control: *mi-1/mi-1*) plants, were randomised in a complete block design and whitefly adults were freely released in the greenhouse. Five days later, the number of whitefly adults on each plant was counted. This count was then repeated on alternate days over a 15-day period. The mean number of adult whiteflies per plant was significantly greater on *rme1* mutants than on Motelle plants and slightly lower, but not significantly different, from on Moneymaker. For the no-choice assay, 11 eight-week-old plants of each genotype were kept in a growing chamber. Five adult female whiteflies were placed in a plastic clip-cage attached to the under surface of one leaf (one cage per plant). After 6 days, the average number of eggs observed on the *rme1* mutant plants was almost identical to that on Moneymaker and significantly greater than that observed on Motelle. These results suggest that the *Rme1* locus is also required for *Mi-1*-mediated resistance to the *B. tabaci* B-biotype.

Key words: *Bemisia tabaci*, whiteflies, *Rme1*, resistance, *Mi-1.2* gene, mutant plants, tomato.

Introduction

The tomato (*Lycopersicon esculentum* Mill.) gene *Mi-1* confers resistance to the three most common species of root knot nematodes: *Meloidogyne arenaria* Neal, *M. incognita* (Kofoid & White) and *M. javanica* (Treub). After cloning, *Mi-1* was found to confer resistance to two additional organisms: potato aphid, *Macrosiphum euphorbiae* (Thomas) (Rossi et al., 1998) and whitefly, *Bemisia tabaci* (Gennadius) (Nombela et al., 2000 and 2003).

There is evidence that *Mi-1*-mediated resistance to root-knot nematodes, potato aphids and whiteflies is rather specific, but it is not clear how *Mi-1* mediates this resistance. Furthermore, it is not clear whether the defense responses mediated by *Mi-1* against the three organisms are identical.

Using a genetic screen to identify suppressors of *Mi-1*, we identified a mutant, *rme1* (resistance to *Meloidogyne*), that is compromised in resistance to *M. javanica* and to potato aphids (Martinez de Ilarduya et al., 2001). Here we present data to suggest that *rme1* mutant plants are also compromised in resistance to the sweet potato whitefly *B. tabaci*.

Material and methods

Plants and insects

We worked with the near isogenic tomato (*L. esculentum*) pair Motelle (*Mi-1/Mi-1*) and Moneymaker (*mi-1/mi-1*) and also used a mutant in the background of the wild-type parent Motelle, *rme1*. Tomato seedlings were transplanted into one-litre pots with perlite. Eight week-old tomato plants were used in the experiments

The experiment was conducted with B-biotype *B. tabaci* adults that had been reared on tomato cv. Marmande for more than 30 generations.

Free-choice assay

Ten plants from each genotype were randomised in a complete block design in an insect-free greenhouse with average temperatures of 23°C (day) and 18°C (night) and a relative humidity of 46% to 69%. Plants were infested by releasing mature adult whiteflies in the centre of the greenhouse. After five days, the number of adult whiteflies was counted *in situ* on all leaves. Similar counts were repeated every other day over a period of 15 days.

No choice assay

Eleven plants from each genotype were kept in a growth chamber at 25°C, with a photoperiod of 16-h-light and 8-h-dark and a relative humidity of 68% to 75%. Five adult female whiteflies were placed in a plastic clip-cage, attached to a single leaflet, in such a way that they had access to the abaxial surface of the leaf (Nombela et al., 2001). One plant was used per cage. At the end of the experiment, the number of eggs laid on each plant was recorded.

Data were $\log_{10}(x+1)$ transformed and compared using a one-way ANOVA and Tukey HSD test (Statsoft, 1994).

Results and discussion

Both free choice and no choice experiments indicated that resistance to the *B. tabaci* B-biotype was completely compromised in the *rme1* mutant. In the free choice assay, the daily infestation rates of *B. tabaci* B-biotype on the *rme1* mutant plants were intermediate between those on Moneymaker and Motelle (Table 1). However, the mean values of the number of adults per plant per day on the *rme1* mutant were similar to those on Moneymaker and significantly ($P<0.05$) greater than on Motelle (Table 1).

Table 1. Average daily number of adults of the *B. tabaci* B-biotype observed per plant during the free-choice experiment, and number of eggs per plant laid by 5 females during 6 days under no-choice conditions*.

	Adults per plant (free-choice assay)	Eggs per plant (no-choice assay)
Rme1 mutant	3.74 a	68.82 a
Motelle	2.25 b	36.82 b
Moneymaker	4.72 a	68.18 a

* Means followed by different letters in a column differ significantly ($P<0.05$) by Tukey's HSD.

In the no choice experiment, the average number of eggs observed on the *rmel* mutant plants six days after infestation was similar to that for Moneymaker and significantly ($P < 0.05$) greater than that observed on Motelle (Table 1).

We previously reported that *rmel* was compromised in its resistance to *M. javanica* and to potato aphid (Martinez de Ilarduya et al., 2001). Comparing the present results with those for responses to other tomato pests and diseases may help us to determine whether *Rmel* is required for *Mi-1*-mediated resistance and for characterising such resistance.

References

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