Chapter 14

Host Plant Resistance for the Management of *Bemisia tabaci*: A Multi-crop Survey with Emphasis on Tomato

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Introduction

Control measures commonly used against insect pests in horticultural crops rely mainly on the use of pesticides, but these products are often toxic to the environment and to non-target species. Moreover, control of insects is difficult because many are polyphagous and develop insecticide resistance quickly. Also, the use of these compounds favors the development of resistant populations, rendering their application counter-productive in the long term. Consequently, there is a general opinion that the best way to solve the pest problem is by Integrated Pest Management (IPM), based on the rational and coordinated application of appropriate selective, economical and environmentally friendly techniques. As an example, prevention and management of virus-transmitting insects (specifically whiteflies) on horticultural crops was declared officially in 2004 as a public benefit issue in Spain. In this context, the use of more adequate control measures in place of conventional chemical control should be recommended when feasible for virus vector control.

Host plant resistance is one of the main basic components of IPM, and the utilization of resistant plants has long been considered as one of the most effective components of insect control (Russell 1978). As a consequence, an increase in research aiming to favor the use of varieties resistant to pest organisms has been observed during recent years in many countries. This has produced an important advance in the available knowledge of the defensive plant response against attack by certain organisms. The phenomenon of plant resistance can be described as the relative reduction of pest population size compared with the standard varieties due to genetic characteristics of the host plant (Ponti et al. 1990). The “gene-to-gene” hypothesis (Flor 1971) was for many years the main concept supporting the whole theory of plant-pathogen interactions. However, this concept is being reformulated in the context of recent advances in understanding the molecular structure of plant resistance genes. Earlier data suggested that resistance usually results from

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a biochemical pathway wherein pathogen specificity is controlled only by a few components (Beynon 1997). More recent investigations reveal that plant resistance has evolved by an integration of biochemical processes in a general network of responses induced by parasite attack.

The resistance process can be summarized as follows: When the parasite contacts the plant, plant cells recognize a parasite-produced compound, generating an initial plant signal. This signal is transduced intracellularly, producing substances that also cause synthesis of compounds either directly involved in defence or in transducing the initial signal. Finally, these transducers translocate to the action site. This process is very complex and involves radical changes in cell metabolism and gene expression, either by gene activation or silencing. As a consequence, these changes lead finally to the appearance of different defence mechanisms which can be classified in three main categories: (a) reinforcement of physical barriers by an increase of cell wall consistency and a decrease of cell wall porosity, (b) alteration of phytohormone balance, and (c) increase in the production of toxic substances. The sequence of all these processes involved in plant defense and resistance has been widely reviewed, especially in angiosperms (Dixon et al. 1994; Hutcheson 1998).

Plant resistance can be classified in two categories: natural (also referred to as innate or congenital) and induced (also referred to as acquired). Regarding natural resistance, there are plant species as well as multiple germplasm within a species which possess a high level of resistance that has been selected through the evolutionary process. In many cases this resistance applies also to other physical or chemical conditions that are adverse to the plants. However, induced resistance is acquired in plants after being attacked in such a way that in many instances if a plant survives an attack, it defends itself much better from a later attack from the same organism or even other organisms. Inducible responses are thought to be a means of avoiding costs associated with constitutively expressed resistance mechanism (Stout et al. 2002).

Innate Resistance in Tomato: The *MI-1* Gene

Tomato, *Solanum lycopersicum* L. (Solanaceae), is widely distributed, being one of the economically most important crops in the Mediterranean area and elsewhere. One of the limiting factors affecting the success of this crop is the existence of whiteflies. Among them, the whitefly *Bemisia tabaci* (Gennadius) is one of the most significant. Damage caused by this insect to commercial tomato may be directly through phloem feeding, or indirectly by the transmission of plant viruses such as *Tomato Yellow Leaf Curl Virus* (TYLCV) (Mehta et al. 1994; Moriones and Navas-Castillo 2000).

Resistance to whiteflies and other insects found on the wild species of tomato *Solanum pennellii* (Corr.) D'Arcy were mostly attributed for the past to the presence of sugar esters in the glandular exudate of type IV trichomes (Gentile et al. 1968; Juvik et al. 1994; Heinz and Zalom 1995). It was therefore concluded that selection for sugar ester accumulation should be an efficient technique in selecting

for general insect resistance in *S. pennellii* (Goffreda et al. 1990) and other tomato plants (Kisha 1981; Berlinger 1986). Choice and non-choice assays revealed a correlation between resistance to *B. tabaci* (reported as *B. argentifolii*) and type IV trichome densities of six wild accessions of *Solanum habrochaites* (reported as *L. hirsutum*); the accessions included LA1777 (Snyder et al. 1998). More recently, multigenic resistance to *B. tabaci* in LA1777 was further demonstrated to be correlated with the presence of type IV trichomes (Momotaz et al. 2005). Similarly, leaf-trichome densities and presence of acyl sugars in the exudate of glandular trichomes as well as type of trichomes were reported to be important factors affecting whitefly-tomato relationships (Williams et al. 1980; Kisha 1981; Berlinger 1986; Kishaba et al. 1992; Simmons 1994; Freitas et al. 2002; Baldin et al. 2005; Srinivasan and Uthamasamy 2005; Simmons and Gurr 2005; Sanchez-Pena et al. 2006). However, the actual effect of these, and other factors, on tomato resistance has been broadly questioned (Goffreda and Mutschler 1989; Steffens and Walters 1991; Liedl et al. 1995; Toscano et al. 2002a, b; Muigai et al. 2003). Field resistance to *B. tabaci* was reported in different lines of the wild species *L. peruvianum* (L.) Mill (Hassan et al. 1982; Channarayappa et al. 1992), a close relative of commercial tomato, *S. lycopersicum*. This resistance was shown to be due to factors other than the presence of trichomes and their exudates, because Va and Vb non-glandular trichomes were predominant and VIc glandular trichomes were absent (Channarayappa et al. 1992). These authors also reported that commercial cultivars of tomato mostly exhibit non-glandular type III and V trichomes and four-lobed glandular VIa trichomes which are not considered to be important in whitefly control mechanisms (Ponti et al. 1990). So, differential host response to insects in these commercial cultivars might be due to alternative mechanisms of resistance regulated by genes that were previously unknown.

In a study carried out in our laboratory (Nombela et al. 2000), two commercial cultivars of tomato, 'Alta' and 'Peto 95', the accession LA716 of *S. pennellii* and lines 94GH-006 and 94GH-033 (backcrosses between 'Peto 95' and LA716), with different leaf acyl sugar contents were screened for resistance to the Spanish B-biotype of *B. tabaci* in greenhouse and field no-choice experiments. There was no oviposition on LA716 (with the highest acyl sugar content) while the greatest fecundity and fertility values were observed on the cultivar Alta (no acyl sugar content). However, no clear relationship was found between the low acyl sugar content in the other tomato cultivars tested and whitefly reproduction. Resistance to *B. tabaci* appears to be independent of acyl sugar content below a threshold level of $37.8 \mu\text{g cm}^{-2}$ leaf (Fig. 14.1). Moreover, feeding similarities between piercing-sucking insects, such as aphids and whiteflies, and root-knot nematodes (Byrne and Bellows 1991; Walker and Perring 1994; Kaloshian et al. 1995) suggested that a specific gene-for-gene defence response (Flor 1955) could be highly effective in regulating the prolonged interaction of the stylets of these insects and the plant cell contents (Fernandes 1990).

There are many examples of single resistance genes conferring gene-for-gene resistance to piercing-sucking insects and nematodes. The *Mi-1* gene, present in many varieties of cultivated tomato (*S. lycopersicum*) and introduced into this plant

Fig. 14.1 Mean daily egg, pupa and adult production per female and adult emergence of *Bemisia tabaci* on tomato, represented as bars scaled on the Y1 axis, in greenhouse (black) and field (white) no-choice experiments. Error bars indicate the SEM.

Different letters on bars from the same experiment indicate that corresponding means differ significantly ($p < 0.05$) by Fisher's Protected LSD. Black points connected by dashed lines in the first graph represent the Total Acyl sugar Content (TAC = glucose + sucrose) of each cultivar, backcrossing line or wild species accession, scaled on the Y2 axis. A = Alta, P = Peto 95, 06 = 94GH-006, 33 = 94GH-033, L = LA716

from its wild relative, *S. peruvianum* (Smith 1944), confers resistance characterized by a hypersensitive reaction to three species of root-knot nematodes *Meloidogyne* spp. (Roberts and Thomason 1986). It was since demonstrated that *Mi-1* in tomato also regulates resistance to insects such as the potato aphid *Macrosiphum euphorbiae* (Thomas) (Kaloshian et al. 1995; Rossi et al. 1998).

In a greenhouse choice assay (Nombela et al. 2000), *B. tabaci* exhibited reduced host preference and reproduction on three commercial tomato cultivars with the *Mi-1* gene ('Motelle', 'VFN8' and 'Ronita') compared with three *Mi-1*-negative

cultivars ('Moneymaker', 'Río Fuego' and 'Roma'). When data of *Mi-1*-positive tomato plants were pooled, the mean values for daily infestation and pupal production of *B. tabaci* were significantly lower than those of *Mi-1*-negative plants (Table 14.1).

Similarly, three tomato varieties ('Motelle', 'Ronita', and 'VFN8') bearing the *Mi-1* gene, and three varieties not bearing this gene ('Moneymaker', 'Roma', and 'Río Fuego'), were compared by choice and no-choice assays for host preference using the Q-biotype of *B. tabaci* (Nombela et al. 2001). The most preferred hosts, determined by infestation levels and numbers of feeding adults were 'Moneymaker', 'Río Fuego' and 'Roma', all of which were not carrying the *Mi-1* gene. 'Ronita' and 'Motelle', both of which bore the *Mi-1* gene, were the least preferred hosts. In a no-choice assay, *B. tabaci* females laid significantly fewer eggs on varieties that carried the *Mi-1* gene than on those lacking the gene. Detectable differences were more dramatic when plants carrying the *Mi-1* gene were pooled together and compared with pooled plants without this gene. Significantly greater values were obtained for the *Mi-1*-negative group for all parameters tested (Fig. 14.2). Comparing these results with those from our previous study on the B-biotype of *B. tabaci* (Nombela et al. 2000), Q-biotype was found to produce higher daily infestation rates on most of the tomato varieties. When results from plants carrying *Mi-1* were pooled, they showed lower infestation levels of Q-biotype than B-biotype. Q-biotype infested fewer *Mi-1* plants and more non-*Mi-1* plants than B-biotype. Q-biotype females produced significantly fewer pupae than B-biotype females on both groups of plants. These results suggested the existence of an antixenosis and antibiosis-based resistance to the Q-biotype of *B. tabaci* in *Mi*-bearing commercial tomato varieties, which is greater than that previously reported for the B-biotype. More importantly, these results indicated that *Mi-1*, or another closely linked gene, might be implicated in a partial resistance which was not associated with either the presence of glandular trichomes or their exudates. These findings supported the general hypothesis for the existence of similarities among the resistance mechanisms to whiteflies, aphids and nematodes in commercial tomato plants.

The *Mi-1* gene was cloned and this locus in chromosome six of tomato was found to contain two transcribed genes, *Mi-1.1* and *Mi-1.2*, with 91% homology (Milligan et al. 1998). *Mi-1.2*, but not *Mi-1.1*, is sufficient to confer resistance to root-knot nematodes (Milligan et al. 1998) and aphids (Rossi et al. 1998), and we refer to this gene as *Mi-1*. Cloning of *Mi-1* allowed us to finally demonstrate that this gene mediates resistance to both B- and Q-biotypes of *B. tabaci* in 2-month-old tomato plants (Nombela et al. 2003). The response of whiteflies to tomato plants carrying the cloned *Mi-1* was compared to that of the isogenic untransformed tomato line 'Moneymaker' in both free-choice and no-choice assays. The transgenic lines used in that work differed from susceptible 'Moneymaker' only in the presence of a 14.7 kb DNA insert containing *Mi-1.2*. The daily infestation rates of both B- and Q-biotypes during the free-choice assays were lower on the transgenic line 143-11-16-36 than on 'Moneymaker' at all time points examined. Mean values of the percentage of adults (Fig. 14.3A) as well as the final numbers of pupae per plant (Fig. 14.3B) or per leaf (Fig. 14.3C) were also significantly reduced on the

Table 14.1 Daily infestation rate and pupal production (Mean $\pm$ SEM) of *Bemisia tabaci* (B-biotype) on six cultivars of tomato of varying trichome density, with and without *Mi* gene, under greenhouse conditions

Cultivars	Daily infestation					Pupal production					
	n	Trichomes/cm ²	Plants infested by females (%)		Plants infested by adults (%)	Females on plants (%)	Adults on plants (%)	n	Pupae per plant	Pupae per leaflet	Leaflets infested by pupae (%)
<i>Separated</i>											
Moneymaker (- <i>Mi</i>)	20	68 ± 7.0 ab	25	74 ± 3.8 a	82 ± 3.5 a	24 ± 0.9 a	23 ± 0.8 a	10	372 ± 73.1 ac	3 ± 0.6 b	26 ± 2.6 ab
Río Fuego (- <i>Mi</i>)	20	56 ± 4.9 b	25	70 ± 3.5 a	82 ± 2.9 a	23 ± 1.7 a	25 ± 1.8 a	10	435 ± 68.4 a	5 ± 0.9 a	31 ± 3.1 a
Roma (- <i>Mi</i>)	20	75 ± 5.0 a	25	66 ± 3.9 ab	76 ± 3.4 ab	14 ± 0.6 b	15 ± 0.6 b	10	369 ± 69.6 ac	3 ± 0.6 ab	26 ± 2.7 ab
Motelle (+ <i>Mi</i>)	20	42 ± 4.8 c	25	60 ± 4.7 bc	68 ± 3.9 bc	14 ± 1.0 b	13 ± 0.7 bc	10	230 ± 62.5 b	2 ± 0.5 b	19 ± 2.9 b
VFN8 (+ <i>Mi</i>)	20	35 ± 3.9 c	25	56 ± 4.5 c	62 ± 4.4 c	14 ± 0.9 b	13 ± 0.8 bc	10	191 ± 45.3 bc	2 ± 0.3 b	21 ± 2.0 b
Ronita (+ <i>Mi</i>)	20	68 ± 4.6 ab	25	54 ± 4.3 c	70 ± 3.6 bc	11 ± 0.7 c	12 ± 0.6 c	10	283 ± 75.6 ab	3 ± 0.7 b	22 ± 2.4 b
<i>Pooled</i>											
Without <i>Mi</i>	25		25	70 ± 18.6 a	80 ± 16.4 a	61 ± 1.7 a	62 ± 1.4 a	30	392 ± 39.6 a	4 ± 0.4 a	28 ± 1.6 a
With <i>Mi</i>	25		25	57 ± 22.3 b	67 ± 19.9 b	39 ± 1.7 b	38 ± 1.4 b	30	235 ± 35.5 b	2 ± 0.3 b	20 ± 1.4 b

Means followed by the same letter in the same columns are not significantly different ($p < 0.05$) by Fisher's Protected LSD.

Fig. 14.2 Pupal production and fecundity of *B. tabaci* (Q-biotype) on pooled tomato plants with and without *Mi* gene. (A) number of pupae per plant. (B) number of pupae per leaflet. (C) percentage of leaflets infested by pupae. (D) daily number of eggs per female

transgenic line 143-11-16-36. At the end of the no-choice assays, the total number of insects from the B-biotype (Fig. 14.3D) and Q-biotype (Fig. 14.3E) were significantly lower on the transgenic line than on 'Moneymaker'. In summary, our results indicated that *Mi-1.2* is responsible for the resistance in tomato plants to both B- and Q-biotypes when tomato plants were 2-months old. We had previously observed that the development of *B. tabaci* on younger tomato plants (2–4 true leaf stage) did not differ in relation to the presence/absence of the *Mi* gene (Pascual et al. 2000), suggesting that the activity of this gene against *B. tabaci* could be developmentally regulated. Similarly, *Mi-1.2* plants were resistant to aphids only at 4–5 weeks of age (Kaloshian et al. 1995).

The *Mi-1* gene belongs to the NB-LRR class of R genes as it encodes a protein with a nucleotide binding site and leucine rich repeat (NB-LRR) motifs. Proteins of this motif structure make up the largest class of cloned plant resistance genes with specificity against diverse pathogens (including viruses, bacteria and fungi), nematodes and insects (Dangl and Jones 2001). Recent findings have shown that resistance genes of the NB-LRR type could confer resistance to insects. So far, *Mi-1* gene is the only cloned resistance gene that has been demonstrated to mediate resistance to insects (Kaloshian and Walling 2005).

Probing and feeding behaviour of *B. tabaci* B-biotype on the near-isogenic tomato lines 'Moneymaker' (without *Mi-1*) and 'Motelle' (with *Mi-1*) was studied using a DC Electrical Penetration Graph (EPG) to characterize *Mi-1*-mediated resistance (Jiang et al. 2001). Significant differences between tomato lines were found

Fig. 14.3 Reproductive activity of B- and Q-biotypes *B. tabaci* on tomato transgenic line 143-11-16-36 (Mi^+) in black and cv. Moneymaker (Mi^-) in white. Different letters for the same biotype indicate significant differences by ANOVA ($p < 0.05$). (A) percentages of adults present during the free-choice assays under greenhouse conditions. (B) average number of pupae per plant at the end of the greenhouse free-choice experiments. (C) average number of pupae per leaf at the end of the greenhouse free-choice experiments. (D) total number of B-biotype individuals (N3, pupae and new adults) at the end of the no-choice assay. (E) total number of Q-biotype individuals (N3, pupae and new adults) at the end of the no-choice assay ($p = 0.073$)

in EPG parameters related to epidermis and/or mesophyll tissues (Table 14.2). On 'Motelle', a lower percentage of whiteflies achieved phloem phase and more probes were made before first phloem phase was attained. Also, a higher ratio (number of probes before first phloem phase)/(total number of probes) was observed as were more non-probing events, and longer times to achieve first intracellular puncture and first phloem phase. In contrast, most of the parameters related to phloem phase were found not to differ significantly between these near-isogenic lines. The behavioural

Table 14.2 Electrical Penetration Graph (EPG) parameters, possible relations to resistance factors in specific plant tissue and comparison of EPG parameters of B-biotype of *B. tabaci* on two isogenic tomato lines during 6-h recordings

EPG parameters	Tissue(s)/factors involved	Moneymaker mean $\pm$ SE (n)	Motelle mean $\pm$ SE (n)
<i>Non-phloem parameters</i>			
Time to the first probe	Surface factors	39.09 $\pm$ 14.94 (25)	61.68 $\pm$ 25.76 (26) NS
Duration of 1st probe	Surface factors + epidermis/mesophyll	89.48 $\pm$ 24.96 (25)	99.78 $\pm$ 31.11 (26) NS
Total number of probes	Surface factors + all plant tissues	44.36 $\pm$ 5.05 (25)	57.23 $\pm$ 8.38 (26) NS
Number of probes made before attaining the first phloem phase (1st E(pd))	Epidermis, mesophyll and other parenchyma	27.08 $\pm$ 3.65 (25)	53.00 $\pm$ 8.87 (26)*
Ratio of probes before 1st E(pd)/total number of probes	Epidermis, mesophyll and other parenchyma	0.65 $\pm$ 0.06 (25)	0.86 $\pm$ 0.05 (26)**
Total duration of non-probing time	All plant tissues except phloem	65.81 $\pm$ 7.95 (25)	100.39 $\pm$ 11.50 (26)*
Total duration of F	All plant tissues except phloem	21.12 $\pm$ 4.00 (12)	32.54 $\pm$ 6.65 (19) NS
Total duration of G	Xylem factors	20.31 $\pm$ 3.25 (9)	42.88 $\pm$ 15.62 (14) NS
Time whitefly took to make its first intracellular puncture (1st pd) from the start of the 6 h exper. (min)	All plant tissues except phloem	94.31 $\pm$ 12.44 (24)	154.38 $\pm$ 20.32 (25)*
Time whitefly took to the 1st E(pd) from the start	All plants tissues except phloem	163.25 $\pm$ 20.01(25)	272.16 $\pm$ 22.82 (26)**
Time whitefly took to 1st E(pd) within probe	All plants tissues except phloem	21.19 $\pm$ 2.28 (22)	38.97 $\pm$ 7.15 (13) NS
Percentage of whitefly reaching the phloem phase	Epidermis, mesophyll and other parenchyma	21.19 $\pm$ 2.28 (22)	38.97 $\pm$ 7.15 (13) NS
		88.00	50.00**
<i>Phloem parameters</i>			
Duration between time to the 1st E(pd) and time to the first sustained ingestion (1st SI) (E(pd)2>15 min)	Vascular tissue and sieve elements (initial factors)	35.99 $\pm$ 17.79 (19)	11.34 $\pm$ 9.72 (11) NS
Time whitefly took to 1st SI from start	All plant tissues	188.62 $\pm$ 21.12 (25)	277.79 $\pm$ 21.45 (26)**
Percentage of time whitefly spent in E(pd)2 after the 1st phloem phase	Phloem factors	51.96 $\pm$ 28.31 (21)	62.4 $\pm$ 35.35(13) NS

Table 14.2 (continued)

EPG parameters	Tissue(s)/factors involved	Moneymaker mean $\pm$ SE (n)	Motelle mean $\pm$ SE (n)
Duration of E(pd)1 preceding the first E(pd)2 >15 min	Phloem factors	3.17 $\pm$ 0.72 (22)	1.63 $\pm$ 0.43 (13) NS
Duration of E(pd)2 after the 1st SI	Phloem factors	29.35 $\pm$ 9.18 (22)	13.39 $\pm$ 7.73 (13) NS
Total duration of E(pd)1	Phloem factors	5.85 $\pm$ 1.09 (22)	3.05 $\pm$ 0.87 (13) NS
Mean duration of E(pd)1	Phloem factors	2.73 $\pm$ 0.48 (22)	1.67 $\pm$ 0.44 (13) NS
No. of E(pd)1	Phloem factors	2.09 $\pm$ 0.24 (22)	2.08 $\pm$ 0.50 (13) NS
Total duration of E(pd)2	Phloem factors	106.44 $\pm$ 17.55 (21)	105.10 $\pm$ 24.00 (13) NS
Mean duration of E(pd)2	Phloem factors	64.88 $\pm$ 14.63 (21)	84.41 $\pm$ 25.14 (13) NS
No. of E(pd)2	Phloem factors	1.90 $\pm$ 0.25 (21)	1.69 $\pm$ 0.33 (13) NS
Percentage of whiteflies showing sustained ingestion (E(pd)>15 min	All plant tissues	76.00 (19/25)	42.30 (11/26)*

* $p < 0.05$
** $p < 0.01$.
Significant differences were compared at the 0.05 and 0.01 level according to the Mann-Whitney U test (Statview, Abacus Concepts, 1987). In the case of percentage or ratio, a chi-square test was used at 0.05 and 0.01 levels. NS, no significant differences ($p>0.05$).

data strongly suggest that tomato resistance mediated by *Mi-1* is due to factors in the epidermis and/or mesophyll that inhibit whiteflies from reaching phloem sieve elements. However, once the stylets reach a sieve element, whitefly behaviour did not differ between the two varieties. Thus, phloem sap of the two varieties appears to be equally acceptable to the whiteflies.

Avirulence effectors that interact with *Mi-1* protein have not yet been identified in any root-knot nematodes, aphids or whiteflies. Apparently, *Mi-1* either recognizes more than one distinct avirulence product or recognizes perturbations in a host protein conveyed by these three organisms (Dangl and Jones 2001; Mackey et al. 2002). Using a genetic screen to identify suppressors of *Mi-1*, a tomato mutant, *rme1* (resistant to *Meloidogyne*), compromised in resistance to the root-knot nematode *M. javanica*, and to the potato aphid *M. euphorbiae*, was identified (Martínez de Ilarduya et al. 2001). Subsequently, our group demonstrated that *rme1* mutant plants were also compromised in resistance to *B. tabaci* and to two other 2 species of root-knot nematode (*M. incognita* and *M. arenaria*), indicating that the *Rme1* gene is required for *Mi-1* resistance (Martínez de Ilarduya et al. 2004). In a free-choice assay, the mean values of the number of adults per plant per day on the *rme1* mutant were similar to those on susceptible 'Moneymaker' and significantly greater than on resistant 'Motelle' (Fig. 14.4A). Moreover, the average number of eggs

Fig. 14.4 Infestation of *B. tabaci* B-biotype on wild-type 'Motelle' (*Mi-1/Mi-1*), *rme1* mutant (*Mi-1/Mi-1*), and 'Moneymaker' (*mi-1/mi-1*) tomato plants. (A) Average number of adult whiteflies per sampling time on each plant genotype in the free-choice assay. Sampling was done every other day. Values represent the mean and standard error of 10 plant replicates. (B) Average number of eggs produced by *B. tabaci* B-biotype on each plant genotype during the no choice assay. Five adult female whiteflies were confined to a single leaflet per plant for 6 days. Each bar represents the mean and standard error of 11 replicates. Significantly different means are indicated by different letters ($p < 0.05$)

observed on the *rme1* mutant plants 6 days after infestation (no-choice experiment) was similar to that on 'MoneyMaker' and significantly greater than that observed on 'Motelle' (Fig. 14.4B). This work also provided evidence that *Rme1* acts early in the *Mi-1*-pathway, either at the same step as the *Mi-1* product or earlier in the response cascade.

In addition to resistance mediated by *Mi-1* gene, other studies are in progress to identify new sources of tomato resistance to *B. tabaci*, with interesting results during the last few years. From a screening of 25 wild and cultivated tomato genotypes, *S. habrochaites* LA1777 was determined to be resistant to both *B. tabaci* and the transmitted virus ToLCBV-[Ban4] (Maruthi et al. 2003). Ninety-four recombinant inbred lines were developed from wild accession LA1777 of *S. habrochaites*, and were tested for whitefly resistance together with the resistant parent (LA1777), susceptible parent (E6203), and three interspecific F₁ hybrids (Momotaz et al. 2005). No resistance was detected in any recombinant inbred lines. From an interspecific F₂ population, 11 resistant and 10 susceptible plants were selected to locate resistance genes by testing them with 400 molecular markers. So far, markers in five regions on four different chromosomes appear to be associated with resistance and subsequent research is in progress to identify the resistant loci (Momotaz et al. 2006). Other authors investigated eight wild populations of *S. lycopersicum* var. *cerasiforme* and one population of *S. habrochaites* (C-360), and found lower numbers of *B. tabaci* than on the cultivated variety 'Rio Grande'; the lowest whitefly incidence was on *S. habrochaites* (Sanchez-Pena et al. 2006).

Induced Resistance in Tomato

In addition to innate resistance, plants can activate protective mechanisms upon contact by invaders; this is termed induced or acquired resistance (Sticher et al. 1997). Induced resistance is a long known phenomenon. However, only a few laboratories around the world had studied it until the last decade. Since then, induced resistance has received growing attention as a new and environmentally friendly control method, as well as a model to study genes involved in defence and control signaling. It was initially observed that plants inoculated with attenuated micro-organisms were protected against later infections by the same or another pathogen, but no relationship was established with the activation of host defence mechanisms (reviewed in Hammerschmidt and Kuc 1995). The activation of true resistance mechanisms was demonstrated when it was observed that a localized infection could lead to resistance against later infections by very different pathogens (Ross 1966). Induced resistance in plants can be compared as a whole with immunization in humans and other animals, although underlying resistant mechanisms are different in animals. Acquired or induced resistance can be expressed locally, at the site of primary inoculation (localized acquired resistance or LAR). It may also be expressed systemically in tissues far away from the initial inoculation site, and this is termed systemic acquired resistance or SAR (Agrawal et al. 1999).

Some biological agents such as certain bacteria, fungi or viruses can induce plant resistance to other pathogens (Agrawal et al. 1999; Hammerschmidt et al. 2001; Siddiqui and Shaukat 2004). Similarly, positive and negative associations as a result of cross-talk between insect- and pathogen-induced defence pathways have been widely reported (reviewed by Hunter 2000), including the induction of plant resistance to insects due to a previous attack by the same or another organism. Available information on induced resistance to arthropods mostly refers to chewing herbivores, which usually cause extensive leaf damage to infested plants. A less studied phenomenon is the induction of plant resistance to or by phloem-feeding insects, such as whiteflies, which maintain a longer interaction with their host plant but causing only limited direct damage to the plant tissues with their stylets (Walling 2000). Little is known to date about plant responses to whiteflies induced after previous attacks by arthropods or by other inducer organisms (Agrawal et al. 2000; Inbar et al. 1999; Mayer et al. 2002; Murugan and Dhandapani 2007).

Several different bioassays have been carried out recently under controlled conditions in our laboratory to determine if resistance against *B. tabaci* could be induced in susceptible tomato plants (lacking the *Mi-1* gene) after a previous infestation by the same or another organism. To date, we found that 3 days of contact with 20 apterous adults of the potato aphid *M. euphorbiae* were enough for the plants to acquire resistance to *B. tabaci* (Nombela et al. 2004). This resistance was transient because whitefly numbers were significantly reduced when aphid attack occurred between 1 and 18 h prior to *B. tabaci* infestation, whereas the reduction was less detectable when 4 days passed between aphid and whitefly infestations. The resistance observed when *B. tabaci* infested the plants 18 h after aphid contact on a single leaflet was both locally (LAR) and systemically (SAR) induced (Nombela et al. 2009). However, our results indicate that the tomato responses induced by whitefly feeding depended on the aphid clone.

In addition to biological inducers, various chemicals have been discovered that seem to mimic all or part of the biological activation of resistance, but only a few have reached commercialization (Oostendorp et al. 2001). Benzo[1,2,3]thiadiazole-7-carbothioic acid-*S*-methyl ester (BTH) is the active ingredient of the Syngenta plant activator Bion[®] or Actigard[®]. Benzo[1,2,3]thiadiazole derivatives have been shown to mimic the biological activation of systemic acquired resistance by necrogenic pathogens (Kunz et al. 1997). A number of reports exist on the efficacy of Bion[®] as an inducer of resistance in different cultivated plants against a broad spectrum of fungal, bacterial, and viral diseases (Oostendorp et al. 2001; Smith-Becker et al. 2003). Treatment with this product in combination with other crop management strategies has been tested for protection against TYLCV and its vector *B. tabaci*, resulting in increased fruit production (Monci et al. 2003). However, little is known about the actual effect of BTH on insect pest populations and the available information on the effect of BTH treatment on whitefly densities is even more limited.

Recently, a reduction of whitefly densities was reported on cotton due to BTH treatment, although the authors considered the effect negligible (Inbar et al. 2001). In tomato, a trend toward reduced densities of whiteflies, although not significant,

Fig. 14.5 Mean ($\pm$ SE) numbers of total pupae and pupae per tomato leaflet of *B. tabaci* (B-biotype) observed on BTH and control plants at 29 days after treatment in the greenhouse free-choice assay. Different letters indicate significant differences between treatments: $p = 0.005$ for total pupae (capital letters) and $p = 0.006$ for pupae per leaflet (lower case letters)

was observed on BTH-treated plants (Inbar et al. 1998). More recently, we carried out a study to test the possible induction of resistance to the B- and Q-biotypes of *B. tabaci* by BTH in susceptible tomato plants of cv. Marmande (Nombela et al. 2005). The BTH treatment affected host preference of adults from both biotypes on plants sprayed with Bion[®] at 0.2 and 0.4 g/L during early free-choice assays. Consequently, a decrease in the total number of eggs the final number of pupae and empty pupal cases was observed (Fig. 14.5). The test for an effect produced by BTH applied at 0.1 g/L Bion[®] was not significant. In no-choice assays, a reduction in number of first-stage nymphs and total individuals, and a delay in insect development were observed when the local treatment was restricted to one leaflet per plant 5 days before *B. tabaci* (biotype B) infestation (Table 14.3). This acquired resistance induced by BTH was locally expressed because of the differences between treated and untreated leaflets on the same plants (Table 14.4), whereas no differences in untreated leaflets were observed between BTH-treated and control plants (Nombela et al. 2005).

Plant Resistance to *B. tabaci* in Other Crops

Although other genes for resistance against *B. tabaci* have not been cloned to date, a number of studies on the resistance to this pest in plants other than tomato have been carried out during the last decade. Diverse research has been carried out to compare the biotic potential of whiteflies in different cultivated plants such as sesame, beans, cucumber, cantaloupe, zucchini, cassava, corn, poinsettia, cabbage and tomato (Morales and Cermeli 2001; Villas-Bôas et al. 2002). However, most studies focused on testing different cultivars or accessions as potential sources of resistance to *B. tabaci* on a single crop.

There have been many studies searching for host plant resistance on cotton because of the severity of whitefly problems in this crop, mostly investigating the relative resistance of different cotton genotypes (Meagher et al. 1997; Wahla et al.

Table 14.3 Mean ($\pm$ SE) numbers of *B. tabaci* (B-biotype) eggs, N1, N2 and N3 observed on treated and untreated contiguous tomato leaflets, comparing treatments with BTH and water (control) at the end of the first (RT, n=10) and the second (CC, n=12) no-choice assays

Treated leaflets				No-treated (contiguous) leaflets				
Life stage	BTH plants	Control plants	<i>t</i>	<i>P</i>	BTH plants	Control plants	<i>t</i>	<i>P</i>
<i>RT assay</i>								
Eggs	0.4 ± 0.3 a	0.8 ± 0.3 a	0.92	0.37	0.2 ± 0.1 a	2.3 ± 1.8 a	1.21	0.24
N1	3.1 ± 1.3 b	5.3 ± 1.0 a	2.07	0.05	6.9 ± 1.4 a	7.8 ± 2.5 a	0.04	0.97
N2	0.0 ± 0.0 a	0.8 ± 0.5 a	1.61	0.13	0.1 ± 0.1 a	1.1 ± 0.7 a	1.27	0.22
Total	3.5 ± 1.3 b	6.9 ± 0.7 a	3.17	0.01	7.2 ± 1.4 a	11.1 ± 4.1 a	0.77	0.45
<i>CC assay</i>								
Eggs	3.0 ± 1.0 a	3.6 ± 0.7 a	0.95	0.35	3.8 ± 0.8 a	3.7 ± 1.0 a	0.03	0.98
N1	4.3 ± 1.4 b	11.7 ± 2.8 a	3.04	0.01	11.7 ± 3.5 a	14.3 ± 3.1 a	1.13	0.27
N2	0.0 ± 0.0 b	5.8 ± 1.0 a	14.97	0.00	3.0 ± 1.1 a	4.2 ± 0.8 a	1.66	0.11
N3	0.0 ± 0.0 a	1.0 ± 0.7 a	1.66	0.11	0.3 ± 0.3 a	0.0 ± 0.0 a	0.96	0.35
Total	7.3 ± 1.6 b	22.0 ± 3.5 a	4.09	0.00	18.8 ± 4.0 a	22.2 ± 2.6 a	1.30	0.21

RT: room temperature; CC: controlled conditions = 24°C, 16:8(L:D) h. Means in within a column followed by the same letter do not differ significantly ($P \leq 0.05$) by the Student's *t* test. N1, N2 and N3 are 1st, 2nd and 3rd instar, respectively.

Table 14.4 Mean ($\pm$ SE) numbers per leaflet of *Bemisia tabaci* (B-biotype) eggs, N1, N2 and N3 observed on tomato plants treated with BTH or water (control), comparing treated and untreated leaflets from the same plants at the end of the first (RT, n=10) and the second (CC, n=12) no-choice assays

BTH plants		Control plants			
		RT assay		CC assay	
Life stage		Treated leaflet	No-treated leaflet	<i>t</i>	<i>P</i>
<i>RT assay</i>					
Eggs		0.4 $\pm$ 0.3 a	0.2 $\pm$ 0.1 a	0.39	0.70
N1		3.1 $\pm$ 1.3 b	6.9 $\pm$ 1.4 a	2.71	0.02
N2		0.0 $\pm$ 0.0 a	0.1 $\pm$ 0.1 a	0.94	0.36
Total		3.5 $\pm$ 1.3 b	7.2 $\pm$ 1.4 a	2.52	0.02
<i>CC assay</i>					
Eggs		3.0 $\pm$ 1.0 a	3.8 $\pm$ 0.8 a	0.66	0.52
N1		4.3 $\pm$ 1.4 b	11.7 $\pm$ 3.5 a	2.07	0.05
N2		0.0 $\pm$ 0.0 b	3.0 $\pm$ 1.1 a	3.15	0.00
N3		0.0 $\pm$ 0.0 a	0.3 $\pm$ 0.3 a	1.00	0.33
Total		7.3 $\pm$ 1.6 b	18.8 $\pm$ 4.0 a	3.02	0.01
		Treated leaflet	No-treated leaflet	<i>t</i>	<i>P</i>
		0.8 $\pm$ 0.3 a	2.3 $\pm$ 1.8 a	0.34	0.74
		5.3 $\pm$ 1.0 a	7.8 $\pm$ 2.5 a	0.75	0.46
		0.8 $\pm$ 0.5 a	1.1 $\pm$ 0.7 a	0.21	0.84
		6.9 $\pm$ 0.7 a	11.1 $\pm$ 4.1 a	0.75	0.47
		3.6 $\pm$ 0.7 a	3.7 $\pm$ 1.0 a	0.23	0.82
		11.7 $\pm$ 2.8 a	14.3 $\pm$ 3.1 a	0.71	0.48
		5.8 $\pm$ 1.0 a	4.2 $\pm$ 0.8 a	1.44	0.17
		1.0 $\pm$ 0.7 a	0.0 $\pm$ 0.0 a	1.58	0.13
		22.0 $\pm$ 3.5 a	22.2 $\pm$ 2.6 a	0.27	0.79

RT: room temperature; CC: controlled conditions = 24°C, 16:8(L:D) h. Means in a column followed by the same letter do not differ significantly ($P \leq 0.05$) by the Student's *t* test. N1, N2 and N3 are 1st, 2nd and 3rd instar, respectively.

1998; Chu et al. 2002; Syed et al. 2003; Ripple 2004). The structure of cotton leaves was suggested to have potential for breeding whitefly-resistant upland cotton cultivars (Chu et al. 1999). However, as occurred with tomato, the actual effect of this factor on *B. tabaci* populations has been broadly questioned. For example, whitefly adults and nymphs showed positive correlations with hair density and length of hair on leaf lamina, midrib and vein (Raza et al. 2000; Bashir et al. 2001; Aslam et al. 2004). However, while hair density on midrib and gossypol glands on veins was positive and highly significantly correlated to whitefly population, the length of hair on leaf lamina was negatively and highly significantly correlated (Sial et al. 2003). Research carried out in Arizona, USA (Chu et al. 1998, 1999, 2002) to identify cotton plant characteristics related to *B. tabaci* colonization agreed with previous results (Ellsworth et al. 1993; Norman and Sparks 1996, 1997) that hairy leaf cotton cultivars harbor higher populations compared with glabrous cultivars. More recently, Chu et al. (2000, 2001) reported that other factors, including leaf color, morphology and leaf-age related effects on lysigenous glands and leaf trichome densities, may affect *B. tabaci* biotype B oviposition and nymphal densities. In another screening for cotton resistance against B-biotype of *B. tabaci*, adults, eggs and nymphs were significantly correlated to leaf hairiness, with seasonal variability due to leaf color, shape, and hairiness types (Alexander et al. 2004). A brief review by Walker and Natwick (2006) pointed out the mixed and sometimes contradictory results presented in these and other previous studies which associate the two different traits in cotton (smooth-leaf and okra-leaf) with reduced whitefly susceptibility, while in other studies, a slight effect, no effect, or even the opposite effect occurred. All these results together indicate that many morphological plant traits cumulatively contribute to whitefly population fluctuation (Sial et al. 2003).

Other studies have focused on the relationship of biochemical constituents of cotton leaves with whitefly populations. Significant negative correlation for total phenols, *o*-dihydroxy phenols, gossypol and tannins with egg, nymph and adult was established. Reducing sugars showed a significant positive correlation for egg and nymph, but not adults. Total and nonreducing sugars did not show any significant correlation with insect population (Raghuraman et al. 2004).

Recent field studies in California's Imperial Valley revealed consistent very high levels of resistance against the silverleaf whitefly in *Gossypium thurberi* Todaro, a wild cotton species native to Mexico and parts of the southwestern USA (Walker and Natwick 2006). However, the mechanisms of this resistance remains an enigma because both choice and no-choice experiments comparing oviposition and nymphal survival among *G. thurberi* and commercial cotton cultivars did not detect antibiosis or antixenosis (Walker and Natwick 2008).

Research on host plant resistance to *B. tabaci* in melon (*Cucumis melo* L.) is relatively recent, with most of the results obtained during the last decade. Simmons and McCreight (1996) developed a method in a 2-week open-choice greenhouse test to screen germplasm of 31 selected melon entries based on whitefly immature density and changes in plant biomass and tolerance. Another screening of 8 cultivars of export cantaloupes by counts of eggs, live nymphs and pupae, showed

that 'Amarelo' and 'Concorde' were the most resistant cultivars, with no correlation between whitefly densities and leaf pubescence (Morales 1997). This contradicted previous and later results which suggested the presence of the glabrous character of leaves as a resistance factor (Riley and Palumbo 1995a, b; Riley et al. 2001). Soria et al. (1999) reported the existence of genetic resistance to *B. tabaci* in both genotypes, *C. melo* variety *agrestis* 87 and *C. melo* TGR-1551. In the French West Indies, field trials have been conducted since 1997 to test a number of genotypes from the germplasm collection of INRA-Avignon, France. Results indicated that 10 genotypes had potential partial resistance against the B-biotype of *B. tabaci* and that this resistance would be independent from resistance to *Aphis gossypii* (Boissot and Pavis 1999). Later assays indicated that three Indian accessions, PI 414723, PI 164723, and 90625, and one Korean accession, PI 161375, had partial resistance to *B. tabaci*, although higher levels of resistance are needed for a genetic analysis (Boissot et al. 2000, 2003). After introgression of the *Vat* gene (responsible of *A. gossypii* resistance) into melon breeding lines from both the Korean and Indian germplasm sources, the ineffectiveness of *Vat* against *B. tabaci* was demonstrated, and a strategy to breed lines that express resistance to aphids and whiteflies in the short term was proposed (Sauvion et al. 2005).

The information on host resistance to *B. tabaci* in watermelon (*Citrullus lanatus*) is much more limited. Although current levels of resistance in commercial watermelon is quite inadequate, some useful sources of germplasm have been identified that can be used for the improvement of this crop and to incorporate resistance from wild species such as *C. colocynthis* (L.), into advanced breeding lines (Simmons and Levi 2002; Simmons et al. 2006).

Different breeding lines and varieties of *Cucurbita pepo* L. (zucchini and yellow crookneck squash) and accessions of two wild species, *C. ecuadorensis* Cutler and Whitaker and *C. martinii* Bailey, were evaluated for resistance to *B. argentifolii* and for severity of silvering symptoms, but no clear relationship was found between both factors (McAuslane et al. 1996). Other screenings have been conducted to compare whitefly resistance in different genotypes of *C. moschata* and *C. maxima* (Wessel-Beaver 1997a; Baldin et al. 2000). Several sources of silver-leaf resistance have been identified in *C. moschata* controlled by a single recessive gene (Wessel-Beaver 1997b; Wessel-Beaver and Paris 2000; Gonzalez-Roman and Wessel-Beaver 2002). In a more recent study under greenhouse conditions, the main squash cultivars available in the Brazilian market were compared for resistance to the B-biotype of *B. tabaci* (Alves et al. 2005).

Whitefly resistance in soybean (*Glycine max* L.) has been carried out during recent years mostly in the USA, Brazil, Pakistan and India. Significant differences in *B. argentifolii* densities were observed among 14 soybean genotypes in Georgia (USA), with Perrin, Cook and N88-91 harboring the lowest mean numbers of whiteflies (McPherson 1996; Lambert et al. 1997). Low abundance, parasitism and oviposition rates of *B. argentifolii* on certain soybean isolines in Florida (USA) were related to low trichome density (McAuslane et al. 1995a; McAuslane, 1996). Different field and controlled condition trials were conducted in Brazil to evaluate oviposition, non-preference and antibiosis of *B. tabaci* biotype B on different

soybean genotypes (Lima et al. 2002; Lima and Lara 2004). The obtained results strongly suggest that the resistance observed in some of these genotypes was stable (Do Valle and Lourencao, 2002). In Pakistan, 23 varieties of soybean were tested; G-9956 and AGS-344 were the most resistant against *B. tabaci* (Khaliq et al. 2000). In India, soybean line DS 1016 was consistently found to be a promising source of resistance to *B. tabaci* (Sridhar and Siddiqui 2001; Sridhar et al. 2003).

Elite germplasm from the peanut breeding program at the University of Florida (USA) and several commercial cultivars were evaluated for resistance to *B. argentifolii*, but only two genotypes supported fewer whiteflies (although not significantly) than the cultivar "Southern Runner" and no resistance was found in the peanut germplasm tested (McAuslane et al. 1995b). Another study was carried out to determine whether soybean could be used as a trap crop to reduce whitefly infestation in peanut and whiteflies preferred soybean (McAuslane et al. 1995a).

Research to evaluate whitefly resistance in mungbean (*Vigna radiata* L.) has been conducted in the past, especially in Pakistan. To highlight the results obtained during the past decade, out of 23 mungbean accessions, VC2755A was least susceptible while VCA 82 was the most susceptible accession (Arutkani and Ayyanathan 1999; Fargali et al. 1996). Moreover, NM-92 and NM-98 showed significantly lower mean whitefly population/leaf as compared with three other tested varieties (Khattak et al. 2004).

The resistance level of 19 common bean (*Phaseolus vulgaris* L.) genotypes to whiteflies was studied for 2 years in Brazil, with variable results depending on the plant age and the rainy or dry season (Boiça et al. 2008).

Wide screening assays were conducted in India on many genotypes of *Mentha arvensis*, *M. piperita*, *M. cardica* (*M. gracilis*), *M. citrata* (*M. piperita* var. *citrata*) and *M. spicata*, and their resistance potential against *B. tabaci* was compared (Singh and Singh 2004; Singh et al. 2004).

Thirty-eight plants of alfalfa (*Medicago sativa* L.) were evaluated for resistance to *B. argentifolii* in California, USA; 17 of them displayed low whitefly infestation and were categorized as potentially resistant. The plants were propagated vegetatively so that replicated measurements of whitefly performance could be made on each genotype. After different greenhouse and field assays, four genotypes demonstrated high whitefly resistance and three demonstrated moderate resistance (Teuber et al. 1999; Jiang et al. 2003).

Nine sweet-pepper (*Capsicum annuum* L.) genotypes obtained from the Asian Vegetable Research and Development Centre (Taiwan) and local cultivars were screened at the University of Agricultural Sciences, Bangalore (India); none were suitable for the development of *B. tabaci* (Maruthi et al. 2003).

Much of the research on whitefly resistance in cassava (*Manihot esculenta* Crantz) in recent years has been carried out at the International Center for Tropical Agriculture, in Colombia, and several cultivars were identified with high levels of resistance to the cassava whitefly, *Aleurotrachelus socialis* Bondar (Bellotti and Arias 2001). However, research on cassava resistance to *B. tabaci* continues to be limited. Nevertheless, a range of new cassava elite clones were assessed in experimental fields of the International Institute of Tropical Agriculture, in Ibadan,

Nigeria, and the researchers found that cassava genotypes 96/1089A and TMS 30572 supported the lowest whitefly infestation in all locations (Ariyo et al. 2005).

Three genotypes (PN 2KS, SH 3322 and IBD-2KS) of sunflower (*Helianthus annuus* L.) were found to be resistant against *B. tabaci* among nine genotypes tested in Pakistan, with a negative correlation between pest population and yield of genotypes (Aslam and Misbah-ul-Haq 2003).

Glossy-leaf phenotypes ('SC Glaze', 'SC Landrace', 'Green Glaze') of collard (*Brassica oleraceae* L.) were found to be the most resistant to *B. tabaci* as compared with normal non-glossy cultivars and hybrids (Jackson et al. 2000). The glossy-leaf characteristic results from phenotypes which express a reduced amount of leaf surface wax, and this gives the leaf a glossy appearance. Nonpreference appears to be the primary mode of resistance in certain collards.

Studies on the relative resistance or susceptibility of four sesame cultivars (*Sesamum indicum* L.) were conducted in Pakistan, and 'PR-14-2' was found to be the least susceptible to *B. tabaci* (Wadhero et al. 1998). Additional relevant research was conducted in Venezuela to characterize six sesame genotypes in relation to foliar acidity and the presence or absence of certain secondary metabolites, by an analysis of principal components based on these features. This analysis separated two sesame genotypes with greater foliar acidity values, which harbored less eggs and nymphs of whiteflies (nearly 10 and 50% of the total incidence in the other genotypes), relating foliar acidity with resistance of sesame to whiteflies (Laurentin et al. 2003).

Final Remarks

The past decade has been fruitful for the identification of new sources of host plant resistance against *B. tabaci*. The unique to date cloning of *Mi-1* gene in tomato underscores the necessity of isolating and cloning other resistance genes with direct application in IPM programs for other crops. This will be possible by a combination of classical genetic breeding programs with characterization of the already available plant resistance (innate and induced). It is currently indispensable to have a better understanding of how resistance genes work to increase the capacity of producing stable and long-lasting resistances to this pest. This can be achieved in the near future with the help of the tools of genetics, genomics, proteomics, and biochemistry (Kaloshian and Walling 2005). Very recent insights obtained from research on model organisms, such as *Arabidopsis* (Kempema et al. 2007; Zarate et al. 2007) will facilitate the advances in the knowledge of the plant-*Bemisia* interactions in cultivated plants.

In the case of tomato, research with mutant plants is being carried out in our laboratory to determine the main defense signaling pathways activated in the *Mi-1*-mediated resistance against *B. tabaci*. Moreover, high throughput technologies such as microarray analysis have been recently used to detect changes in the gene expression of tomato plants in response to whitefly feeding (McKenzie et al. 2005). Our group is currently starting to use this technology as well as the virus induced

gene silencing or VIGS, to select and characterize genes differentially up- or down-regulated in the innate and induced resistant responses of tomato against *B. tabaci*, in combination with plant bioassays to detect possible changes in resistance levels. This may also allow detection of possible overlaps in genetic profiles of the innate and induced resistant responses.

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