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# Neonicotinoid insecticide resistance among populations of *Bemisia tabaci* in the Mediterranean region of Turkey

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## Abstract

The study was conducted to evaluate whether *Bemisia tabaci* (Gennadius) (MEAM1, formerly B biotype) populations in Turkey have developed resistance to neonicotinoid insecticides. We collected *B. tabaci* from vegetable and cotton growing areas in the Mediterranean coastal region of Turkey. *B. tabaci* populations were collected from the following crops (and areas): *Solanum lycopersicum* L. (Aydıncık and Erdemli-Mersin), *Gossypium hirsutum* L. (Karataş-Adana), *Capsicum annuum* L. (Kumluca-Antalya), and *Cucumis sativus* L. (Samandağ-Hatay). We performed insecticide bioassays and biochemical assays to determine levels of susceptibility to acetamiprid, imidacloprid, thiacloprid, and thiamethoxam. The bioassays showed that most of the *B. tabaci* populations were resistant to all the neonicotinoids tested when compared with the laboratory insecticide-susceptible strain, SUD-S. The highest resistance factor was 2060 for imidacloprid at Kumluca and the lowest was 5.36 for thiamethoxam at Samandağ. Furthermore, the highest and lowest monooxygenase enzyme activity level of *B. tabaci* was in the Kumluca and Samandağ populations, respectively. The CYP6CM1 protein lateral flow assay results supported those of the biochemical assays. Our results support those reported elsewhere that enhanced monooxygenase activity, at least in part, is responsible for neonicotinoid resistance in *B. tabaci* populations.

**Key words:** whitefly, neonicotinoid resistance, bioassay, monooxigenase, CYP6CM1.

## Introduction

*Bemisia tabaci* (Gennadius), the sweet potato whitefly, (Hemiptera Aleyrodidae) is one of the major pests of cotton, soybean, tomato, pepper, eggplant and many other plants in greenhouses and farms in the Mediterranean region of Turkey (Tunç *et al*., 1983; Ulubilir and Yabas, 1995; Ulusoy *et al*., 1996). The sweet potato whitefly is a sap sucking insect that transmits more than 100 virus species and indirectly causes leaf damage by excreting honeydew which serves as a substrate for sooty mold (EFSA, 2013). Chemical control is the major pest suppression method applied in the Mediterranean region, and many different active ingredients are used. Because even a single individual can potentially cause considerable damage by virus transmission, that there aren't functioning thresholds for *B. tabaci* when virus is the concern because very low numbers of viruliferous whiteflies can cause significant loss, particularly if the crop is infected early in the production cycle. Therefore, farmers tend to use insecticides more frequently to control whitefly populations in a single crop cycle in the region. There are many support of government to biological and biotechnical control usage especially in the greenhouses, but the application of these techniques are limited. The Mediterranean region uses the largest proportion of total pesticide usage in Turkey, being up to 40% (Delen *et al*., 2005). Although organophosphates, carbamates, and pyrethroids are still commonly used insecticide groups in Turkey, the usage of neonicotinoids increased by 900-fold from 2001 to 2011 (Anonymous, 2012).

Imidacloprid, the first neonicotinoid introduced, exhibits excellent contact and systemic activity and, therefore, has been largely responsible for the sustained management of *B. tabaci* in horticultural and agronomic production systems worldwide (Jeschke and Nauen, 2008). In addition to imidacloprid, there are other neonicotinoids, e.g., acetamiprid and thiamethoxam, with have demonstrated efficacy against sucking insects including aphids, leafhoppers, mealybugs and whiteflies (Rauch and Nauen, 2003).

By 2013, the global neonicotinoid market was worth $4.65 billion (Sparks and Nauen, 2015). High selection pressure on some of the world's most destructive pests has increased problems of insecticide resistance. Since their introduction in 1991, resistance to neonicotinoids has developed rather slowly, but it is now established in some major field and greenhouse pests such as the brown planthopper, *Nilaparvata lugens* (Stal) (Liu *et al*., 2006), sweet potato whitefly, *B. tabaci* (Nauen *et al*., 2008), greenhouse whitefly, *Trialeurodes vaporariorum* Westwood, peach aphid, *Myzus persicae* (Sulzer), and housefly, *Musca domestica* L. (Nauen and Denholm, 2005; Bass *et al*., 2015). Neonicotinoid resistance in whiteflies is mainly attributable to enhanced monooxygenase activity (P450) due to elevated levels of CYP6CM1 (Karunker *et al*., 2008; Roditakis *et al*., 2011; Nauen *et al*., 2013). However, target site mutations in nAChR subunits have been detected in a few other pest species, such as *N. lugens* and *M. persicae* (Zewen *et al*., 2003; Tan *et al*., 2008; Bass *et al*., 2011; 2015).

Investigations into neonicotinoid resistance in whiteflies collected in Turkey have been limited to bioassay studies (Dağlı *et al*., 2007; Bahşi *et al*., 2012; Şahin *et al*., 2014). There have been no reports on the mechanisms of neonicotinoid resistance in Turkey, although it

is reasonable to assume that resistance is conferred by similar mechanisms to those described for whiteflies collected elsewhere, such as overexpression of CYP6CM1 (Karunker *et al*., 2008; Bass *et al*., 2015). However, the determination of the actual resistance status, as well as the resistance mechanisms, is important for whitefly management. For these reasons, the present study was carried out to survey whitefly populations collected in the Mediterranean Region of Turkey for resistance to four neonicotinoid insecticides by using leaf-dip bioassays, and secondly, to better understand the mechanism of resistance by using biochemical studies.

## Materials and methods

### Insecticides

Acetamiprid (20% SP, Mospilan; Nippon Soda), imidacloprid (350 g/l, Confidor; Bayer Crop Science), thiacloprid (250 g/l, Calypso; Bayer Crop Science) and thiamethoxam (240 g/l, Actara, Syngenta) were five serially diluted in distilled water that contained 0.1 g $L^{-1}$ Triton X-100 to improve leaf wetting to the required concentration (acetamiprid: 3000-0.5 ppm; imidacloprid: 10000-0.35 ppm; thiacloprid: 3000-1 ppm; thiamethoxam: 2400-0.1 ppm), depending on the whitefly population and insecticide.

### Whitefly strains

SUD-S is an insecticide-susceptible laboratory strain of *B. tabaci* obtained from Martin Williamson (Rothamsted Research, United Kingdom) for this study. The Karatas population was collected from a cotton (*Gossypium hirsutum* L.) field in Adana; the Aydıncık and Erdemli populations from tomato (*Solanum lycopersicum* L.) greenhouses in Mersin; the Samandağ population from a cucumber (*Cucumis sativus* L.) field in Hatay, and the Kumluca population from a pepper (*Capsicum annuum* L.) greenhouse in Antalya, in 2009 (figure 1). All populations were classified as MEAM1 (formerly B biotype), as described previously (Satar and Ulusoy, 2016). Each strain were cultured with at least 200-300 individuals and reared in the laboratory on cotton plants until they reach F2 and F3 generation. Each population was maintained without insecticide exposure at a 16 h photoperiod and 25 °C.

### Adult leaf-dip bioassays

For the adult *B. tabaci* leaf-dip bioassay, the experimental protocol of Nauen *et al*. (2008) was followed. Cotton leaf discs were dipped for 20 s into insecticide solutions diluted to the required test concentration. The leaf discs dipped in the diluent only served as controls. Leaf discs were then laid on an agar bed (10 g $L^{-1}$) in a plastic Petri dish, and air dried for 2 hours. Adults were anesthetized with $CO_2$ and the females were placed on the leaf discs. Afterwards, the Petri dishes were covered with a close-fitting, ventilated lid. Bioassays consisted of four replicates per concentration, each with a group of 20-30 females. They were maintained at 25 °C, with adult mortality scored after 48 hours.

### Fluorometric microplate assay to measure 7-ethoxycoumarin O-de-ethylation

Cytochrome P450-dependent monooxygenase activity was determined with the O-de-ethylation of 7-ethoxycoumarin, according to the methodology of Stumpf and Nauen (2001). The amount of 7-hydroxycoumarin released from the sample during incubation was quantified with a fluorimeter (Victor 3V, PerkinElmer) at 465 nm emission wavelength h and 390 nm excitement wavelength. The assay was replicated twice with each strain. The total protein amount was determined at 600 nm, according to the Bradford Reagent Method (Sigma), using Bovine Serum Albumin (BSA) as the standard.

### Detection of CYP6CM1 protein level

A lateral flow assay assembly described by Nauen *et al*. (2015) was used to determine whether the resistance of neonicotinoids was related to the CYP6CM1 protein. The test was carried out with three individuals. The results were then associated with neonicotinoid resistance for each population. To determine CYP6CM1 protein levels, seven *B. tabaci* individuals from each population were used. The tests were conducted as described by Nauen *et al*. (2015) and replicated three times.

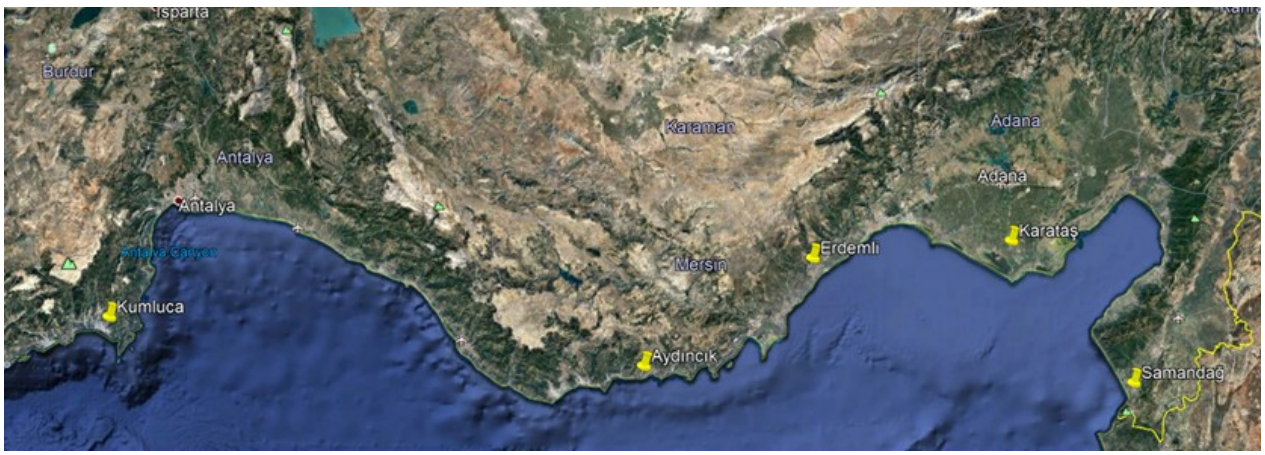


**Figure 1.** Locations of *B. tabaci* collected in Mediterranean Region of Turkey.

### Data analysis

Dose-response data were subjected to probit analysis with the Polo Plus software (LeOra Software, Berkeley, CA). The resistance factor was calculated by dividing the $LC_{50}$ of the field or greenhouse strains by that of the susceptible reference strain. Monooxygenase activity was determined by dividing the total protein amount by the level of monooxygenase activity. Data for monooxygenase activity were analyzed with one-way ANOVA, followed by the Tukey test ($P \leq 0.05$), using the SPSS statistics (Version 17) program.

## Results and discussion

In this study, five *B. tabaci* populations collected in the Mediterranean region of Turkey were examined for resistance to four neonicotinoid insecticides (table 1). Each population was distinct but in most cases there was a similar pattern of resistance to these four compounds. While the Samandağ population showed the lowest level of resistance to imidacloprid, thiacloprid, and thiamethoxam, the Kumluca population was the most resistant to both acetamiprid and imidacloprid, with a 68-fold resistance factor (RF) and an over 2,000-fold RF, respectively. The Erdemli population was most resistant to thiacloprid with an RF of 272. The Karatas population was the least resistant to acetamiprid, but most resistant to thiacloprid. This population was omitted for the thiamethoxam treatment because of experimental problems. Dağlı *et al*. (2007) and Bahşi *et al*. (2012) reported that whiteflies collected in Kumluca had the highest RF for different neonicotinoid insecticides, a result confirmed by our study. Şahin and İkten (2017) examined some Antalya populations, including Kumluca, but they found much lower RF values than our study. This difference could be related to the population of the pest, the amount of the neonicotinoid insecticide applied and the frequency of application.

In the present study, whole populations showed some degree of cross-resistance to all insecticides. In particular, the Kumluca and Aydıncık populations had very high cross-resistance, namely for imidacloprid (2059.6 RF, 455.2 RF, respectively) and thiamethoxam (219.8 RF, 155.3 RF, respectively). Different factors including biological, operational, and ecological factors can affect the development of resistance in a pest and these parameters at the different regions can cause variation at cross-resistance among neonicotinoids (Prabhaker *et al*., 2005). The slope value obtained from probit analysis gives information about the variance of the population. If the population produces a high slope ($b > 2$), this is an indication of a relatively homogeneous population. The line shows low slope ($b < 1$) is indicative of a heterogeneous population showing large variance in its response (Yu, 2015). In this study, the slopes of most dose-mortality curves were extremely low, indicating heterogeneity in neonicotinoid resistance in these populations.

The Karatas (cotton) and Samandağ (cucumber) populations were sampled from open production areas (fields) and were less resistant than the Erdemli, Aydıncık, Kumluca populations collected from greenhouses (table 1). While neonicotinoid resistance fluctuated in open fields, there was a continual increase in protected areas in China from 2005 to 2014 (Yao *et al*., 2017). Similar results were also reported by Roditakis *et al*. (2005) who detected 760-fold resistance to imidacloprid in a greenhouse population in Crete (Greece) whereas populations from open areas in the same study were sometimes even more susceptible than the sensitive reference strain.

The greenhouse environment reduces evaporation, and minimizes both pest entry and the escape of expensive biological control agents. Thus, less mixing of resistant and susceptible populations in greenhouses leads to increase of insecticide resistance than the open field. Routine practices can lead to lower population size but also to the selection of resistant individuals with more genetic differentiation than in the open field (Ovcarenko *et al*., 2014). Kumluca is an important greenhouse area of Turkey and pesticide usage is comparatively high. Because populations in greenhouse environments can survive all year round without migrating, it is likely that the proportion of surviving heterozygotes results in homozygotes expressing higher levels of resistance. Since the profit from greenhouse products is generally higher, the tolerance of farmers to pests is lower. Continuing epidemic between 1970s-1980s caused changing of the crop pattern. The cotton production dramatically got abounded and the farmers directed to other crops such as vegetables, corn, citrus and greenhouse production (Yurdakul and Emeksiz, 1994). The whitefly tolerance of the farmers in the region due to this historical event is low. Therefore, insecticides can be applied even at the lowest pest population levels at above label rates and frequency but such practices can cause the development of resistance.

Karatas and Samandağ are close provinces, at sea level and for the most part their climate is cooler in the summer and warmer and wetter in the winter than the typical Mediterranean climate. The farm areas are quite small and owners try to meet their own needs, especially in Samandağ is a microclimatic region, so the insecticide resistance very low compare to others. The low resistance can be caused by the migrating individuals which is from heavily sprayed some fields. Besides that, Karatas has the irrigation problem and government support for cotton is not sufficient for the area farmers. So, they try to minimize their cost, including belongs to insecticide usage. These areas have many different cultivated plants that serve as harbours for many hemipteran pest species. The owner of each cultivated area uses distinctive agricultural practices, including different pest control methods, and irrigation and fertilization systems. Pests are therefore exposed to different pesticide regimes that can trigger different physiological and genetic responses, such as resistance development. The whitefly populations having different resistance levels can easily migrate from one cultivated field to another. Whiteflies can fly 2.2 miles within three hours under farm conditions (Byrne *et al*., 1994). Mating between whiteflies with higher and lower insecticide resistance can reduce the speed of the development of resistance.

**Table 1.** Log-dose probit mortality data obtained for different neonicotinoids tested against female adults of Turkish *B. tabaci* populations (F2-F3 generation) in leaf dips bioassays (48 h).

| Insecticide | n | Population | $LC_{50}$ (mg $L^{-1}$) (95% confidence limits) | $LC_{95}$ (mg $L^{-1}$) (95% confidence limits) | Slope (± SE) | RF |
|---|---|---|---|---|---|---|
| Acetamiprid | 429 | Aydıncık | 95.8 (66.8-125.5) | 828.9 (600.1-1315.8) | 1.8 ± 0.2 | 30.7 |
| | 455 | Erdemli | 50.1 (33.5-69.5) | 1334.7 (802.7-2763.6) | 1.2 ± 0.1 | 16.0 |
| | 496 | Karataş | 39.9 (19.8-65.3) | 1920.5 (1051.7-4791.5) | 1.0 ± 0.1 | 12.8 |
| | 408 | Kumluca | 213.1 (175.5-252.8) | 948.6 (716.5-1438.1) | 2.5 ± 0.3 | 68.2 |
| | 412 | Samandağ | 95.5 (60.4-141.4) | 3450.2 (1963.8-7447.2) | 1.1 ± 0.1 | 30.6 |
| | 574 | SUD-S | 3.1 (2.0-4.6) | 61.2 (32.9-163.2) | 1.3 ± 0.1 | |
| Imidacloprid | 414 | Aydıncık | 277.2 (186.4-401.9) | 16981.7 (8773.2-41991.0) | 0.9 ± 0.1 | 455.2 |
| | 485 | Erdemli | 130.6 (38.6-262.2) | 24158.5 (7111.2-331400.5) | 0.7 ± 0.1 | 214.5 |
| | 504 | Karataş | 56.9 (33.8-97.2) | 2557.0 (992.7-12133.7) | 1.0 ± 0.1 | 93.4 |
| | 409 | Kumluca | 1254. 3 (831.9-1837.2) | 28088.6 (13771.2-91743.0) | 1.2 ± 0.1 | 2059.6 |
| | 441 | Samandağ | 14.4 (4.9-32.4) | 14383.23 (5600.6-54234.1) | 0.6 ± 0.1 | 23.7 |
| | 556 | SUD-S | 0.7 (0.4-0.9) | 7.6 (4.8-15.2) | 1.5 ± 0.2 | |
| Thiacloprid | 405 | Aydıncık | 282.4 (203.3-372.8) | 3616.0 (2280.7-7201.2) | 1.5 ± 0.2 | 88.1 |
| | 435 | Erdemli | 872.3 (573.6-1092.0) | 3516.6 (2512.3-7557.3) | 2.7 ± 0.5 | 272.0 |
| | 334 | Karataş | 334.7 (193.4-425.0) | 817.8 (631.5-1570.3) | 4.2 ± 0.7 | 104.4 |
| | 407 | Kumluca | 442.1 (272.7- 624.4) | 4057.5 (2460.5-9866.3) | 1.7±0.2 | 137.8 |
| | 405 | Samandağ | 228.4 (146.6 -343.2) | 13572.2 (6246.0-43628.4) | 0.9 ± 0.1 | 71.2 |
| | 436 | SUD-S | 3.2 (1.7-5.4) | 23.7 (12.1-95.0) | 1.9 ± 0.3 | |
| Thiamethoxam | 395 | Aydıncık | 323.2 (216.5-473.6) | 2504.1 (1357.3-8303.6) | 1.9 ± 0.2 | 155.3 |
| | 420 | Erdemli | 281.6 (196.5-370.2) | 2862.3 (1746.0-6795.8) | 1.6 ± 0.2 | 135.3 |
| | 442 | Kumluca | 457.7 (330.2-630.7) | 4618.8 (2566.3-12853.4) | 1.6 ± 0.1 | 219.8 |
| | 292 | Samandağ | 11.2 (4.6-20.8) | 646.2 (281.2-2641.4) | 0.9 ± 0.1 | 5.4 |
| | 487 | SUD-S | 2.1 (0.7-3.0) | 16.7 (9.4-127.1) | 1.8 ± 0.4 | |

RF represents the resistance factor which was calculated by dividing the $LC_{50}$ of a field population by that of SUD-S.

All populations in the present study showed an increased level of 7-ethoxycoumarin O-deethylase (ECOD) activity compared to the SUD-S strain (table 2). Although the lowest level of enzyme activity was detected in the SUD-S strain, significant statistical differences were not detected between it and the other populations, except for the Kumluca population. The Kumluca population exhibited the highest level of enzyme activity, which was significantly different from the SUD-S, the Karataş and Samandağ populations ($F = 14.606$, $df = 6, 7$, $P < 0.001$; table 2). Regarding the bioassay results, the RF values for all insecticides were low for the Samandağ population, followed by the Karatas population (table 2). The Kumluca population exhibited the highest RF values for three of the neonicotinoids and significantly higher enzyme activity than all other populations. Some researchers have shown that neonicotinoid resistance in some economically important

**Table 2.** Monooxigenase activity of different Turkish *B. tabaci* populations (ng/30 min/ngprot).

| Populations | Monooxigenase ± SE (pmol/30 min/mgprot) |
|---|---|
| Aydıncık | 168.0 ± 7.0 ab |
| Erdemli | 188.5 ± 26.5 ab |
| Karataş | 139.5 ± 8.5 a |
| Kumluca | 230.0 ± 19.0 b |
| Samandağ | 132.5 ± 3.5 a |
| SUD-S | 129.5 ± 0.5 a |

Means within the column followed by the same letter are not significantly different (Tukey HSD test, α:0.05).

pests is conferred by mutations on the nAChR subunits (Zewen *et al*., 2003; Tan *et al*., 2008; Bass *et al*., 2011; Shi *et al*., 2012), but this has not been reported in whiteflies, for which the major mechanism reported is detoxification by the enzyme cytochrome P450 monooxygenases (Karunker *et al*., 2008; Nauen *et al*., 2008; Wang *et al*., 2009; Bass *et al*., 2015). A significant correlation between ECOD activity and imidacloprid resistance level was demonstrated for neonicotinoid resistant whiteflies collected in Germany, Greece, Israel and Spain (Nauen *et al*., 2002; Rauch and Nauen, 2003; Roditakis *et al*., 2009). The neonicotinoid resistance levels registered in our study were also linked to elevated monooxygenase activity, even though the general P450 enzyme activity level determined for 7-EC was not as high as the bioassay results.

Over-expression of CYP6CM1 has been demonstrated to confer imidacloprid resistance in MEAM1 and MED (formerly Q biotype) of *B. tabaci* (Karunker *et al*., 2008; Roditakis *et al*., 2011; Nauen *et al*., 2013). Recombinantly expressed CYP6CM1vQ was shown to me-

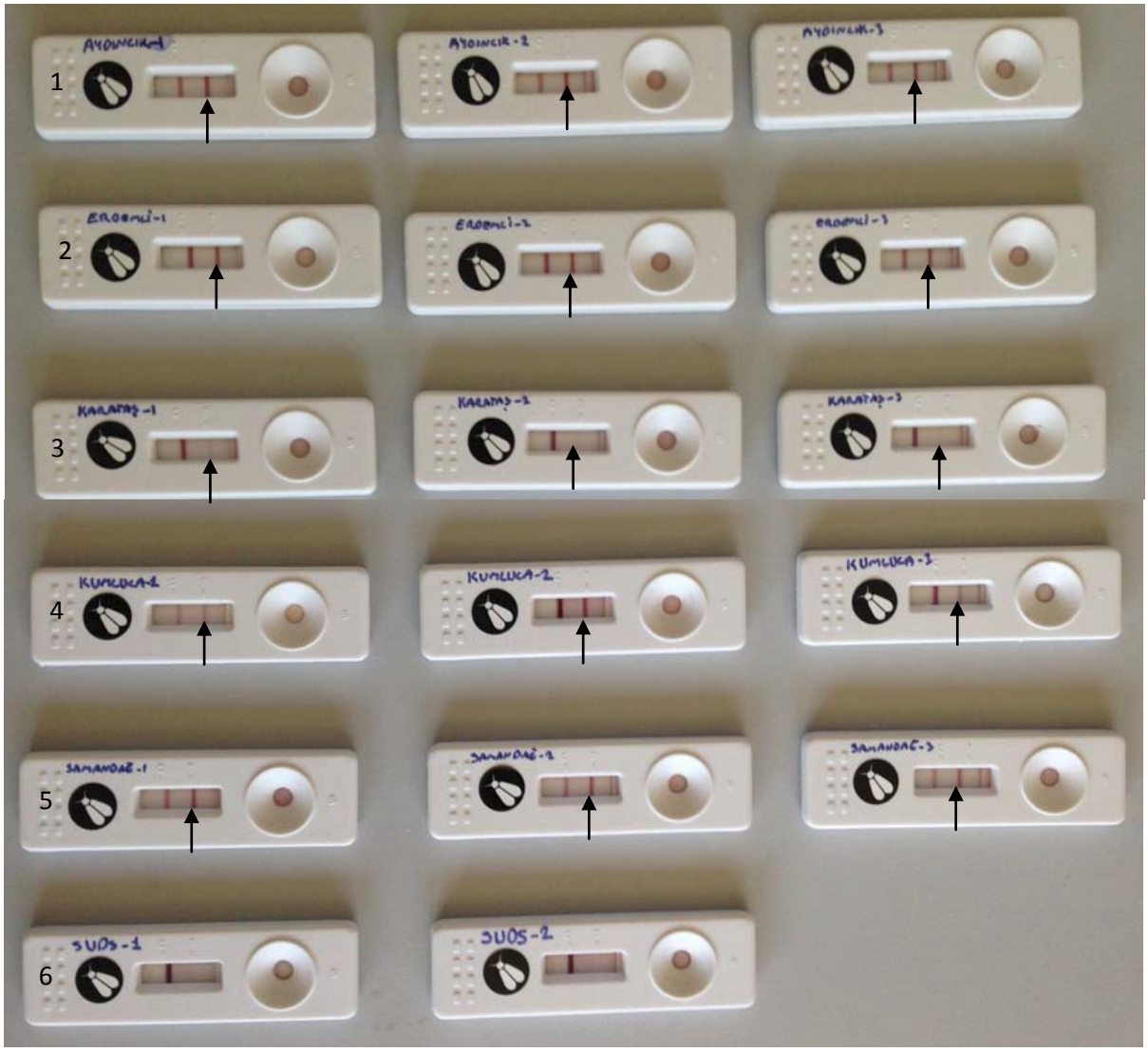


**Figure 2.** Determination of the CYP6CM1 protein level with a recently developed lateral flow assay machine (Nauen *et al*., 2015) in females of strains of Turkish populations of *B. tabaci*, namely (1) Aydıncık, (2) Erdemli, (3) Karataş, (4) Kumluca, (5) Samandağ, and the reference population, (6) SUD-S. The arrow indicates the second band which only occurs if the CYP6CM1 protein expression level is high enough to confer significant levels of imidacloprid resistance.

tabolize imidacloprid to its 5-hydroxy form (Roditakis *et al*., 2011), thus explaining the resistance in the phenotype. An increased transcription level is directly correlated with an increase in the amount of enzyme (Roditakis *et al*., 2011). The lateral flow assay kit (Nauen *et al*., 2015) used in this project confirmed elevated levels of CYP6CM1 in the resistant populations collected in this study in Turkey (figure 2). The test is simple, and reliable results are obtained within minutes, even under field conditions (Nauen *et al*., 2015). Based on our results, we conclude that the over-expression of CYP6CM1 contributes to the high levels of neonicotinoid resistance observed in all the studied Turkish populations of *B. tabaci*. Illias *et al*. (2015) found that neonicotinoid resistance is related to the P450 monooxygenase enzyme in *B. tabaci* by using the RNA-Seq method, and that the eight P450 genes which were reported in previous studies are also overexpressed in the resistant population. However, the correlation detected was only CYP303 and CYP6CX3 genes with imidacloprid and acetamiprid resistance. In another study, there was a significant increase in the amount of the CYP6CM1 gene in response to thiacloprid applications, with and without the P450 inhibitor piperonyl butoxide (PBO) (Zimmer *et al*., 2016). These studies showed that resistance to neonicotinoids in the monooxygenase family of enzymes may be associated with different genes in different populations. Therefore, on the basis of the results of our study, different genes should be examined for a more comprehensive evaluation.

In conclusion, our results confirmed the presence of neonicotinoid resistance in five Turkish whitefly populations and provided evidence for the first time that resistance is biochemically driven by elevated levels of cytochrome P450 monooxygenases, in particular CYP6CM1. Future experiments should focus on mutations in nAChR subunits and other P450 monooxygenase enzyme genes with RNA-Seq methods to more clearly understand the resistance mechanisms of *B. tabaci* to neonicotinoids. The growers should take into account resistance is important problem of *B. tabaci* for the region and rotation of insecticide has different mode of action is the first step to stop this process especially for open field like cotton. Other control methods like cultural and biological control especially at greenhouse can be a good alternative for sustainable agriculture.

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GS and MRU conceived and designed research. GS conducted experiments. RN contributed new reagents. GS, KD and RN analyzed data. GS wrote the manuscript. GS thanks the Cukurova University BAP Department for their support of the doctorate project (D3BAP2012). The authors thanks Martin Williamson (Rothamsted Research, UK) for supplying SUD-S susceptible whiteflies strain. We also thank Recep Ay (Suleyman Demirel University, Plant Protection Department, Isparta, Turkey), Graham Moores, Selcan Alptekin (Rothamsted Research, UK), Sedef Nehir El and Şebnem Şimşek (Ege University, Food Engineering Department, İzmir, Turkey) and Osman Gülnaz (Cukurova University, Department of Biology, Faculty of Arts and Sciences, Turkey). The authors also thank Gregory T. Sullivan (School of Earth and Environmental Sciences, Brisbane, Australia) for editing the English in an earlier version of this manuscript.

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