-Communication 1-

Houndété TA, Kétoh GK, Hema OSA, Brévault T, Glitho IA and Martin T (2010). Insecticide resistance in field populations of *Bemisia tabaci* (Hemiptera: Aleyrodidae) in West Africa. *Pest Manag. Sci*. **66**: 1181-1185. *site web:* wileyonlinelibrary.com) DOI 10.1002/ps.2008

Pest Management Science: http://onlinelibrary.wiley.com/journal/10.1002/(ISSN)1526-4998;jsessionid=E6B74AAE3F29061B769F91166D3469DF.f03t04

Pest Management Science SCI

Editor-in-Chief: Stephen O. Duke

Impact Factor: 2.743

ISI Journal Citation Reports © Ranking: 2013: 4/90 (Entomology); 11/79 (Agronomy)

Online ISSN: 1526-4998

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Received: 18 September 2009 Revised: 26 April 2010 Accepted: 15 June 2010 Published online in Wiley Online Library: 18 August 2010

(wileyonlinelibrary.com) DOI 10.1002/ps.2008

Insecticide resistance in field populations of *Bemisia tabaci* (Hemiptera: Aleyrodidae) in West Africa

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Abstract

BACKGROUND: The tobacco whitefly, *Bemisia tabaci* Gennadius (Hemiptera: Aleyrodidae), has developed a high degree of resistance to several chemical classes of insecticides throughout the world. To evaluate the resistance status in West Africa, eight insecticides from different chemical families were tested using the leaf-dip method on four field populations collected from cotton in Benin, Togo and Burkina Faso.

RESULTS: Some field populations showed a significant loss of susceptibility to pyrethroids such as deltamethrin [resistance ratio (RR) 3–5] and bifenthrin (RR 4–36), to organophosphates (OPs) such as dimethoate (RR 8–15) and chlorpyrifos (RR 5–7) and to neonicotinoids such as acetamiprid (RR 7–8) and thiamethoxam (RR 3–7). *Bemisia tabaci* was also resistant to pymetrozine (RR 3–18) and to endosulfan (RR 14–30).

CONCLUSION: The resistance of *B. tabaci* to pyrethroids and OPs is certainly due to their systematic use in cotton treatments for more than 30 years. Acetamiprid has been recently introduced for the control of whiteflies. Unfortunately, *B. tabaci* populations from Burkina Faso seem to be already resistant. Because cross-resistance between these compounds has never been observed elsewhere, resistance to neonicotinoids could be due to the presence of an invasive *B. tabaci* biotype recently detected in the region.

Keywords: *Bemisia tabaci*; insecticide resistance; cotton; West Africa

1 INTRODUCTION

The tobacco whitefly, *Bemisia tabaci* Gennadius (Hemiptera: Aleyrodidae) is a serious pest in many cropping systems throughout the world.[1] *Bemisia tabaci* causes hundreds of millions of dollars in crop damage and lost yields annually.[1,2] In addition, *B. tabaci* has developed resistance to many insecticides from different chemical classes.[3–6] For cassava crops, it is considered a pest of primary importance owing to high infestation levels on plants.[7] Cotton and vegetable crops such as tomato, pepper and melon can be seriously damaged by whiteflies.[3] In tropical and subtropical agricultural systems, the expansion of *B. tabaci* has been largely promoted by the indiscriminate use of insecticides and monoculture.[7] In West Africa, population outbreaks were observed in 1998 in cotton fields in Burkina Faso, Mali and Côte d'Ivoire.[8,9] In addition, development of broad-spectrum resistance to both organophosphates and pyrethroids was described in populations found on Sudanese cotton.[4,5] Although the development of insecticide resistance in whiteflies has long been recognised around the world, associations with biotypes were not made until 1986–1989.[10] In Burkina Faso, *B. tabaci* populations were shown to be resistant to cypermethrin, methamidophos and omethoate.[8] In Benin and Togo, no baseline data are available on *B. tabaci* population susceptibility to insecticides. However, recent data on chlorpyriphos insecticide efficacy from laboratory bioassays on adults showed that this insecticide is no longer effective in Benin populations, and modified AChE was found to be responsible for resistance.[11]

The present study was initiated to establish baseline susceptibility of *B. tabaci* to different families of insecticide sprayed on cotton fields in Benin, Togo and Burkina Faso (West Africa). Leaf-dip bioassays using eight insecticides were conducted on adults collected from cotton.

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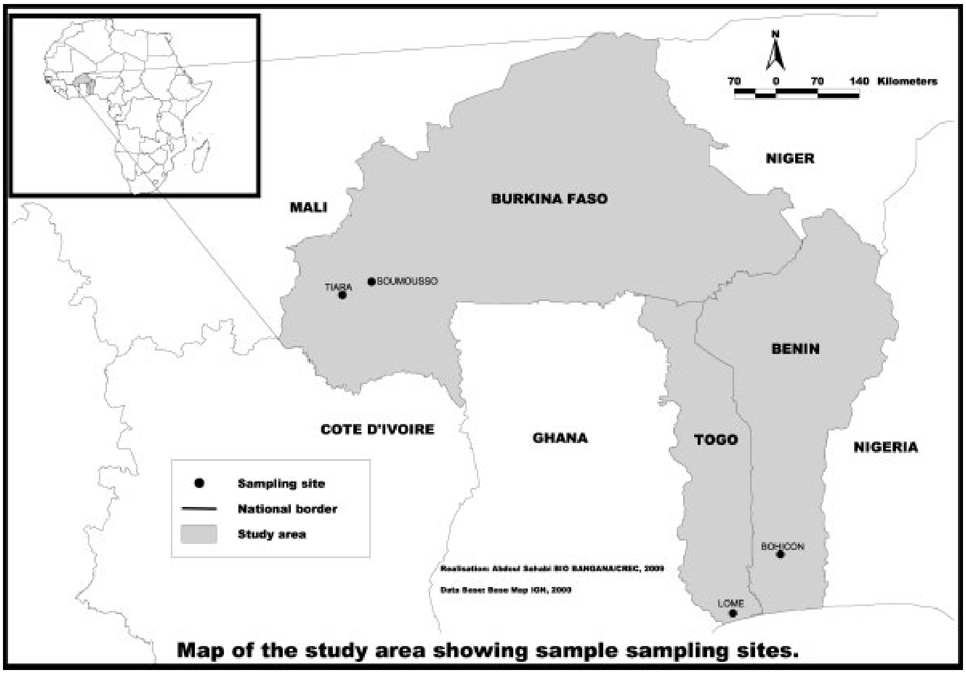

Figure 1. Map of the study area, showing the sampling sites.

2 MATERIALS AND METHODS

2.1 Whiteflies

Adults of *B. tabaci* were collected in 2006 and 2008 from cotton (*Gossypium hirsutum* L.) at the INRAB experimental research station at Bohicon (Benin), at the University of Lome (Togo) and in commercial fields at Soumousso and Tiara (Burkina Faso) (Fig. 1). *Bemisia tabaci* adults were collected from plants using a mouth aspirator, then confined in a wooden rearing cage (50 × 35 × 35 cm) containing cotton seedlings and returned to the laboratory within 2–5 h. Whiteflies of both sexes and variable age were tested the same day or the following day.

2.2 Insecticides

The following formulated insecticides were used for bioassays: bifenthrin 100 g L^{-1} EC (Talstar 10 EC), dimethoate 400 g L^{-1} EC (Callidim 400 EC), chlorpyrifos 480 g L^{-1} EC (Pyrical 480 EC), endosulfan 350 g L^{-1} EC (Rocky 350 EC) and acetamiprid 200 g L^{-1} SL (Mospilan 200 SL) provided by Arysta LifeScience (Noguères, France); deltamethrin 25 g L^{-1} EC (Decis 25 EC) obtained from Aventis CropScience (Lyon, France); thiamethoxam 240 g L^{-1} SC (Actara 240 SC) and pymetrozine 500 g kg^{-1} WG (Chess/Plenum 50 WG) from Syngenta Crop Protection AG (Basel, Switzerland).

2.3 Leaf-dip bioassay

A leaf-dip bioassay method was performed on the basis of previous studies.[12,13] Discs (55 mm diameter) of cotton leaves were dipped for 10 s in aqueous dispersions of insecticide formulation, or distilled water for controls. Leaf discs were air dried for 20 min. The discs were then positioned on an agar-coated (7 g L^{-1}) petri dish (55 mm diameter). Adults of *B. tabaci* (20–30 mixed sex) were removed from cotton leaves with a mouth aspirator, transferred into small plastic vials, held at −20 °C for 80 s and placed onto the treated leaf discs. Each petri dish was then sealed with a transparent ventilated lid. When adults recovered from chilling, dishes were stored upside down and maintained at 24 ± 2 °C, 52 ± 5% RH and a 12 : 12 h light : dark photoperiod. Mortality was recorded 48 h later. Three replicates were carried out for each concentration of insecticide and untreated control. Mortality in the control was always <10%, and data from all bioassays were corrected for control mortality using Abbott's formula.[14]

2.4 Data analysis

All bioassay replicates were combined for analysis. LC_{50} values were calculated by global optimisation by simulated annealing (GOSA), available at http://bio-log.biz. The resistance ratios (RRs) were calculated relative to the most susceptible field populations.

3 RESULTS

Populations from Burkina Faso showed higher resistance to all insecticides tested than populations from Benin and frequently from Togo (Table 1). The Bohicon (Benin) population was the most susceptible to deltamethrin, chlorpyrifos, dimethoate, endosulfan and acetamiprid, but not to bifenthrin, thiamethoxam and pymetrozine, which were more toxic on the Lome (Togo) population. These two populations were used as reference to calculate the resistance ratio. The highest resistance to deltamethrin and bifenthrin was observed in the Tiara (BF) population, which exhibited respectively a 4.7-fold and a 36-fold resistance. Whitefly populations in Tiara (BF) and Soumousso (BF) showed greater resistance to dimethoate (15.1- and 8.4-fold respectively) than populations in Lome (Togo) (1.6-fold). Populations displayed significant resistance to endosulfan, ranging from 14.3-fold in Lome (Togo) to 15.8-fold in Soumousso (BF) and 30-fold in Tiara (BF). *Bemisia tabaci* from Burkina Faso appeared to be significantly resistant to acetamiprid (7–8-fold). The highest resistance to acetamiprid was measured for Tiara (BF) (7.8-fold), followed by Soumousso (BF) (6.7-fold). Lome (Togo) was susceptible to acetamiprid (1.5-fold). This population also displayed high susceptibility to thiamethoxam, while the Tiara (BF) population displayed slight but significant resistance (6.6-fold). In Tiara (BF), the *B. tabaci* population displayed

Table 1. Toxicity of various insecticides against field populations of *Bemisia tabaci* collected from cotton in Benin, Togo and Burkina Faso (West Africa) using leaf-dip bioassays

Insecticide	Population	N^a	$LC_{50}{}^b$ (mg L^{-1})	Confidence limits 95%	Slope (± SE)^c	RR^d
Deltamethrin	Bohicon - Benin	1422	11 c	7.4–15	1.4(±0.4)	–
	Lomé - Togo	681	17 bc	10–25	1.7(±0.8)	1.6
	Soumousso - BF	1060	34 ab	16–53	1.5(±0.7)	3.1
	Tiara - BF	1017	53 a	33–73	1.2(±0.3)	4.7
Bifenthrin	Bohicon - Benin	1509	2.3 c	0.91–3.7	0.9(±0.3)	4.0
	Lomé - Togo	725	0.57 d	0.29–0.85	1.2(±0.3)	–
	Soumousso - BF	1184	6.5 b	5.4–7.7	4.8(±2.2)	11
	Tiara - BF	986	21 a	14–27	1.5(±0.4)	36
Chlorpyrifos	Bohicon - Benin	1309	3.6 b	2.6–4.6	0.8(±0.1)	–
	Lomé - Togo	698	19 a	14–24	2.4(±0.6)	5.3
	Soumousso - BF	1094	24 a	15–34	2.0(±1.0)	6.7
	Tiara - BF	1018	20 a	15–25	2.2(±0.8)	5.6
Dimethoate	Bohicon - Benin	513	350 b	193–506	1.5(±0.6)	–
	Lomé - Togo	581	541 b	388–695	2.8(±2.0)	1.6
	Soumousso - BF	1277	2927 a	2057–3797	1.9(±0.6)	8.4
	Tiara - BF	1255	5292 a	3452–7133	1.1(±0.3)	15
Endosulfan	Bohicon - Benin	1201	0.29 b	0.2–0.35	1.8(±0.4)	–
	Lomé - Togo	694	4.1 a	2.7–5.6	3.1(±2.2)	14
	Soumousso - BF	1045	4.6 a	2.9–6.3	2.0(±1.0)	16
	Tiara - BF	999	8.8 a	4.8–13	1.2(±0.4)	30
Thiamethoxam	Bohicon - Benin	426	5.8 b	3.9–7.7	1.2(±0.3)	3.4
	Lomé - Togo	646	1.7 c	0.90–2.5	1.7(±0.7)	–
	Soumousso - BF	1109	8.6 ab	4.2–13	1.1(±0.4)	5.1
	Tiara - BF	1184	11 a	8.1–14	1.1(±0.2)	6.6
Acetamiprid	Bohicon - Benin	500	3.0 b	1.3–4.7	0.8(±0.2)	–
	Lomé - Togo	664	4.5 b	2.4–6.6	3.0(±2.3)	1.5
	Soumousso - BF	1118	20 a	9.8–30	0.9(±0.3)	6.7
	Tiara - BF	1158	24 a	13–34	1.2(±0.3)	7.8
Pymetrozine	Bohicon - Benin	486	4.8 b	3.8–5.8	0.8(±0.1)	2.9
	Lomé - Togo	613	1.7 c	1.0–2.3	1.1(±0.3)	–
	Soumousso - BF	1004	12 abc	1.0–28	0.5(±0.2)	7.3
	Tiara - BF	1046	30 a	15–45	0.8(±0.2)	18

^{a} N = number of whiteflies tested.
^{b} For each insecticide, LC_{50} values with the same letter are not significantly different.
^{c} SE = standard error.
^{d} RR: resistance ratio = LC_{50} field population/LC_{50} Bohicon (Benin) population.

significant resistance (18-fold) to pymetrozine but similar susceptibility to that in Soumousso (BF). The population from Bohicon (Benin) was significantly more resistant to pymetrozine (2.9-fold) and thiamethoxam (3.4-fold) than the Lome (Togo) population.

4 DISCUSSION

This study showed evidence of resistance to pyrethroids and organophophates (OPs) of *B. tabaci* populations collected in cotton fields from Burkina Faso. However, populations from all three countries showed a significant loss of susceptibility to pyrethroids such as deltamethrin and to organophosphates such as dimethoate compared with the usual susceptible reference strain Sud-S[15–17] tested elsewhere using the same method. Resistance was higher for deltamethrin than for bifenthrin, and higher for dimethoate than for chlorpyrifos. This pattern of resistance could be linked to the use of these insecticides in cotton farming systems. Bifenthrin has been scarcely used in cotton in West African countries because of its high cost compared with other pyrethroids such as cypermethrin and deltamethrin primarily aimed at controlling bollworms. Whitefly populations from Benin and Togo seem to be susceptible to bifenthrin compared with the Sud-S strain.[15] Accordingly, bifenthrin could potentially be used in resistance management programmes for whitefly control, particularly on vegetables in the southern growing areas. Among organophosphates, dimethoate and omethoate were largely used during the 1980s and 1990s against *B. tabaci* at the end of the cotton season for the prevention of cotton stickiness.[18] These results are consistent with the insecticide resistance of *B. tabaci* populations from Burkina Faso already observed with the yellow sticky trap technique used in cotton fields.[8] The failure of conventional insecticides such as pyrethroids and organophosphates to control field populations of *B. tabaci* has already been reported in Burkina Faso,[19] as in other parts of the world.[3,15,16,20] The occurrence of resistance in *B. tabaci* populations from West Africa to some pyrethroids and OPs which have different modes of action indicates the possible presence of multiple resistance mechanisms. Such mechanisms may involve metabolic resistance associated with elevated activity of esterase in association with target-site resistance due to the selection of modified sodium channel (knockdown resistance) or insensitive synaptic acetylcholinesterase.[11,17,21]

Since 1999, endosulfan has replaced pyrethroids from the beginning of the cotton flowering stage to mid-August, in order to manage pyrethroid resistance in the cotton bollworm *H. armigera*.[22,23] As shown among populations of whiteflies, resistance to endosulfan is associated with a mutation in the γ-aminobutyric acid (GABA) receptor subunit gene.[24] Because of the high risk of negative impact on user health and environment, endosulfan has been banned in West Africa since 2008. Although resistance frequencies generally decline in the absence of insecticide selection, resistance alleles can persist at sufficient frequency to confer cross-resistance to novel insecticides interacting with the cyclodiene binding site, such as fipronil.[25]

One of the most interesting findings is the obvious resistance of Burkina Faso populations to both neonicotinoids tested and to pymetrozine, a compound that has never been commercially used in that region. Cross-resistance between neonicotinoids and pymetrozine was already observed in a *B. tabaci* strain from Spain.[26] Moreover, these authors showed evidence for the stability of this type of resistance in the absence of selection pressure and a steady decrease in the potency of all the neonicotinoids against field strains of *B. tabaci* over a period of 4 years. The existence of strong resistance in the cotton whitefly in Spain has demonstrated the potential of this pest to adapt and resist field applications of neonicotinoids.[26,27] In addition, a common oxidative detoxifying resistance mechanism due to the overexpression of monooxygenases against insecticides of this class has recently been demonstrated in *B. tabaci*.[28] Generally, neonicotinoids have been the fastest growing class of insecticides. They exhibit excellent contact and systemic activity and therefore have become widely used for sustained management of *B. tabaci*.[27] In Israel, after 2 years of use in cotton, no apparent resistance to imidacloprid and acetamiprid was reported when used in resistance management strategies.[29] However, as reported by Nauen and Denholm,[27] the ongoing introduction of these new molecules, unless carefully regulated and coordinated, seems bound to increase exposure to neonicotinoids and to enhance conditions favouring resistant phenotypes. Thus, the spread of whiteflies resistant to neonicotinoids, already observed in Egypt,[30] may be expected in the rest of West Africa in the near future, particularly in growing areas where pesticides are intensively used.[31]

The presence of multiple biotypes could explain the high level of insecticide resistance and the multiresistance in *B. tabaci* populations from Burkina Faso compared with those collected in Benin and Togo. The presence of whiteflies from the Q1-biotype living in sympatry with the local biotype, sub-Saharan Africa silverleafing (A-SL), has been recently observed in Burkina Faso on cotton and vegetables.[32] The Q1-biotype was expected to be dominant on cotton in Burkina Faso, but it was not observed in populations collected from Benin and Togo, where A-SL was the only biotype already observed in southern West Africa by Brown and Idris[33] and De la Rua *et al.*[34] The Q1-biotype, originating from the Mediterranean region,[35] is generally considered to be an invasive biotype like the B-biotype. It was originally considered to be restricted to the Iberian Peninsula, but has recently been established not only in other Mediterranean countries[1,3,36,37] but also in China[38] and the United States.[39]

The resistance and cross-resistance of *B. tabaci* to neonicotinoids shown in the present study might support the presence of Q-biotype in Burkina Faso populations. In such a case, the widespread use of acetamiprid in cotton and vegetables will probably select for the Q-biotype. Furthermore, some crops such as tomatoes could be threatened if Q-biotype were to acquire higher performance (its fecundity and longevity were to increase) from virus infection than the local biotype, as shown in China.[38] As a result, evolution of genetic diversity (particularly Q-biotype presence) associated with insecticide resistance of *B. tabaci* populations should be monitored in Burkina Faso and neighbouring countries where neonicotinoids are increasingly used in place of OPs. The general rule is that, the fewer applications of materials with a similar mode of action, the smaller is the potential for the development of resistance. The use of genetically modified *Bt* cotton could be a way to avoid the selection of *B. tabaci*-resistant populations by reduction in insecticide use and their outbreak by the preservation of natural enemies.[40] In this case, threshold sprays using selective insecticides with different modes of action could replace the calendar-based programme.

ACKNOWLEDGEMENTS

This work is part of the PhD thesis of Thomas Houndété, funded by the Service de Coopération et d'Action Culturelle (SCAC) de France au Bénin. The authors are grateful to Dr Roch Dabire (IRSS, Bobo-Dioulasso) and Blaise Zagre (INERA, Bobo-Dioulasso), both from Burkina Faso, for technical assistance. They are grateful to Seth Irish for corrections and comments. They also thank all insecticide suppliers.

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