# Variation in Resistance to Pyrethroids in *Helicoverpa armigera* From Benin Republic, West Africa

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**ABSTRACT** Pyrethroid resistance in *Helicoverpa armigera* (Hübner) field populations was investigated in Benin over several years by using third- and fourth-instar larval topical bioassays. *H. armigera* was resistant to pyrethroids tested as cypermethrin, deltamethrin, bifenthrin, and fenvalerate. Pretreatment with piperonyl butoxide significantly decreased the $LD_{50}$ value of cypermethrin and deltamethrin, and the resistance suppression by this synergist effect was observed. No significant decrease in the $LD_{50}$ value was obtained when S,S,S-tributyl phosphoro-trithioate was applied before deltamethrin. In the field, cypermethrin's $LD_{50}$ value varied, and the highest $LD_{50}$ values were observed during the rainy season, the cotton, *Gossypium hirsutum* L., crop period, when pyrethroids are extensively used. In the dry season when there was no cotton cultivation, the lowest $LD_{50}$ values were obtained. However, reversion was never total in the field; resistance did not revert to the level we observed in the susceptible strain. In the laboratory, when field populations were reared in insecticide-free conditions, resistance decreased and total reversion was observed. Results are discussed with regard to insecticide resistance fitness cost and resistance management strategies.

**KEY WORDS** *Helicoverpa armigera*, pyrethroids resistance, resistance stability, cotton, Benin Republic

The *Helicoverpa armigera* (Hübner) is one of the world's most destructive agricultural pests in general, and cotton, *Gossypium hirsutum* L., insect pests in particular. Development of resistant *H. armigera* populations has been reported in many countries and has caused huge economic losses and even catastrophes to cotton production. The first case of *H. armigera* resistance to pyrethroids was noted in Australia in 1982 (Gunning et al. 1984). Since then, resistance in this insect has been reported in several countries and was observed in West Africa at the end of the 1990s (Ahmad and McCaffery 1988, McCaffery et al. 1989, McCaffery and Walker 1991, Armes et al. 1992, Martin et al. 2000). In this area, several countries (Benin, Burkina Faso, Côte d'Ivoire, Mali, Senegal, and Togo) have pooled their resources to prevent and to manage resistance since 1998.

Various mechanisms of resistance have been developed by this insect, such as oxidative metabolism of insecticides, nerve insensitivity to pyrethroids, penetration resistance, and metabolism because of esterase (Ahmad et al. 1989; Gunning et al. 1991; Gunning 1996; Kranthi et al. 1997, 2001; Martin et al. 2002a; Yang et al. 2008). In West Africa, when resistance mechanism was analyzed in Côte d'Ivoire, only oxidative metabolism was detected (Martin et al. 2002a).

In the absence of insecticide treatments, insecticide resistance may be stable or unstable. The most likely cause of instability of insecticide resistance in the absence of insecticide treatments is a fitness cost associated with resistance (McKenzie and Clarke 1988; Raymond et al. 1993; Tabashnik 1994; Guillemaud et al. 1998, 1999; Wang et al. 1998; Foster et al. 1999, 2002; Miyo et al. 2000).

Early work suggested that insecticide resistance may be costly in the absence of chemical treatment (Caspari 1952, Crow 1957). Before the introduction of an insecticide, resistant insects were at a selective disadvantage as evidenced by the general rarity of resistant insects in populations. Upon the introduction of an insecticide, these resistant insects become selectively favored and rapidly spread throughout the population. If insecticide treatments favor resistance, it seems that most of mechanisms leading to resistance are disadvantaged in an environment free from all insecticide treatment. The cost is the negative effect that a resistance gene exerts on the selective value in the absence of insecticide. In natural population, when resistance is costly, it is therefore interesting to take it into account in resistant pests management.

Rotation of insecticides involving alternation of insecticide selection and no-selection periods is one of the resistance management strategies which can be used when fitness costs are associated with resistance,

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**Table 1. Strains of *H. armigera* collected in Benin**

| Strain | Locality of collection | | Date of collection (mo/yr) | Host plant |
|---|---|---|---|---|
| | Region | Village | | |
| ANG | North | Angaradebou | Sept. 1997 | Cotton |
| MAR | North | Mareborou | Aug. 1998 | Cotton |
| GOU | North | Gounin | Sept. 1998 | Cotton |
| SEK | South | Sékou | Nov. 1998 | Cotton |
| KOM | North | Komiguia | Sept. 1999 | Cotton |
| DRI | Central | Driji | Oct. 1999 | Cotton |
| AGB99 | Central | Agblakindji | Nov. 1999 | Cotton |
| OKP99 | North | Okpara | Nov. 1999 | Cotton |
| TAN | North | Tanéka-Koko | Feb. 2000 | Tomato |
| SAV00 | Central | Savalou | May 2000 | *C. viscosa* |
| THY | North | Thya | Oct. 2000 | Cotton |
| AGB01 | Central | Agblakindji | April 2001 | *C. viscosa* |
| OKP01 | North | Okpara | Sept. 2001 | Cotton |
| GOB | Central | Gobé | Nov. 2001 | Cotton |
| SAV01 | Central | Savalou | Nov. 2001 | Cotton |
| ANA | North | Anandana | March 2002 | Tomato |
| AGB02 | Central | Agblakindji | April 2002 | *C. viscosa* |
| OKP02 | North | Okpara | Oct. 2002 | Cotton |
| TIN02 | North | Tintinmou | Oct. 2002 | Coton |
| ANG02 | North | Angaradebou | Oct. 2002 | Coton |
| BOU02 | North | Bouhanrou | Oct. 2002 | Coton |
| AGB03 | Central | Agblakindji | May 2003 | *C. viscosa* |
| OKP03 | North | Okpara | Sept. 2003 | Cotton |
| PAO04 | Central | Paouignan | Sept. 2004 | Cotton |
| OKP04 | North | Okpara | Sept. 2004 | Cotton |
| THU04 | Central | Thui | Oct. 2004 | Cotton |
| DAN05 | South | Dan | Jan. 2005 | Tomato |
| ADJ | South | Adjaouèrè | June 2005 | Tomato |
| GOB05 | Central | Gobé | Sept. 2005 | Cotton |
| OKP05 | North | Okpara | Oct. 2005 | Cotton |
| ANG05 | North | Angaradebou | Oct. 2005 | Cotton |
| GOM06 | North | Gomparou | Oct. 2006 | Cotton |

whereas this strategy cannot be helpful when resistance is stable (Tabashnik 1994).

Different strategies were then tested to manage resistance (Djihinto 2004, Martin et al. 2005, Katary and Djihinto 2007a). Here, we report the status of *H. armigera* pyrethroid resistance in Benin Republic.

## Materials and Methods

**Strains.** A susceptible strain (BK77) was collected in 1977 on cotton in Bouake (Côte d'Ivoire) before the use of pyrethroid insecticides in field and was reared in CIRAD-Montpellier free from all insecticide treatment. The ANG strain was collected in 1997 from Angaradebou (in the North of Benin), one of the most important areas of cotton production in the country in which six pyrethroid treatments have been applied each year from the beginning of the 1980s. In 1997, first failures of treatment in field led to suspect the development of pyrethroid resistance. The ANG strain was reared in insecticide free conditions for four generations. To obtain a more homogeneous population, selection was performed at the fifth generation with deltamethrin at the $LD_{60}$ value (5 $\mu$g/g). The experiments described in this article used the sixth and seventh generations. Additional field strains of *H. armigera* were collected during several years on farms, from south to north of Benin. The locality where the strains have been collected and the collection dates are reported in Table 1. From August to November, strains were collected from pyrethroid treated cotton fields. From December to July, *H. armigera* was collected on *Cleome viscosa* L. and tomato (*Lycopersicon* spp.) plants that had received no or few treatment with pyrethroids. Strains collected in the field were reared in the laboratory for one to a maximum of eight generations in the absence of insecticide treatment.

Insects were reared at 25°C, 75% RH, and a photoperiod of 12:12 (L:D) h in the laboratory as described by Couilloud and Giret (1980). Larvae were reared on an artificial diet composed of maize flour (143 g/liter), brewers yeast (37.5 g/liter), wheat germ (37.5 g/liter), oil of maize (*Zea mays* L.) (1.5 ml/liter), sorbic acid (1.5 g/liter), ascorbic acid (12.5 g/liter), agar-agar (16,2 g/liter), and rifampicin (0.05 g/liter). Larvae were reared with the density of 150–200 per box (25 by 26 by 9 cm). At the end of their development, 100 larvae were placed for pupation in boxes with artificial diet and sand separated by a 2-mm polystyrene film. Pupae were collected, male and female were separated and kept in box until adults emerged. Five to 10 couples of adults were placed per box and fed on a 5% sugar solution for mating and oviposition. Their eggs were collected every morning on sterilized gauze placed on the top of the box until the adults' death. Eggs were washed with 1% bleach (sodium hypochlorite) and kept in the box (11 cm of diameter and 8 cm of height) for incubation. After 3–4 d, eggs hatched and neonates were placed on artificial medium cut in small peaces.

**Insecticides and Synergists.** Technical grade insecticides used in this study were as follows: cypermethrin (two batches at 93.2 and 96.6%), deltamethrin (95 and 99.6%), bifenthrin (95.05%), and fenvalerate (96.3%). For cypermethrin, the concentration of the *cis*-form was 48.4 and 50.7%. Cypermethrin and bifenthrin came from FMC Corporation (Philadelphia, PA). Deltamethrin and fenvalerate were kindly provided by Agrevo (Marseille, France) and Sumitomo (Tokyo, Japan), respectively. Cypermethrin and deltamethrin were chosen in this survey because they are the pyrethroids most widely used in cotton protection in Benin since the 1980s.

Two synergists were used in this study: piperonyl butoxide (PB) and S,S,S-tributyl phosphoro-trithioate (DEF). These synergists were applied topically in acetone at sublethal doses 1 h before the treatment of cypermethrin or deltamethrin. The sublethal doses were determined in preliminary bioassays using five doses of each synergist (1.25 mg/g for PB and 0.125 mg/g for DEF).

**Laboratory Bioassay.** The method used for susceptibility test was as advised by the American Entomological Society (Anonymous 1970, Martin et al. 2000, Djihinto 2004). Third- and fourth-instar larvae were weighed and sorted out in five weight classes (25–35, 35–45, 45–55, 55–65, and 65–75 mg). Insecticide solutions obtained by dilution in acetone were applied onto the thorax with an Arnold micro-applicator from Burkard Manufacturing (Rickmansworth, United Kingdom). Then, 0.2 $\mu$l was applied for 10 mg of larvae (0.6 $\mu$l on the smaller larvae and 1.4 $\mu$l on the bigger larvae); control larvae were treated with acetone.

**Table 2. Toxicity of pyrethroids to ANG, AGB99, and BK77 strains with and without the enzymes inhibitors PB and DEF**

| Insecticide | Strain | $LD_{50}$ (μg/g) | CI (95%) | Slope ± SE | Resistance factor |
|---|---|---|---|---|---|
| Bifenthrin | BK77 | 0.36 | 0.30–0.45 | 2.20 ± 0.28 | 34 |
| | ANG | 12 | 9.5–18 | 1.71 ± 0.26 | |
| Cypermethrin | BK77 | 0.44 | 0.38–0.58 | 2.98 ± 0.42 | 56 |
| | ANG | 26 | 22–30 | 3.04 ± −0.44 | |
| Fenvalerate | BK77 | 0.52 | 0.44–0.67 | 2.87 ± 0.56 | 177 |
| | ANG | 92 | 63–137 | 1.58 ± 0.36 | |
| Deltamethrin | BK77 | 0.08 | 0.05–0.11 | 1.81 ± 0.76 | 250 |
| | ANG | 21 | 7–32 | 1.99 ± 1.56 | |
| Deltamethrin + PB | BK77 | 0.14 | 0.11–0.19 | 2.41 ± 0.56 | 3 |
| | ANG | 0.37 | 0.11–1.2 | 1.36 ± 1.60 | |
| Cypermethrin | BK77 | 0.44 | 0.38–0.58 | 2.98 ± 0.42 | 86 |
| | AGB99 | 37.7 | 16.5–59.5 | 1.72 ± 0.36 | |
| Cypermethrin+ PB | BK77 | 0.44 | 0.30–0.58 | 2.16 ± 0.32 | 5 |
| | AGB99 | 2.4 | 1.04–5.8 | 1.16 ± 0.65 | |
| Deltamethrin + DEF | BK77 | 0.06 | 0.04–0.08 | 1.91 ± 0.65 | 320 |
| | ANG | 21 | 14–32 | 1.37 ± 0.82 | |

Doses were applied in order of increasing concentration, and the same syringe was used to apply all doses of the same insecticide. After the application of the insecticide, the larvae were placed in individual cells on an artificial media layer. Mortality was observed 48 h after treatment. A minimum of five doses with 30 insects per dose were applied. Statistical analyses of data were performed according to the log-probit method (Finney 1971) with WinLD software (CIRAD, Montpellier, France). Mortality in the control was always <10%. Data from all bioassays were corrected for control mortality using the formula of Abbott (1925). $LD_{50s}$ of two different strains were considered as significantly different when their 95% confidence intervals did not overlap. The resistance factor was determined as the ratio of the lethal dose for 50% ($LD_{50}$) of resistant strain to the $LD_{50}$ of the susceptible strain.

**Stability.** The stability of resistance can be observed in the absence of insecticides. The parameter R used to estimate response to selection is used to quantify the rate of change in $LD_{50}$ when selection is stopped (Tabashnik 1994):

$$R = [\log[\text{final } LD_{50}] - \log[\text{initial } LD_{50}]]/n$$

where n is the number of generations not exposed to insecticide, final $LD_{50}$ is the $LD_{50}$ after $n$ generations without selection, and initial $LD_{50}$ is the $LD_{50}$ before $n$ generations without selection. Negative values of R reflect decreases in $LD_{50}$; the inverse of R is the number of generations required for a 10-fold change in $LD_{50}$.

## Results

Table 2 presents the $LD_{50}$ values for BK77 and ANG strains for several pyrethroid insecticides. For each insecticide tested, the $LD_{50}$ values for BK77 and ANG strains were significantly different (confidence intervals do not overlap). However, resistance factors were different; deltamethrin and fenvalerate resistance factors were, respectively, 7 and 5 times more important than the bifenthrin resistance factor. Pretreatment with PB significantly decreased the $LD_{50}$ value of cypermethrin and deltamethrin. The ANG strain deltamethrin $LD_{50}$ value with PB pretreatment was not significantly different from that of the susceptible strain. No significant decrease in the $LD_{50}$ was observed when DEF was applied before deltamethrin.

Cypermethrin toxicity for field strains of *H. armigera* collected during several years is shown in Table 3. Pyrethroid treatment periods for cotton plant protection and the $LD_{50}$ value for each of these strains as a function of the collection date are reported in Fig. 1. From 1998 to 2000, the $LD_{50}$ from strains collected at different times during the cotton cropping season are significantly different (Table 3). Resistance factors increased during pyrethroid treatment periods (September–November) and decreased when pyrethroid treatments were suspended in cotton farms (December–August) as shown in Fig. 1. The decrease led to lower cypermethrin $LD_{50}$ values at the beginning of the pyrethroid selection period in 1999 (KOM strain in Table 3) than those observed during this period in 1998 (GOU strain in Table 3). The cypermethrin $LD_{50}$ values obtained for the TAN strain collected in February 2000 indicated that the $LD_{50}$ values could be very low, ≈4 μg/g, in an insecticide-free period, but they never reach the level of the BK77 susceptible strain. No significant difference in the $LD_{50}$ value was observed in 2001 and 2002 (Table 3). Afterward, sometimes significant difference was observed and the highest $LD_{50}$ values were obtained in pyrethroid treatment periods.

Seven *H. armigera* field strains have been reared in the absence of insecticide treatment over several generations (Table 4). For the KOM, OKP01, OKP02, and TIN02 strains, we have observed a significant decrease of the $LD_{50}$ values from the first generation through the following generations and resistance has completely disappeared after seven generations for the OKP01 strain. For seven strains of *H. armigera* (Table 4), the parameter R used to estimate response to selection is ranged from −0.03 to −0.28, with a mean value of −0.15, which indicates that, less than seven generations are required for a 10-fold decrease in $LD_{50}$. These results of laboratory tests indicate that *H. armigera* resistance to pyrethroids in Benin Republic is unstable and may have associated fitness costs.

**Table 3. Cypermethrin toxicity to *H. armigera* strains collected in fields and tested in generation 1 or 2 after their introduction in laboratory in function of their collection dates (1998–2006)**

| Strain | Date of collection M/Y | $LD_{50}$ (μg/g) | CI (95%) | $\chi^2$ | df | Slope ± SE | Resistance factor |
|---|---|---|---|---|---|---|---|
| BK77 | | 0.44 | 0.38–0.58 | 11.4 | 7 | 2.98 ± 0.42 | |
| MAR | 8/98 | 20.00 | 15–27 | 0.1 | 2 | 2.84 ± 0.72 | 45 |
| GOU | 9/98 | 33.00 | 27–39 | 5.0 | 3 | 3.00 ± 0.40 | 75 |
| SEK | 11/98 | 48.00 | 37–63 | 5.5 | 2 | 2.16 ± 0.36 | 109 |
| KOM | 9/99 | 14.20 | 9–20 | 6.0 | 5 | 1.41 ± 0.22 | 32 |
| DRI | 10/99 | 12.00 | 5–26 | 14.2 | 5 | 1.61 ± 0.31 | 27 |
| AGB99 | 11/99 | 38.00 | 27–52 | 8.5 | 4 | 1.72 ± 0.22 | 86 |
| OKP99 | 11/99 | 45.00 | 29–74 | 8.8 | 5 | 1.03 ± 0.14 | 102 |
| TAN | 2/00 | 4.00 | 2–6 | 1.2 | 5 | 1.14 ± 0.18 | 9 |
| SAV00 | 5/00 | 5.50 | 3.5–7.8 | 4.3 | 4 | 1.58 ± 0.23 | 13 |
| THY | 10/00 | 63.00 | 38–136 | 2.6 | 6 | 0.80 ± 0.13 | 143 |
| AGB01 | 04/01 | 15.00 | 9–27 | 7.4 | 4 | 0.96 ± 0.16 | 34 |
| OKP01 | 9/01 | 21.00 | 15–29 | 2.1 | 5 | 1.49 ± 0.17 | 48 |
| GOB | 11/01 | 29.00 | 20–44 | 2.8 | 7 | 1.08 ± 0.11 | 66 |
| SAV01 | 11/01 | 29.80 | 21–44 | 2.3 | 7 | 1.18 ± 0.12 | 68 |
| ANA | 3/02 | 32.00 | 18–64 | 1.9 | 5 | 0.76 ± 0.13 | 73 |
| AGB02 | 4/02 | 17.00 | 12–24 | 7.7 | 7 | 1.14 ± 0.13 | 39 |
| OKP02 | 10/02 | 26.00 | 17–42 | 4.3 | 6 | 1.18 ± 0.17 | 59 |
| ANG02 | 10/02 | 20.50 | 13–32 | 1.5 | 4 | 1.27 ± 0.21 | 47 |
| AGB03 | 5/03 | 40.2 | 29.9–54.7 | 7.6 | 8 | 1.64 ± 0.18 | 91.4 |
| OKP03 | 9/03 | 20.00 | 15–26 | 9.1 | 8 | 2.16 ± 0.27 | 45.5 |
| PAO04 | 9/04 | 3.80 | 2.5–5.4 | 5.1 | 7 | 1.15 ± 0.27 | 8.6 |
| OKP04 | 9/04 | 30.40 | 16–57 | 6.3 | 5 | 0.71 ± 0.48 | 69.1 |
| THU04 | 10/04 | 15.20 | 8–27 | 12.9 | 6 | 1.56 ± 0.37 | 34.5 |
| DAN05 | 1/05 | 12.4 | 10.8–14.3 | 6.7 | 8 | 2.91 ± 0.26 | 28.2 |
| ADJ | 6/05 | 25.4 | 18.1–36.5 | 10.7 | 8 | 1.21 ± 0.15 | 51 |
| GOB05 | 9/05 | 61.2 | 39.4–105 | 12.2 | 8 | 0.84 ± 0.15 | 125 |
| OKP05 | 10/05 | 50.3 | 36.1–72.8 | 4.0 | 7 | 1.31 ± 0.17 | 102 |
| ANG05 | 10/05 | 42.3 | 22.7–68.3 | 2.2 | 7 | 1.15 ± 0.20 | 87 |
| GOM06 | 10/06 | 30.9 | 21.9–44.9 | 9.1 | 8 | 1.02 ± 0.1 | 70.2 |

Toxicity to the susceptible strain, BK77, is used as a reference.

## Discussion

Field treatments with pyrethroids to manage *H. armigera* have induced resistance to most of the active ingredients of this insecticide family in several cotton producing countries, such as Australia (Forrester et al. 1993), Pakistan (Ahmad et al. 1995), India (Sekhar et al. 1995), China (Tan and McCaffery 1999), and Côte d'Ivoire (Martin et al. 2000). In Côte d'Ivoire, resistance extends to all the pyrethroids tested, including deltamethrin, cypermethrin, and fenvalerate; the results obtained in Benin were not in contradiction with these findings. The use of synergists showed that PB reduces the $LD_{50}$ value of cypermethrin and deltamethrin and suppresses the resistance for the ANG strain, whereas DEF has not a synergistic effect on this strain. This *H. armigera* response against cypermethrin and deltamethrin, in the presence of synergists, is consistent with results of survey about the effects of synergists (PB and DEF) on *H. armigera* strains resistant to pyrethroids collected in West Africa (Ehouman 1999; Martin et al. 2000, 2002a).

During the insecticide pressure period, the resistance factor increase in *H. armigera* natural populations suggests that resistant insects became dominant in the population. The same evolution was observed in the dynamic of *H. armigera* pyrethroid-resistant field

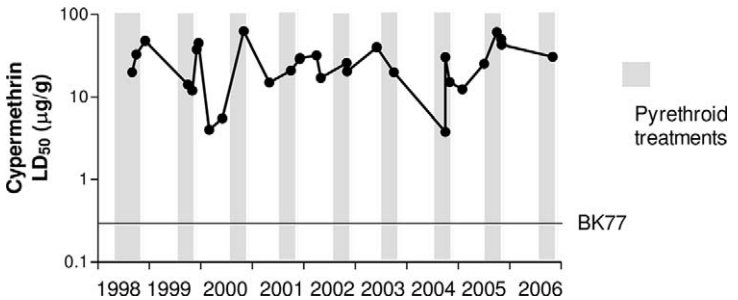


**Fig. 1.** Cypermethrin $LD_{50}$ for *H. armigera* strains (see Table 3) collected in fields and tested in generation one or two after their introduction in laboratory in function of the collection dates (1998–2006). In 1998, six pyrethroid treatments were performed on cotton from 1 August to 30 October. Insecticide resistance management is applied since 1999, and four pyrethroid treatments are realized each year on cotton from 30 August to 30 October.

**Table 4. Results of tests with topical application of cypermethrin in the laboratory without insecticide selection pressure over several generations**

| Strain/generation tested | $LD_{50}$ ($\mu$g/g) | CI (95%) | $\chi^2$ | df | Slope ± SE | Resistance factor |
|---|---|---|---|---|---|---|
| KOM/G1 | 14 | 9–20 | 6.0 | 5 | 1.41 ± 0.22 | 32 |
| G3 | 5 | 3–8 | 7.2 | 5 | 1.18 ± 0.16 | 11 |
| G4 | 2 | 1.5–3 | 6.5 | 4 | 1.52 ± 0.20 | 5 |
| AGB99/G1 | 44 | 30–70 | 7.0 | 5 | 1.35 ± 0.18 | 100 |
| G2 | 38 | 27–52 | 8.5 | 4 | 1.72 ± 0.22 | 86 |
| OKP99/G1 | 60 | 41–95 | 0.8 | 3 | 1.44 ± 0.24 | 136 |
| G2 | 45 | 29–74 | 8.8 | 5 | 1.03 ± 0.14 | 102 |
| OKP01/G2 | 21 | 15–29 | 2.1 | 5 | 1.49 ± 0.17 | 48 |
| G7 | 0.5 | 0.2–1 | 10.4 | 7 | 0.78 ± 0.11 | 1 |
| G8 | 1 | 0.6–2 | 3.7 | 4 | 0.94 ± 0.14 | 2 |
| GOB01/G2 | 29 | 20–44 | 2.8 | 7 | 1.08 ± 0.11 | 66 |
| G7 | 14 | 10–21 | 5.8 | 4 | 1.51 ± 0.22 | 32 |
| OKP02/G1 | 26 | 17–42 | 4.3 | 6 | 1.18 ± 0.17 | 59 |
| G3 | 5 | 3.5–7 | 6.1 | 5 | 1.38 ± 0.18 | 11 |
| TIN02/G1 | 72 | 46–138 | 1.0 | 6 | 1.14 ± 0.17 | 164 |
| G3 | 10.7 | 2.6–33 | 7.9 | 3 | 1.78 ± 0.43 | 24 |

Some generations were not tested because the number of larvae available was not sufficient to test. For example, KOM strain was not tested in $G_2$.

population in India and China (Madden et al. 1994, Han et al. 1999). In China, resistance has increased rapidly in an *H. armigera* field population when the population density became higher and insecticides were applied repeatedly. Lenormand et al. (1999) have shown a similar phenomenon in *Culex pipiens* L. populations in the South of France. Selection coefficients because of insecticide treatments were positive, leading to an increase of resistance alleles frequencies.

However, *H. armigera* resistance decreased when insecticide treatment was suspended in both cases, in the field between two treatment periods for cotton plant protection (from 1998 to 2000) and in the laboratory when a field strain was maintained for several generations in the absence of insecticide treatment. Sometimes no decrease of the resistance was observed in the field when insecticide treatment was suspended in cotton farms. In Benin, *H. armigera* resistance to pyrethroids was unstable between 1998 and 2006, except for 2001 and 2002, when cypermethrin resistance level did not fluctuate significantly in the field. The instability of pyrethroid resistance in *H. armigera* is similar to that described by Wu et al. (1996) and Han et al. (1999) in China: the level of pyrethroid resistance was unstable and then stabilized at ≈2 to 9 times the susceptible strain level. It is difficult to assess whether the resistance instability is general or infrequent in insecticide resistance, we can only note that it has already been reported for some other insects, such as *Musca domestica* L. (Georghiou 1964), *C. pipiens* (Lenormand et al. 1999, Rivet and Pasteur 1993), *Anopheles sacharovi* (Favre) (Hemingway et al. 1992), or *Myzus persicae* (Sulzer) (Foster et al. 2002).

There are two nonexclusive explanations to account for the decrease of resistance when there is no selection pressure. First, the resistance has a fitness cost and resistant insects could be counterselected in the lack of insecticide treatments. Second, resistant populations could be diluted by immigration of susceptible populations from nontreated areas (Madden et al., 1994, Han et al. 1999). Because resistance decreased in laboratory conditions, under the lack of treatment, we can hypothesize that a fitness cost contributes to the resistance instability in the field as it has been shown for Chinese resistant populations of *H. armigera* (Xiaoxia et al. 2001). Such association of resistance and fitness cost has been described in insects (Clarke and McKenzie 1987, McKenzie and Clarke 1988, Raymond et al. 1993, Guillemaud et al. 1999, Foster et al. 1999). Fluctuation in resistance during the year can then be explained as the result of local adaptation to a finite environment where insecticide selection pressure and resistance fitness costs coexist and act as antagonistic forces (Guillemaud et al. 1998, Miyo et al. 2000). The nonfluctuation of the resistance level in the field during 2001 and 2002 yr and the instability observed in the laboratory at the same time can be explained by the difference between the laboratory and the field conditions. In the laboratory, insects are reared without insecticide treatment, whereas in the field four pyrethroid treatments are used each year for cotton plants protection. In natural population, another host plants of this insect as tomato plants also are treated with pyrethroids. If resistance is costly, the fitness cost that decreases the resistance level when insecticide treatments are stopped and the insecticide selection pressure that increases the resistance level in insecticide treatment periods have reached an equilibrium in 2001 and 2002 yr in field and no significant difference is observed in resistance level fluctuation. The measure of the fitness parameter will bring information toward a better understanding of resistance fitness cost in *H. armigera* from Benin (unpublished data).

Based on antagonistic selective pressures principle that insecticide selection favors resistant individuals and resistance fitness cost disadvantages them in the absence of insecticides and in accordance with Georghiou et al. (1983), Mallet (1989), Bonning and Hemingway (1991), Hemingway et al. (1992), Rodriguez et al. (1993), Tabashnik (1994), Guillemaud et al.

(1998), Mason (1998), and Lenormand and Raymond (1998), several strategies can be used in resistance management: 1) Temporal (window strategy) or spatial (mosaic strategy) rotation involves alternation of insecticide selection and no-selection periods. Nevertheless, insecticide rotation efficiency can encounter the problem of modifier gene selection that reduces resistance fitness costs (Mason 1998). 2) Insecticide mixtures can be used (Lenormand and Raymond 1998). A precise understanding of the mechanisms involved in insecticide resistance will be useful for the design of successful management strategies. 3) Dilution (mating) with susceptible populations can be used. Resistance reduction can be increased when resistant populations mate with other populations from untreated or less treated areas. Thus, to calculate the critical size of the treated area (stable zone), it is important to take into account insect migration and the dynamic of the *H. armigera* populations between the different parts of the crop system (Lenormand and Raymond 1998, Lenormand et al. 1999).

The treatment strategy used in Benin for the management of *H. armigera* resistance to pyrethroid (Djihinto 2004, Martin et al. 2005, Katary and Djihinto 2007a) as well as in the other West Africa countries involves the use of endosulfan at the beginning of the season because there is no cross-resistance with this insecticide (Martin et al. 2002b). The last treatments use mixed formulations containing pyrethroids and organophosphates. Organophosphate insecticides synergize pyrethroids because they compete with oxydases that metabolize pyrethroids (Martin et al. 2003). This strategy seems to give satisfaction, as there is not any longer problem with *H. armigera* in this area (Djihinto 2004; Martin et al. 2005; Katary and Djihinto 2007a, 2007b) but stay a short-term strategy because of relative toxicity of endosulfan and use of pyrethroids for the last treatments did not allow to revert the resistance.

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