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INSECTICIDE RESISTANCE FITNESS COST AND RESISTANCE STABILITY

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ABSTRACT: *This review focuses on insecticide resistance fitness cost and its consequences on resistance stability. Many cases of insecticide resistance are associated with fitness cost even though sometimes, resistance is not costly. The cost may be evidenced by different methods. Thus decrease of insecticide resistance in heterogeneous populations in the absence of any insecticide treatment supposes a hypothesis that fitness cost is associated with resistance. This hypothesis can be tested by comparing biological, morphological and ecological parameters of susceptible and resistant strains. Quantitative and qualitative enzyme overproduction and mutation are major resistance fitness cost mechanisms. The cost may be explained by the energy invested to synthesize protein for enzyme overproduction. At the beginning of the resistance, the cost appears to be unstable, but with time it stabilizes. Sometimes modifier gene is selected during resistance appearance process and resistance fitness cost is deleted. When resistance is associated with fitness cost the latter can be used in insecticide rotation strategy for resistance management.*

Keywords: *Insecticide, resistance fitness cost, resistance stability, enzyme overproduction*

INTRODUCTION

Since the 1940's, the entire planet has been spread with insecticides for agricultural pest management and a lot of insects have developed resistance. As environment is contaminated with toxic molecules and treatment is not continuous, insects have to adapt, to new environments, to be competitive in alternating periods with and without treatments. Thus, insecticide resistance offers the opportunity to study the adaptation of insects to variable environments.

Several mechanisms of resistance to insecticides are developed by insects. Among them, increased degradation of insecticide (metabolisation by esterase, oxydase or transferase enzymes), insecticide target modification, reduction of insecticide penetration, and behavioral resistance have been listed (Magnin *et al.,* 1985; Ahmad *et al.,* 1989; Gunning *et al.,* 1991; Gunning, 1996; Kranthi *et al.,* 1997; 2001; Martin *et al.,* 2002; Yang *et al.,* 2008) as four main mechanisms of resistance to insecticides.

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; Wang *et al.,* 1998; Guillemaud *et al.,* 1999; Foster *et al.,* 1999; Miyo *et al.,* 2000; Foster *et al.,* 2002). It is not surprising that genes responsible for insecticide resistance, for an adaptation to a new environment, are usually costly in the absence of chemical treatment (Berticat *et al.,* 2008; Djihinto, 2004; Crow, 1957; Caspari, 1952). 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 appears 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.

An explosion of interest in adaptive response of insects has spurred publication of numerous articles about insecticide resistance fitness cost (Djihinto *et al.,* 2012; Martins *et al.,* 2012; Fang *et al.,* 2011; Paris *et al.,* 2011; Roy *et al.,* 2010; Djogbénou *et al.,* 2010; Gassmann *et al.,* 2009; Ellison, 2007; Bourguet *et al.,* 2004; Xiaoxia *et al.,* 2001; Gazave *et al.,* 2001; Haubruge and Arnaud, 2001; Oppert et al., 2000; Wang *et al.,* 1998; Ferrari and Georghiou, 1981) and resistance

stability (Yang *et al.,* 2013; Djihinto *et al.,* 2009; Djihinto, 2004; Kristensen *et al.,* 2000; Han *et al.,* 1999; Wu *et al.,* 1996; Tabashnik, 1994; Raymond *et al.,* 1993; Fournier *et al.,* 1988; Georghiou, 1964; Varzandeh *et al.,* 1954) but relatively few reviews have focused on it (Gassmann *et al.,* 2009; Crow, 1957).

This review therefore emphasizes insecticide resistance fitness cost and resistance stability. Evidence of cost and cost explanation in relation with mechanisms involved are firstly approached. Then cost evolution and it explanation in relation with mechanisms responsible are secondly presented. At the end, cost may be used as a tool for resistance management.

EVIDENCE OF COST

Insecticide resistance fitness cost can be evidenced by two mains methods. The first method is the stability of resistance in absence of insecticide and the second one is the study of biological parameters of resistant insects in comparison with susceptible individuals.

Resistance stability: The instability of the insecticide resistance can be observed only if the population of insect is heterogeneous and contains some susceptible individuals with the resistant. Indeed, if resistance of a population is stable without insecticide treatment, it means either that the population is homogeneous (and contains only resistant individuals) or that the fitness cost is not associated with resistance (Fournier *et al.,* 1988; Raymond *et al.,* 1993). Stability of insect resistance to insecticide may be observed in field and in laboratory rearing conditions.

Stability in laboratory: Stability of resistance monitoring in the absence of any insecticide treatment in laboratory is one of the ways to point out resistance fitness cost. When the resistance is costly, several studies have evidenced that resistance levels in absence of insecticide in laboratory conditions are not stable and then decrease (Varzandeh *et al.,* 1954; Georghiou, 1964; Raymond *et al.,* 1993; Tabashnik, 1994; Wu *et al.,* 1996; Djihinto, 2004; Djihinto *et al.,* 2009). There are two nonexclusive situations which can be observed when the resistance is unstable and resistance levels decrease in laboratory conditions. First, regression to susceptibility was not observed when selection for resistance is relaxed (Crow, 1954). In that moment, reversion to susceptibility can not be total in absence of treatment; it seemed very difficult to recover susceptibility completely (Wu *et al.,* 1996). Second, total reversion was observed in the laboratory, when field populations were reared in insecticide-free conditions, resistance decreased and can completely disappear (Djihinto, 2004; Djihinto *et al.,* 2009).

One limitation of the study of resistance stability in heterogeneous strain, obtained by mixing resistant and susceptible individuals, is that susceptible strains are often laboratory strains well adapted to laboratory conditions and thus may have an advantage to resistant strains which have been usually recently introduced in the laboratory, and then have less adapted. Thus a decrease of resistance in laboratory after mixing to a susceptible strain may reflect more the selection of the laboratory strain than the fitness cost of the resistant strain.

Stability in field: Resistant individuals are favored during treatment moments and their frequency can increase independently of resistance fitness cost. When resistance is associated with fitness cost, resistant individuals are disadvantaged during treatment cessation periods, their frequency can decrease and accordingly resistance levels of the population decrease. Some of cases of stability of insecticide resistance in absence of insecticide in field population have been studied and reported here.

Regression of resistance during the non-treated period has been often observed and the proportion of insecticide resistant individuals of the cotton bollworm *Helicoverpa armigera* (Hübner) decreased gradually when the use of insecticides was suspended in China (Yang *et al.,* 2013; Han *et al.,* 1999), but increased quickly again when repeated sprays of insecticides were applied late. In *Culex pipiens* L. resistance in France, alleles responsible for resistance were unstable and decreased in winter (Lenormand *et al.,* 1999). The instability of pyrethroid resistance in *H. armigera* from Benin republic (Djihinto *et al.,* 2009) is similar to that described by Wu *et al.* (1996) and Han *et al.* (1999) in China: the level of resistance increased in insecticide pressure period and decreased when insecticide treatment was suspended. However, reversion was never total in the field; resistance did not revert to the level observed in the susceptible strain. Decrease of resistance was also

reported when an insecticide was no longer used. A break of azamethiphos use to control housefly *Musca domestica* L. in Danmark from 1987 to 1995 led to a decline in resistance. In 1995 azamethiphos toxicity was equivalent to that seen before the introduction of this insecticide (Kristensen *et al.,* 2000).

However, instability of resistance in the field may also originate from migration of susceptible insects from less treated crops or from non cultivated plants. It can also originate from the application of new insecticides with anti resistance properties which are more efficient against the resistant insect than the susceptible one. Application of these insecticides would select susceptible insects.

EFFECT OF RESISTANCE ON BIOLOGICAL, MORPHOLOGICAL AND ECOLOGICAL PARAMETERS

A lot of parameters were compared in resistant and susceptible strains to evidence the cost of insecticide resistance. Here, such parameters are grouped as biological, morphological and ecological parameters. Biological parameters underlie the life of insect whereas morphological parameters are interested in insect organ shape. Ecological parameters describe insect population.

BIOLOGICAL PARAMETERS

Principal biological parameters compared of resistant and susceptible strains observed were fecundity, fertility, time of development and survival through each stage of insect and insect longevity. Several cases of resistance were associated with modification of these parameters and disadvantaged resistant individuals.

Fecundity: A lot of cases of resistance were correlated with decrease production of eggs, and then, the resistance was costly (Djihinto *et al.,* 2012; Martins *et al.,* 2012; Fang *et al.,* 2011; Paris *et al.,* 2011; Roy *et al.,* 2010; Xiaoxia *et al.,* 2001; Wang *et al.,* 1998; Ferrari and Georghiou, 1981). Djihinto *et al.* (2012) and Xiaoxia *et al.* (2001) have evaluated, in Benin and in China respectively, the effects of insecticide resistance on fitness of cotton bollworm, *H. armigera* in terms of fecundity or number of eggs laid per female of resistant and susceptible strains. Result revealed that fecundity of resistant strain was lower than susceptible. In Taiwan, the same phenomenon has been obtained in another insect when Fang *et al.* (2011) have studied the effects of insecticide resistance on fecundity of the oriental fruit fly, *Bactrocera dorsalis* (Hendel), and result showed that resistant strain had the lowest fecundity. Other cases of decrease of fecundity have been observed in several resistant insects including pyrethroid and organophosphate resistant strain of the dengue vector *Aedes aegypti* (L.) from South America (Martins et al., 2012), bacterio-insecticide *Bacillus thuringiensis* subsp *israelensis* (*Bti*) resistant strain of *A. aegypti* (Paris *et al.,* 2011), dipterex, temephos and chlorpyrifos resistant strains of *Culex pipiens pallens* (L.) (Wang *et al.,* 1998), temephos resistant strain of *Culex quinquefasciatus* Say (Ferrari and Georghiou, 1981) and chlorpyrifos resistant strain of *C. quinquefasciatus* from Tanzania (Amin and White, 1984).

Fertility: Like fecundity, insecticide resistance has induced the decrease of fertility or percentage of eggs hatch in several resistant insects. In Benin and in China, the resistant strain compared with the susceptible strain of *H. armigera* revealed that resistant strain was less fertile (Djihinto *et al.,* 2012; Xiaoxia *et al.,* 2001). The study in Benin concluded that one of the main costs found for *H. armigera* pyrethroid resistant strain involved low fertility. Insecticide resistance had very significantly reduced also fertility of deltamethrin resistant strain of *A. aegypti* from Brazil (Martins *et al.,* 2012), spinosad resistant strain of *Plutella xylostella* (L.) from Hawaii (Ellison, 2007), temephos resistant strain of *C. quinquefasciatus* (El-khatib and Georghiou, 1985), chlorpyrifos resistant strain of *C. quinquefasciatus* from Tanzania (Amin and White, 1984) compared to a susceptible strain.

Time of development: About time of development through each stage, resistance increases development time in several insects (Djihinto *et al.,* 2012; Roy *et al.,* 2010; Gassmann *et al.,* 2009). In natural populations, shorter development times of insect reduced risk of insect mortality due to predation or their environmental conditions, whereas longer development times of resistant individual disadvantaged it. Measurement of larval development times indicated that a fitness cost was associated with resistance to *Bacillus thuringiensis* Berliner in *Bt*-resistant colonies of *Plodia*

interpunctella (Hübner). In many cases, the development of *Bt*-resistant moths on *Bt*-treated diet was slower than the unselected moths on untreated diet (Oppert *et al.,* 2000). Resistance to pyrethroids of *H. armigera* (Djihinto *et al.,* 2012), to organophosphate of *C. pipiens pallens* (Bourguet *et al.,* 2004), to monocrotophos of *H. armigera* (Xiaoxia *et al.,* 2001), and to malathion of *Tribolium castaneum* (Herbst) (Haubruge and Arnaud, 2001) involved also significant slower development of resistant strains than susceptible. Deltamethrin and diflubenzuron resistance in another insect, the codling moth, *Cydia pomonella* (L.), indicated that resistant individuals developed more slowly than susceptible individuals (Boivin *et al.,* 2001).

Longevity and survival through each stage of development: Kence and Kence (1993) have measured biotic parameters of malathion resistant and susceptible strains of house fly *M. domestica*. Percentages of survival, longevity were compared. To measure percentages of survival from egg to adult, eggs were collected from cages and kept at conditions for the rearing room. When the adults began emerging, the number of emergence was recorded every 12 h until emergence of flies ceased. Percentage of survival was compared by one-way analysis of variance. To estimate longevity, 10 single pairs of flies of resistant and susceptible strains were placed in small plastic cups separately and were fed with milk; dead flies were recorded every day. Longevities were compared by the Kruskal-Wallis test. Survival from egg to adult was significantly different. Susceptible strain had the lowest survival. Resistant male of this insect lived longer than susceptible male.

In *H. armigera* resistance to pyrethroids in Bénin Republic, survival from egg to adult emergence of resistant strain was significantly lower than survival from egg to adult emergence of susceptible strain and this result, represented one of the substantial costs for resistant strain as it directly decreased the proportion of individuals able to contribute to the next generation (Djihinto *et al.,* 2012). In another insect, recently, Djogbénou *et al.* (2010) found that resistance decreased pupae survival of *Anopheles gambiae* (Meigen) and this result constituted the main cost of the resistance. The same phenomenon of decrease of survival from egg to adult emergence of resistant strain has been observed by Gassmann *et al.* (2009) and Gazave *et al.* (2001).

No difference between parameters observed: resistance advantages: Sometimes insecticide resistance has no known negative effect in insects and no difference can be obtained between resistant and susceptible strains. This case should be very interesting when resistant individuals are used as predators for pest management.

For exemple, Fournier *et al.* (1988) have compared fitness in methidathion resistant and susceptible strains of the predatory mite *Phytoseiulus persimilis* (Athias-Henriot). Daily mortality, starvation susceptibility and egg-laying were followed in both strains. Preimaginal mortality, longevity of adult females without food and the numbers of eggs laid by each female were not significantly different in resistant and susceptible strains. In another experiment, the measured values of biological parameters such as mean number eggs laid and longevity without food were similar for methidathion resistant and susceptible strains of *P. persimilis* (Schulten and Van de Klashorst, 1974).

Likewise, the lack of fitness costs of insecticide resistance in the western flower thrips *Frankliniella occidentalis* (Pergande) (Thysanoptera: Thripidae) has been observed and may accelerate the development of insecticide resistance in populations of this insect (Bielza *et al.,* 2008). Resistance can also be favorable for resistant individuals. Thus, fecundity has been increased in malathion-specific resistant beetles in absence of insecticide pressure (Arnaud *et al.,* 2002) and the same phenomenon has been observed by Bielza *et al.* (2008) in resistant females of *F. occidentalis*.

ECOLOGICAL PARAMETERS

Ecological parameters usually observed were intrinsic rate of increase, net reproduction rate and mean generation time. Those parameters allowed to describe population evolution. Resistance can affect ecological parameters and can disadvantage insecticide resistant population. There are four examples studied here to explain ecological parameters. Ferrari and Georghiou (1981) have investigated the effects of the organophosphate insecticide temephos on the intrinsic rate of increase of the resistant and susceptible strains of *C. quinquefasciatus*. The resistant strain had lower intrinsic rate of increase. The same parameter was observed for the same insect but in

another resistant strain to chlorpyrifos by Amin and White (1984). Resistant strain had also lower intrinsic rate of increase.

However, the parameters such as intrinsic rate of increase, net reproduction rate and mean generation time were similar for two different strains of predator *P. persimilis* resistant to parathion (Schulten and Van de Klashorst, 1974), methidathion (Fournier et al., 1988), when they were compared to susceptible strain. It means that resistance can affect population without any modified parameters in certain cases.

ASYMMETRY (MORPHOLOGICAL PARAMETERS)

Morphological parameters may also be used to measure fitness cost. Fluctuating asymmetry, random differences between left and right sides of a normally bilaterally symmetric organism, is used to indicate genetic stresses on development. Estimating the absolute difference in bristle count between the left and right sides of the frontal head stripe, the outer wing margin and the R_{4+5} wing vein, Clarke and McKenzie (1987) showed that diazinon resistant phenotypes of the Australian sheep blowfly, *Lucilia cuprina* Wiedemann showed greater fluctuating asymmetry than susceptible phenotypes. This effect was found to be dominant for dieldrin and diazinon and partially dominant for malathion (Mckenzie and Clarke, 1988; Freebairn *et al.,* 1996; Clarke, 1997). Asymmetry reflects physiological cost and has been intimately linked to the fitness (McKenzie and Batterham, 1994).
A similar result was obtained by Bourguet (1996) in the case of *C. pipiens* resistance due to Ace^R allele which increases asymmetry fluctuation to 41 %. As fluctuating asymmetry, wing length may be considered as indirect measure of selective value. In *C. pipiens* resistance, reduction of wing length can be observed about 2 % by Bourguet (1996).

RELIABILITY OF COST ESTIMATION

Difference between biological parameters of resistant strain compared to susceptible depends on parameters observed, sex and susceptible strain used.

Difference due to parameters observed: In comparing several biological parameters of resistant and susceptible strains, differences may be observed with one parameter whereas no difference is obtained with the other. Thus difference obtained depends on parameter observed and then resistance can affect one parameter without the other. Here four examples are mentioned.

Amin and White (1984) studied the effects of chlorpyrifos resistant on fitness in *C. quinquefasciatus* by comparison of resistant and susceptible strains. Results showed that no difference was observed between time of egg laying to hatching, percentage of egg hatching, percentage of larvae pupating, percentage of survival through all aquatic stages, and sex ratio of resistant and susceptible strains. However significant difference was observed between time of egg laying to first pupation, first pupation to first adult emergence, and mean number of eggs laid by females.

In Red Flour Beetle, *T. castaneum* resistant to malathion, Haubruge and Arnaud (2001) studied consequences of resistance on fitness. They found that egg fertility did not differ whereas fecundity of susceptible females was significantly lower than that of the females of the resistant strains. The fitness cost associated with resistance to transgenic cotton has been investigated in the Pink Bollworm *Pectinophora gossypiella* (Saunders) by Carrière *et al.* (2001). Resistant strains were compared with susceptible. Results indicated that resistance reduced survival on non-Bt cotton by an average of 51.5 % in resistant strain relative to the susceptible. But development time on non-Bt cotton did not differ between resistant and susceptible strains.

Sometimes difference between biological parameters is explained by variation of humidity. Indeed, Gasser (1951) found that a resistant strain of *Tetranychus urticae* Koch developed quicker at 90% relative humidity but slower at low relative humidity (30-60 %) and at temperature of 16-22 °C in comparison with susceptible. Thus, in certain conditions, resistant strain had sometimes the highest fitness and sometimes the lowest and then difficult to conclude.

Difference due to sex: In comparison of biological parameters of resistant strain to susceptible, difference may be observed with one sex (male resistant and male susceptible or female resistant and female susceptible) whereas no difference is obtained with the other. Thus difference obtained depends on sex observed. For example Kence and Kence (1993) found that resistant male of *M. domestica* lived longer than susceptible male whereas no difference was observed between resistant and susceptible females.

THE SUSCEPTIBLE STRAIN USED

Resistant strains originating from field and newly introduced in the laboratory are often compared to laboratory strains established for a long time and well adapted to laboratory conditions. The apparent lower fitness of resistant strain can reflect more the lower adaptation of the resistant strain (a field strain) to laboratory conditions than the effect of resistance. Indeed, genetic changes during insect domestication or rearing in laboratory have been mentioned by Bartlett (1984; 1985). In the process of establishing a population in the laboratory, we generally take a sample of insects from the much larger feral population. The sampling process will tend to change the distribution of gene frequencies; since common alleles from the original population will be represented in the sample, while rare alleles will often be lost since they have a low probability of being included in the sample. The size of the colonizing sample will have a direct effect on the amount of genetic variation in laboratory (the larger the original sample, the smaller the deviations of the sample from the original gene frequencies; the smaller the sample, the greater the observed deviations). Laboratory conditions will also cause other changes not directly related to the environment.

Thus, some behavioral characters are observed to be expressed differently depending on the density of the population in laboratory conditions. Mate searching behavior is restricted in the small mating cages usually provided in the laboratory. Female egg-laying behavior probably changes when few deposition sites are provided. Dispersal characteristics, specifically adult flight response and larval dispersal, may be severely restricted by laboratory rearing conditions. Since environmental conditions in the laboratory are different from those encountered by the field population, certain individuals, which may or may not have been favored in the natural conditions, may now become more fit; that is, able to produce more progeny. Thus natural selection in laboratory increased the frequencies of certain genotypes and decreased the frequency of others. During insect rearing in laboratory, genetic variability decreased and the lost of variability is due to drift, selection and inbreeding (Bartlett, 1984). With time genetic variability stabilized and laboratory strains are stable adapted to laboratory conditions. Then, difference observed between laboratory susceptible strain compared to resistant field strain in laboratory conditions must not necessary due to resistance. Strain adaptation problem to laboratory conditions must be then taken into account.

COST AND RESISTANCE MECHANISMS

Several mechanisms underlie resistance to insecticides in arthropods. First, there is a decrease of insecticide penetration through the cuticle (Ku and Bishop, 1967). Second, resistance can originate from an increase of detoxification by esterases, oxidases or glutathione S-trransferases (Kranthi *et al.,* 1997, 2001; Martin *et al.,* 2002). This increased degradation of the pesticide originates either from gene amplification (Mouches *et al.,* 1986; Field *et al.,* 1988), overtranscription (Fournier *et al.,* 1992a) or point mutation in the enzyme (Newcomb *et al.,* 1997). Thirdly, resistance can be due to target modification. There are three main target sites for most insecticides: -amino-butyric acid receptor is the target of cyclodiene insecticides, the voltage-dependent sodium channel is the target site for DDT and pyrethroids while acetylcholinesterase modification induce resistance to organophosphorus and carbamate. A decrease in target sensitivity due to point mutations has been described for those three targets (Fournier *et al.,* 1992b; ffrench-Constant *et al.,* 1993; Williamson *et al.,* 1996).

Stability of quantitative esterase overproduction via gene amplification has been extensively studied in two species, *C. pipiens* and *Myzus persicae* (Sulzer). The cost may be explained by the energy invested to synthesize protein for esterase overproduction or for gene amplification (Devonshire and Field, 1991; Guillemaud *et al.,* 1998). The cost is then associated with the amount of overproduced enzyme or the number of gene copies.

In *C. pipiens*, the highest amplification level of esterase genes in resistant individual was between 40 to 500 fold (Mouchès *et al.,* 1986; Raymond *et al.,* 1989; Poirié *et al.,* 1992; Guillemaud *et al.,* 1997). The amount of overproduced enzyme differs according to the amplidfied esterase, B4 or A1 accordingly, different fitness costs are associated with these resistance alleles which code for overproduction of each esterase. The cost must then be smaller for B4 overproduced esterases than for A1 in the nontreated area (Guillemaud *et al.,* 1998). Moreover, the amplification level (the gene copy number) in *C. pipiens* resistant strain for overproduced esterase A4-B4 was measured twice on the same strain maintained in absence of selection. It was 25-fold in 1991 (Poirié *et al.,* 1992) and five-to eightfold in 1996. In their review of the relationships between molecular mechanism of resistance and fitness components, Taylor and Feyereisen (1996) noted that moderate gene amplification should be associated with low fitness cost. Likewise Raymond *et al.* (1993) found that mortality was significantly larger in *C. pipiens* with esterase B1 gene amplification which induces OP-resistance than in insects lacking this enzyme.

Amplification of esterase in the peach-potato aphid, *M. persicae* is associated to fitness cost. Resistance decreased during the non-treated season (Foster *et al.,* 2002). Aphids with higher levels of carboxyesterase were counter-selected at low temperature and with increasing rainfall (Foster *et al.,* 1996). Higher carboxyesterase levels was closely associated with maladaptative behavior in the form of lower tendencies to move detect and move away from senescing leaves and in reduced response to alarm pheromone leading to greater vulnerability to parasitoid and predator attacks (Foster *et al.,* 1996; 1997; 1999).

Mutations providing the knock-down (kdr) resistance to DDT and pyrethroids are stable or not stable depending on the insect. In *H. armigera*, it does not seem to be stable since after severe restrictions were placed on the use of pyrethroids in Australia, nerve insensitivy rapidly declined to virtually undetectable levels in field populations (Gunning *et al.,* 1995). By contrast, in *M. persicae* kdr frequency was found high and stable in UK from 1997 to 2000, suggesting that the mutation was not affected with a high cost. However, aphids with kdr resistance to pyrethroids show a greater tendency to remain on deteriorating leaves and show much lower levels of disturbance after exposure to measured amounts of synthetic alarm pheromone than aphids without kdr increasing their vulnerability to natural enemies by predation and parasitism (Foster *et al.,* 1999).

Acetylcholinesterase resistance was found to be unstable in *M. persicae* in UK (Foster *et al.,* 2002). However this instability most probably results from a closed association with the amplified esterase (Foster, 2002). A fitness cost associated with resistance to cyclodiene insecticides has been detected in several species. In the blowfly *L. cuprina*, a fitness cost associated with the *Rdl* locus has been demonstrated (McKenzie, 1990; McKenzie and Yen, 1995). Over-wintering populations of *L. cuprina*, with *rdl/rdl* and *rdl/sdl* flies displaid an increased rate of mortality. Decrease of fitness was reported for other mutations. For example, in *L. cuprina*, alleles of two unlinked genes (diazinon, *Rop-1*) determined resistance to diazinon. Resistant individual showed greater fluctuating asymmetry than susceptible (Clarke and Mckenzie, 1987; Clarke, 1997).

EVOLUTION OF COST

The cost of the resistance may appear high at the beginning of the insecticide selection. Thus the resistance is unstable at the begenning of his appearance. With time the cost disappears and resistance stabilizes suggesting that there is a co-adaptation of resistance and fitness cost. To illustrate this evolution of cost, selection for monocrotophos resistance in pear psylla, *Cacopsylla pyri* (L.) was examined by Berrada *et al.* (1995). Adults of *C. pyri* were collected in 1989 in commercial pear orchards. This colony was reared in the laboratory and was selected for resistance to monocrotophos over a 4-year period for a total of 40 generations. The selection was discontinuous; treatments were not applied at all generation. It appeared that, at the beginning, the resistance was not stable, in absence of treatment the resitance dropped. By contrast after the 30^{th} generation with 21 selected, the resistance was stable and did not decrease in absence of selection. Clarke and Mckenzie (1987) showed also that insecticide resistant Australian sheep blowfly, *L. cuprina*, showed greater fluctuating asymmetry than susceptible phenotypes when resistance first evolved. Continued use of insecticide after resistance developed lead to a return in the asymmetry of resistant phenotypes to the level of susceptible.

When the cost originates from linkage disequilibrium between the gene responsible for resistance and other costly genes, recombination will progressively eliminate the costly gene. However, this may be impossible for species with asexual reproduction (Foster *et al.,* 2002).

Mechanism of evolution of cost has been studied in *C. pipiens*; a mutation decreased the sensitivity of acetylcholinestase but simultaneously decreased the ability to hydrolyse the substrate acetylcholine. In some strain, the gene has been duplicated restoring the function of the neurotransmission (Bourguet *et al.,* 1996; Lenormand *et al.,* 1998).

COST EVOLUTION AND MECHANISMS OF RESISTANCE

At the beginning, mutation cost can be high for homozygous resistant insects and at times heterosis and recombination reduce and stabilize it by compensation effect between alleles. Moreover, resistance fitness cost is sometimes deleted by modifier gene which is selected after resistance appearance or during the process of selection for the character of resistance (Georghiou, 1964).

The phenomenon of modifier gene is particularly well studied in the Australian sheep blowfly *L. cuprina* (Clarke, 1997), in which diazinon resistance due to an altered carboxylesterase encoded by Rop-1 locus was associated with a fitness cost (Mckenzie *et al.,* 1982). The authors have reported that a fitness cost modifier was responsible for the absence of fitness disadvantage of resistant individuals (Clarke and Mckenzie, 1987; Mckenzie and Game, 1987) and have described a gene for this modifier (Davies *et al.,* 1996). In *Oryzaephilus surinamensis* (L.) resistant to malathion, the selection of modifier alleles that reduce the fitness cost of resistance in the absence of insecticide was also examined by Mason (1998). Like modifier alleles, replacing resistance allele by less costly one (Guillemaud *et al.,* 1998) might explain cost evolution. Overproduced esterase A1 in *C. pipiens* resistance is due to such a mechanism (Rooker *et al.,* 1996).

Furthermore, there is strong evidence that some of these costs at least are conditional, becoming apparent only under conditions of environmental or physiological stress. For example aphids expressing high levels of esterase-based resistance suffer higher mortality than their susceptible counterparts during cold, wet and windy weather. Likewise, sex, genotypes frequency and larval density can also influence cost (Raymond *et al.,* 1993). Moreover, the well studied effect of fitness cost might be attenuated by the fact that a high recombination rate can allow the rapid adaptation of the allele towards lower copy number (Guillemaud *et al.,* 1999). Therefore, those authors noted that with an estimated recombination rate of more than 5%, we may expect that high amplification level could not be maintained in the absence of insecticide selection.

FITNESS COST UTILIZATION IN RESISTANCE MANAGEMENT STRATEGIES

Insecticide selection favors resistant individuals when fitness cost disadvantages them in absence of insecticides. The occurrence and stability of these are strong evidence that both phenomenons act as antagonistic selective pressures (Djihinto, 2004; Djihinto *et al.,* 2009; Lenormand *et al.,* 1999). Basing on this principle and in accord with Georghiou *et al.* (1983), Mallet (1989), Bonning and Hemingway (1991), Hemingway *et al.* (1992), Rodriguez *et al.* (1993), Guillemaud *et al.* (1998), Mason (1998), Lenormand and Raymond (1998), Alvi *et al.* (2012), Yang *et al.* (2013), insecticide rotation, when several kind of insecticides are available, is one of the tools to manage insecticide resistance. Thus, insecticide temporal (window strategy) or spatial (mosaic strategy) rotation which involves alternation of insecticide selection and relaxation for insecticide resistance genes can be used for resistance management.

Nevertheless, insecticide rotation efficacy can encounter some problems. Two main problems are usually described such as migration of insects which can increase resistant individuals in absence of insecticide (Rivet and Pasteur, 1993) and selection of modifier genes that reduce resistance fitness cost (Mason, 1998). In purpose to take into account insect migration in insecticide rotation strategy, a critical size of the treated area (stable zone) must be defined in resistant pest management (Lenormand and Raymond, 1998; Lenormand *et al.,* 1999). This strategy consists of applying insecticides in an area smaller than this critical size, so that gene flow from the untreated area, combined with the fitness cost of resistance genes, prevents its frequency reaching high equilibrium value (Lenormand and Raymond, 1998). Concerning modifier genes, resistant individual

population might increase even if insecticides are not applied. In that moment, insecticide which induces resistance can be changed whether it is possible or another strategy like insecticide mixtures and integrated pests management can be used (Khan *et al.,* 2013; Katary and Djihinto, 2007; Martin *et al.,* 2005; Djihinto, 2004; Martin *et al.,* 2003).

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