Papers Presented in a Symposium on "*Pest Resistance to Pesticides*"* at the Eighth Arab Congress of Plant Protection held in El-Beida, Libya, October 12-16, 2003

Insecticide and Acaricide Resistance

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Abstract

Devine, G.J. 2003. Insecticide and Acaricide Resistance. Arab J. Pl. Prot. 21: 175-180.

Insecticide resistance is an example of a dynamic evolutionary process in which chance mutations conferring protection against insecticides are selected for in treated populations. Since the 1940s, synthetic insecticides have been used on an increasing scale to control the insects and mites that cause immense crop losses and pose major threats to public and animal health. However, because many of the target species have evolved resistance, some of these chemical control programs are failing. At the current time, more than 500 arthropod species have evolved resistance to at least one pesticide, and a few populations of some of those species are now resistant to all, or almost all, of the available products. This article will review the diagnosis and mechanisms of resistance, and their extent across species and chemical groups. It will also review the genetic, ecological and operational factors that affect the rate at which resistance develops. Finally, it will examine how best to combat resistance and will consider some recent success stories in the continuing battle between insect evolution and human ingenuity.

Introduction

In eukaryotes, the phenotypic changes (adaptations) that result from environmental selection pressures are seldom visible over the span of a human lifetime. The evolution of pesticide resistance by arthropods however, is a spectacular exception to the rule.

Since the 1940s, synthetic insecticides have been used on an increasing scale to control the insects and mites that cause immense crop losses and pose major threats to public and animal health. However, because many of the target species have evolved resistance, some of these chemical control programs are failing. At the current time, more than 500 arthropod species have evolved resistance to at least one pesticide, and a few populations of some of those species are now resistant to all, or almost all, of the available products (Fig. 1). About 500,000 metric tons of insecticide is now applied each year in the United States alone, with obvious implications for both human health and the environment. Yet resistant insects continue to affect our agricultural productivity and our ability to combat vectors of disease. The economic burden imposed by insecticide resistance on much of the world, is enormous. In the United States alone, annual losses in crop and forest productivity have been estimated at $1.4 billion. Moreover, it is proving impossible to combat resistance by embarking on a chemical arms race. The development of a new insecticide takes 8 to 10 years at a cost of $20 to $40 million, and the rate of discovery of new insecticidal molecules, unaffected by current resistance mechanisms, seems to be on the wane. Within just a few years of the registration of some of these new molecules, resistant insect populations have evolved.

Diagnosis of Resistance

Although a large number of laboratory bioassay methods have been developed for detecting and characterizing resistance, most of these are limited to defining phenotypes and provide little information on the underlying genes or mechanisms. Thus, although bioassays remain the indispensable mainstay of most large-scale resistance monitoring programs, much attention is being paid to developing more incisive techniques that not only offer greater precision and turnover rates, but also diagnose the type of mechanism(s) present and, whenever possible, the genotypes of resistant insects.

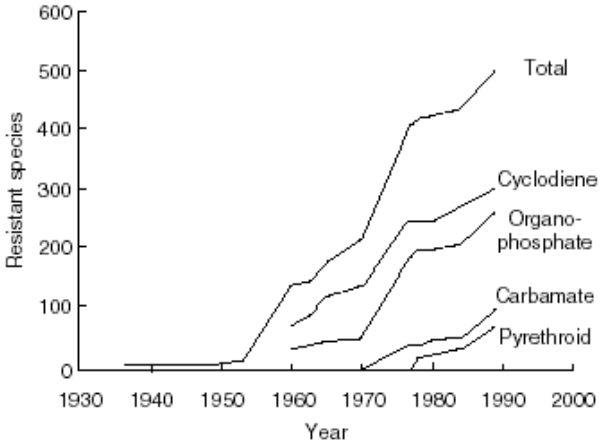

Figure 1. Increase in the number of arthropod species reported to resist insecticides over time, in total, and in response to the four most widely used classes of insecticide [Adapted from Georghiou, G. P. (1990). Overview of insecticide resistance. *In* "Managing Resistance to Agrochemicals" (M. D. Green, H. M. Le Baron and W. K. Moberg, eds.), pp. 18–14. ACS Symposium Series 421. Copyright (1990) American Chemical Society, Washington, DC.]

A variety of approaches are being adopted for this purpose, including electrophoretic or immunological detection of resistance-causing enzymes, kinetic and end-point assays for quantifying the activity of enzymes or their inhibition by insecticides, and DNA-based diagnostics for mutant resistance alleles. The sensitivity of these techniques

* This symposium was sponsored by the FAO Near East Regional Office, Cairo, Egypt.

is exemplified by work on the green peach aphid, *Myzus persicae.* In northern Europe, this insect possesses three coexisting resistance mechanisms: an overproduced carboxylesterase conferring resistance to organophosphates, an altered acetylcholinesterase conferring resistance to certain carbamates, and target-site resistance (i.e., knockdown resistance, kdr) to pyrethroids. These mechanisms collectively confer strong resistance in this species to virtually all available aphicides. Fortunately, it is now possible to diagnose all three mechanisms in individual aphids by using an immunoassay for the overproduced esterase, a kinetic microplate assay for the mutant AChE, and a molecular diagnostic for the *kdr* allele. The combined use of these techniques against field populations provides up-to-date information on the incidence of the mechanisms and serves to inform growers of potential control problems and in the development of optimal strategies for the management of *M. persicae.*

Extent of Resistance

In some insects, resistance extends only to a few closely related compounds in a single chemical class. It may be very weak or restricted to a small part of the insects' geographical range. At the other extreme, some widespread pests, such as anopheline mosquitoes (e.g., *Anopheles gambiae*), the diamondback moth *(Plutella xylostella),* the Colorado potato beetle *(Leptinotarsa decemlineata),* and the sweet potato whitefly *(Bemisia tabaci)* now resist most or all of the insecticides available for their control. The most extensively used insecticide classes— organochlorines, organophosphates, carbamates, and pyrethroids—have generally been the most seriously compromised by resistance, and many principles relating to the origin and evolution of resistance can be demonstrated solely by reference to these fast-acting neurotoxins. In recent years, however, there has also been a worrying increase in resistance to more novel insecticides. These include compounds that attack the developmental pathways of arthropods (e.g., benzoylphenylureas), their respiratory processes [e.g., mitochondrial electron transport inhibiting (METI) acaricides], their digestive systems [e.g., *Bacillus thuringiensis* (Bt) endotoxins], and pathways associated with the regulation of their nervous processes (e.g., neonicotinoids).

Origins and Breadth Resistance

Insecticides are not considered to be mutagenic at their field application rates and are, therefore, not the causative agents of insecticide resistance. Rather they act to select favourable mutations inherent in the population to which they are applied. Some attempts to estimate the rates at which resistant mutations occur have been made. The treatment of blow flies *(Lucilia cuprina)* with a chemical mutagen resulted in the production of dieldrin-resistant target-site mutations in less than one per million individuals. Other studies, however, have found the incidence of resistant mutations to be worryingly high. A recessive allele conferring resistance to Bt toxins in unselected populations of the tobacco budworm, *Heliothis virescens,* was estimated to be present in about one in every thousand individuals in some areas of North America. Sixteen in every hundred insects were found to carry a Bt-resistant allele in unselected populations of the pink bollworm, *Pectinophora gossypiella,* in Arizonan cotton fields. Despite this, Bt cotton remains effective in the control of these species, suggesting that such estimates need to be interpreted carefully. Less empirical measures of mutation rates are extremely variable (10^{-3} to 10^{-16}), but they will undoubtedly be dependent on the resistance mechanism involved.

Resistant mutations seldom confer protection to just a single toxin. Most commonly, they exhibit differing levels of resistance to a range of related and unrelated insecticides. In its strictest sense, the term *cross-resistance* refers to the ability of a single mechanism to confer resistance to several insecticides simultaneously. A more complex situation is that of *multiple resistance,* reflecting the coexistence of two or more resistance mechanisms, each with its own specific cross-resistance characteristics. Disentangling cross-resistance from multiple resistance, even at the phenotypic level, is one of the most challenging aspects of resistance research.

Cross-resistance patterns are inherently difficult to predict in advance, because mechanisms based on both increased detoxification and altered target sites can differ substantially in their specificity. The most commonly encountered patterns of cross-resistance tend to be limited to compounds in the same chemical class. However, even these patterns can be very idiosyncratic. For example, organophosphate resistance based on increased detoxification or target-site alteration can be broad ranging across this group or highly specific to a few chemicals with particular structural similarities. The breadth of target-site resistance to pyrethroids in houseflies is also dependent on the resistance allele present. The *kdr* allele itself affects almost all compounds in this class to a similar extent (ca. 10-fold resistance), whereas resistance due to the more potent *super-kdr* allele is highly dependent on the alcohol moiety of pyrethroid molecules, and ranges from ca. 10-fold to virtual immunity. Cross-resistance between insecticide classes is even harder to anticipate, especially for broad spectrum detoxification systems whose specificity depends not on insecticides having the same mode of action, but on the occurrence of common structural features that bind with detoxifying enzymes. Empirical approaches for distinguishing between cross-resistance and multiple resistance include repeated backcrossing of resistant populations to fully susceptible ones, to establish whether resistance to two chemicals co segregates consistently, and reciprocal selection experiments, whereby populations selected for resistance to one chemical are examined for a correlated change in response to another. If available, biochemical or molecular diagnostics for specific resistance genes can assist considerably with tracking the outcome of genetic crosses or with assigning cross-resistance patterns to particular mechanisms.

Mechanisms of Resistance and their Homology

Depending on the mechanism involved, resistance has been shown to arise through structural alterations of genes encoding target-site proteins or detoxifying enzymes, or through processes affecting gene expression (e.g., amplification or altered transcription). Examples of the former include:

1) Enhanced metabolism of insecticides by cytochrome P450 monoxygenases can potentially confer resistance to most chemical classes. Much of the evidence for this mechanism is indirect, based on the ability of monoxygenase inhibitors to reduce the magnitude of

resistance when used in combination with insecticides in bioassays.

2) Enhanced activity of glutathione *S*-transferases (GSTs) is considered to be potentially important in resistance to some classes of insecticide, including organophosphates. Like monoxygenases, GSTs, exist in numerous molecular forms with distinct properties, making correlations of enzyme activity with resistance very challenging and often ambiguous.
3) Enhanced hydrolysis or sequestration by esterases (e.g., carboxylesterases) capable of binding to and cleaving carboxylester and phosphotriester bonds undoubtedly plays an important role in resistance to organophosphates and pyrethroids. Biochemically, this is the best-characterized detoxification mechanism. Sometimes (e.g., for mosquitoes, blowflies, and *M. persicae*) the esterases have been identified and sequenced at the molecular level. Resistance caused by increased esterase activity can arise through a qualitative change in an enzyme, improving its hydrolytic capacity, or (as in mosquitoes and aphids) a quantitative change in the titer of a particular enzyme that already exists in susceptible insects.

The following examples appear to show that although some adaptations to the environment are unpredictable, the opportunities for insects to modify or reduce binding of insecticides, hence to develop target-site-based resistance mechanisms, are very limited indeed. It is conceivable that most of the mutations that confer such resistance do not allow the organism to retaining normal functioning of the nervous system:

1) Pyrethroids act primarily by binding to and blocking the voltage-gated sodium channel of nerve membranes. Knockdown resistance, or insensitivity of this target site, is the result of structural modifications in a sodium channel protein. The same amino acid substitution (leucine 1014 to phenylalanine) in a sodium channel protein confers a "basal" kdr phenotype in a range of species including house flies, cockroaches, the green peach aphid, the diamondback moth, and a mosquito *(A. gambiae).* This phenotype may subsequently be enhanced (to "super-kdr" resistance) by further mutations that also recur between species.
2) GABA receptors are targets for several insecticide classes including cyclodienes, avermectins, and fipronils. The primary mechanism of resistance to cyclodienes and fipronils involves modification of a particular GABA receptor subunit, resulting in substantial target-site insensitivity to these insecticides. The target-site mechanism of cyclodiene resistance has been attributed to the same amino acid substitution (alanine 302 to serine) in the GABA receptors of several species of diverse taxonomic origin including *Drosophila,* several beetles, a mosquito *(Aedes aegypti),* a whitefly *(B. tabaci),* and a cockroach *(Blatella germanica).*
3) Organophosphates and carbamates exert their toxicity by inhibiting the enzyme acetylcholinesterase (AChE), thereby impairing the transmission of nerve impulses across cholinergic synapses. Mutant forms of AChE showing reduced inhibition by these insecticides have been demonstrated in several insect and mite species. Biochemical and molecular analyses of insecticide-insensitive AChE have shown that pests may possess several different mutant forms of this enzyme with contrasting insensitivity profiles, thereby conferring distinct patterns of resistance to these two insecticide classes. Some of these resistance mechanisms are illustrated schematically in Fig. 2.

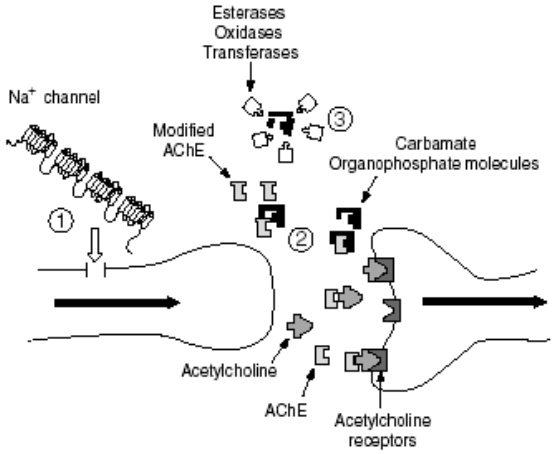

Figure 2. Schematic diagram of a nerve synapse showing examples of insecticide resistance mechanisms: (1) changes in the structure of the sodium channel confer kdr or super-kdr target-site resistance to pyrethroids; (2) modified AChE is no longer bound by organophosphates and remains available to break down acetylcholine molecules after neurotransmission across the synapse; (3) detoxifying enzymes degrade or sequester insecticides before they reach their targets in the nervous system.

Factors Affecting the Evolution of Resistance

As an evolutionary trait, insecticide resistance is unusual in that we can identify the main selection pressure with ease, but the rate at which resistance develops is governed by numerous biotic and abiotic factors. These include the genetics and ecology of the pests and their resistance mechanisms, and the operational factors that relate to the chemical itself and to its application. To manage resistance effectively, an assessment of genetic, ecological, and operational risk is required. Although this can be done empirically on a species-by-species basis, one of the great challenges of the future is to understand why some species seem to have a greater tendency to become resistant than others.

Genetic Influences: To predict how quickly resistance will become established, it is necessary to understand how resistant alleles affect the survival of phenotypes in the field. For example, the dominance of resistance genes exerts a major influence on selection rates. In laboratory bioassays evaluating the relative survival of susceptible homozygotes (SS), heterozygotes (RS), and resistance homozygotes (RR) over several insecticide concentrations, RS individuals usually respond in an intermediate manner. In the field, however, dominance is dependent on the concentration of insecticide applied and its uniformity over space and time. Even when the initial concentration is sufficient to kill RS individuals (rendering resistance effectively recessive), upon weathering or decay of residues, this genotype may later show increased survival, with resistance becoming functionally dominant in expression. When resistance genes are still rare, hence mainly present in heterozygous condition, this sequence can have a profound effect in accelerating the selection of resistance genes to economically damaging frequencies.

The diverse mating systems of insects also influence the rate at which resistance evolves. Although most research has focused on outcrossing diploid species (typified by members of the Lepidoptera, Coleoptera, and Diptera), systems based on haplodiploidy and parthenogenesis also occur among key agricultural pests. In haplodiploid systems, males are usually produced uniparentally from unfertilized, haploid eggs, and females are produced biparentally from fertilized, diploid eggs. The primary consequence of this arrangement (exemplified by whiteflies, spider mites, and phytophagous thrips) is that resistance genes are exposed to selection from the outset in the hemizygous males, irrespective of intrinsic dominance or recessiveness. Whether a resistance gene is dominant, semidominant, or recessive, resistance can develop at a similar rate. Most species of aphid undergo periods of parthenogenesis (in which eggs develop and give rise to live offspring in the absence of a paternal genetic contribution) promoting the selection of clones with the highest levels of resistance and/or the most damaging combination of resistance mechanisms. In fully anholocyclic (asexual) populations, such as those of *M. persicae* in northern Europe, the influence of parthenogenesis has led to strong and persistent associations between resistance mechanisms within clonal lineages.

Ecological Influences: Fecundity and generation times have a huge bearing on the evolution of resistance in a population. The greater the number of individuals, and the faster they reproduce and attain maturity, the higher the likelihood that a favorable mutation will occur, and be maintained in the population. Faster growth and higher population numbers will also have an effect on the size of a pest population, and therefore the need for insecticide treatment. The dispersal capabilities of pests can also act as primary determinants of resistance development. Movement of pests between untreated and treated parts of their range may delay the evolution of resistance because of the diluting effect of susceptible immigrants. Conversely, large-scale movement can also accelerate the spread of resistance by transferring resistance alleles between localities. A good example relates to the two major bollworm species (Lepidoptera: Noctuidae) attacking cotton in Australia. Only the cotton bollworm *Helicoverpa armigera,* has developed strong resistance. *H. punctigera,* despite being an equally important cotton pest, has remained susceptible to all insecticide classes. The most likely explanation is that *H. punctigera* occurs in greater abundance on a larger range of unsprayed hosts than *H. armigera,* thereby maintaining a large pool of unselected, susceptible individuals, which dilute resistant mutations arising on treated crops.

In the absence of insecticidal selection pressure, resistance genes can impose fitness costs on their carriers. Sometimes these costs are quite subtle and difficult to determine. In *M. persicae,* resistant individuals are less inclined to move from senescing to younger leaves and are therefore more vulnerable to isolation and starvation after leaf abscission. These costs appear to contribute to a decline in the frequency of resistant insects between cropping seasons.

Operational Influences: Operational factors are at human discretion and can be manipulated to influence selection rates. Factors exerting a major influence in this respect include the rate, method, and frequency of applications, their biological persistence, and whether insecticides are used singly or as mixtures of active ingredients. Equating operational factors with selection is often difficult, since without detailed knowledge of the mechanisms present it is impossible to test many of the assumptions on which genetic models of resistance are based. If resistance alleles are present, the only entirely nonselecting insecticide doses will be ones sufficiently high to overpower all individuals, regardless of their genetic composition, or ones so low that they kill no insects at all. The latter is obviously a trivial option. Prospects of achieving the former depend critically on the potency and dominance of resistance genes present. A pragmatic solution to this dilemma is to set application doses as far above the tolerance range of homozygous, susceptible individuals as economic and environmental constraints permit, in the hope that any heterozygotes that do arise will be effectively controlled. However, this approach will obviously be ineffective if resistance turns out to be more common than suspected (resulting in the presence of homozygous resistant individuals) or if resistance alleles exhibit an unexpectedly high degree of dominance (and heterozygotes are therefore phenotypically resistant). Unless a high proportion of insects escape exposure altogether, the consequence could then be very rapid and effective selection for homozygous resistant populations. In practice, concerns about optimizing dose rates to avoid resistance are secondary to those related to the application process itself. Delivery systems and/or habitats promoting uneven or inadequate coverage will generally be more prone to select for resistance, because, under these circumstances, pests are likely to encounter suboptimal doses of toxins that will permit survival of heterozygous individuals. The timing of insecticide applications relative to the life cycle of a pest can also be an important determinant of resistance. A good example of this is found in the selection of pyrethroid resistance in *H. armigera* in Australia. On cotton foliage freshly treated with the recommended field dose, pyrethroids killed larvae up to 3 to 4 days old irrespective of whether they were resistant by laboratory criteria. Since the sensitivity to pyrethroids of larvae of all genotypes was found to decline with increasing larval size, the greatest discrimination between susceptible and resistant phenotypes occurred when larvae achieved a threshold age. Targeting of insecticides against newly hatched larvae, as is generally advocated for bollworm control, not only increases the likelihood of contacting larvae at the most exposed stage in their development but also offers the greatest prospect of retarding resistance by overpowering its expression. It may also have the effect of reducing genetic variation and therefore the potential number of resistant mutations. Indeed, it is also possible to impose genetic "bottlenecks" by applying pesticides when populations are already low (e.g, when they are overwintering). Although such a tactic might be beneficial where populations are fully susceptible, if resistant mutations are already present, it might act to increase their frequency.

In theory, the application of two or more unrelated chemicals as insecticide mixtures offers substantial benefits for delaying the selection of resistance. The underlying principle is one of "redundant killing," whereby any individuals already resistant to one insecticide are killed by simultaneous exposure to another, and vice versa. However, achieving this objective requires that each type of resistance be rare and that both ingredients persist throughout the effective life of an application. Otherwise, one compound will exert greater selection pressure than the other, and the advantage of applying a mixture will be lost.

Combating Insecticide Resistance

Insecticide resistance management (IRM) aims to intervene in the evolutionary process and either overcome resistance or prevent its appearance in the first place. There are several practical, economic, and political constraints on IRM tactics that may be chosen and the precision with which they can be applied:

1) The properties of any resistance genes present often are unknown, and knowledge of pest ecology may still be rudimentary.
2) It is often necessary to contend with a whole pest complex rather then just a single pest species.
3) There may be a very limited number of insecticides available for use in management strategies.
4) For highly mobile pests, at least, countermeasures may need to be standardized and synchronized over large areas, sometimes whole countries.
5) Resistance is a dynamic phenomenon; that is, any mechanisms already known to exist may change over time.
6) To promote compliance with management strategies, the tactics adopted should be as unambiguous, rational, and simple as possible.

A strategy first implemented on Australian cotton in 1983 against *H. armigera* illustrated many features of large-scale attempts at resistance management. Introduced in response to unexpected, but still localized, outbreaks of pyrethroid resistance in *H. armigera,* the strategy was based primarily on the concept of insecticide rotation. The threat of pyrethroid resistance was countered by restricting these chemicals to a maximum of three sprays within a prescribed time period coincident with peak bollworm damage. To diversify the selection pressures being applied, farmers were required to use alternative insecticide classes at other stages of the cropping season. Initially, this strategy had the desired effect of preventing a systematic increase in the frequency of pyrethroid-resistant phenotypes. Additional recommendations, including the targeting of insecticides against newly hatched larvae (the most vulnerable life stage) and the ploughing in of cotton stubble to destroy resistant pupae overwintering in the soil, undoubtedly contributed to this success.

Another strategy incorporating a wide range of chemical and non-chemical countermeasures was introduced on Israeli cotton in 1987. The primary objective was conservation of the effectiveness of insecticides against the *B. tabaci.* Under recommendations coordinated by the Israeli Cotton Board, important new whitefly insecticides are restricted to a single application per season within an alternation strategy optimized to contend with the entire cotton pest complex and to exploit biological control agents to the greatest extent possible. One major achievement of this strategy has been a dramatic reduction in the number of insecticide applications against the whole range of cotton pests, but especially against *B. tabaci.* Sprays against whiteflies now average fewer than two per growing season compared with over 14 per season in 1986. Most importantly, the strategy has generated an ideal environment for releasing additional new insecticides onto cotton and for managing them effectively from the outset. An integral part of delaying or preventing the evolution of resistance is the preservation of the innate "susceptibility" of a pest species. The most effective way to conserve susceptibility, based both on evolutionary models and on empirical evidence, is to ensure the presence of pesticide-free "refugia" in which susceptible genotypes may survive and reproduce. The inclusion of refugia as essential components of IRM strategies is a recent phenomenon, signalling that pest management is no longer simply about eradication, but is now at least partially focused on conservation.

Transgenic Plants

A recent development in crop protection has been the release of crop plants genetically engineered to express genes for insecticidal toxins derived from the microbe *B. thuringiensis.* In 2002 the total area worldwide planted to Bt plants was estimated to exceed 15 million ha. Existing toxin genes in Bt cotton and corn are active specifically against certain key lepidopteran pests (especially bollworms and corn borers); another engineered into potatoes provides protection against the Colorado potato beetle. Aside from their commercial prospects, insect-tolerant transgenic crops offer numerous potential benefits to agriculture. By affording constitutive expression of toxins in plant tissues throughout a growing season, the incorporation of Bt genes into crops could reduce dramatically the use of conventional broad-spectrum insecticides against insect pests, as well as remove the dependence of pest control on extrinsic factors such as climate and on the efficiency of traditional application methods. However, this high and persistent level of expression also introduces a considerable risk of pests adapting rapidly to resist genetically engineered toxins. To date, there are no substantiated reports of resistance selected directly by exposure to commercial transgenic crops, but resistance to conventional Bt sprays (selected in either the laboratory or the field) has been reported in more than a dozen insect species. Research into the causes and inheritance of such resistance is providing valuable insights into the threats facing Bt plant and the efficacy of possible countermeasures.

Tactics proposed for sustaining the effectiveness of Bt plants have many parallels with ideas considered for managing resistance to conventional insecticides. However, they are more limited in scope because of the long persistence and constitutive expression of engineered toxins, and because of the limited diversity of transgenes currently available. Indeed, for existing "single-gene" plants, the only prudent and readily implementable tactic is to ensure that substantial numbers of pests survive in non-transgenic refugia. These can be incorporated into the crop itself, or they may comprise alternative host plants. The success of this strategy is dependent on some key assumptions: (1) that resistant mutations are recessive or at least only partially dominant, so that their heterozygous forms can be controlled by the toxins expressed; (2) that refugia will produce enough susceptible insects to ensure that insects carrying resistant alleles do not meet and mate; and (3) that resistant alleles will carry a fitness cost, rendering insects less fit when the selection pressure is removed (e.g, outside the growing season when the insect is dependent on other crops). In the longer term, there are potentially more durable options for resistance management: stacking (or pyramiding) of two or more genes in the same cultivar, or possibly rotations of cultivars expressing different single toxins. Whatever measures are adopted, it is essential that plants expressing transgenes be exploited as components of multitactic strategies rather than as a panacea for resistance problems with conventional insecticides.

الملخص

دفين، جريغور جون. 2003. المقاومة لمبيدات الحشرات والحلم. مجلة وقاية النبات العربية. 21: 175−180.

تعد المقاومة لمبيدات الحشرات مثالاً عن عملية تطور ديناميكي، يتم خلالها انتخاب الطفرات التي تضفي حماية من مبيدات الحشرات من العشيرة المعاملة. ومنذ الأربعينات، استخدمت المبيدات المصنعة للحشرات على نحو متزايد لمكافحة الحشرات والحلم التي تسبب خسائر جسيمة للمحصول وتحدث مخاطر كبيرة لصحة الإنسان والحيوان. ونظراً لأن معظم الأنواع المستهدفة قد طورت سلالات مقاومة فقد أخفقت برامج المكافحة الكيميائية. وفي وقتنا الحاضر، هناك حوالي 500 نوعاً من مفصليات الأرجل قد طورت مقاومة لمبيد واحد على الأقل، كما أن هناك عشائر قليلة من بعض الأنواع قد طورت مقاومة لمعظم المنتجات المتوافرة تقريباً وستعالج المقالة تشخيص المقاومة للمبيدات وآليتها ومدى انتشارها ما بين الأنواع والمجموعات الكيميائية. كما ستراجع العوامل الوراثية والبيئية والعملياتية التي تؤثر في معدل تطور المقاومة. كما أنها ستتناول أفضل الطرائق للتغلب على تطور المقاومة للمبيدات، وستقدم بعض الأمثلة الناجحة في الصراع المستمر ما بين تطور الحشرات وعبقرية الإنسان.

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Further Reading

Denholm, I. and M.W. Rowland. 1992. Tactics for managing pesticide resistance in arthropods—Theory and practice. Annu. Rev. Entomol. 37: 91–112.

Devonshire, A.L., L.M. Field, S.P. Foster, G.D. Moores, M.S. Williamson and R.L. Blackman. 1998. The evolution of insecticide resistance in the peach-potato aphid, *Myzus persicae.* Philos. Trans. R. Soc. Lond. Ser. B, Biol. Sci. 353: 1677–1684.

French Constant, R.H., B. Pittendrigh, A. Vaughan and N. Anthony. 1998. Why are there so few resistance-associated mutations in insecticide target genes? Philos. Trans. R. Soc. Lond. Ser. B, Biol. Sci. 353: 1685–1693.

Forrester, N.W., M. Cahill, L.J. Bird and J.K. Layland. 1993. Management of pyrethroid and endosulfan resistance in *Helicoverpa armigera* (Lepidoptera, Noctuidae) in Australia. Bull. Entomol. Res. supple. 1.

Horowitz, A.R., G. Forer and I. Ishaaya. 1994. Managing resistance in *Bemisia tabaci* in Israel with emphasis on cotton. Pestic. Sci. 42: 113–122.

McKenzie, J.A. 1996. "Ecological and Evolutionary Aspects of Insecticide Resistance." R. G. Landes, Austin, TX.

Pimental, D., H. Acquay, M. Biltonen, P. Rice, M. Silva, J. Nelson, V. Lipner, S. Giordano, A. Horowitz and M. Damore. 1992. Environmental and economic costs of pesticide use. Bioscience 42: 750–760.

Raymond, M., C. Chevillon, T. Guillemaud, T. Lenormand and N. Pasteur. 1998. An overview of the evolution of overproduced esterases in the mosquito *Culex pipiens.* Philos. Trans. R. Soc. Lond. Ser. B, Biol. Sci. 353: 1707–1711.

Roush, R.T. 1989. Designing resistance management programs—how can you choose? Pestic. Sci. 26: 423–441.

Roush, R.T. 1997. Bt-transgenic crops: Just another pretty insecticide or a chance for a new start in resistance management? Pestic. Sci. 51: 328–334.

Soderlund, D.M. and J.R. Bloomquist. 1990. Molecular mechanisms of insecticide resistance. Pages 58-96. *In:* Pesticide Resistance in Arthropods. R.T. Roush and B.E. Tabashnik (editors.). Chapman & Hall, London.

Tabashnik, B.E., Y.B. Liu, T. Malvar, D.G. Heckel, L. Masson and J. Ferre. 1998. Insect resistance to *Bacillus thuringiensis:* Uniform or diverse? Philos. Trans. R. Soc. Lond. Ser. B, Biol. Sci. 353: 1751–1756.