Review

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Western flower thrips resistance to insecticides: detection, mechanisms and management strategies

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Abstract

Insecticide resistance continues to be one of the most important issues facing agricultural production. The challenges in insecticide resistance and its management are exemplified by the situation with the western flower thrips *Frankliniella occidentalis* (Pergande) (Thysanoptera: Thripidae). This highly invasive pest has a great propensity for developing insecticide resistance because of its biological attributes, and cases of resistance to most classes of insecticides used for its management have been detected. To combat insecticide resistance in the western flower thrips, several insecticide resistance management (IRM) programs have been developed around the world, and these are discussed. Successful programs rely on non-insecticidal tactics, such as biological and cultural controls and host plant resistance, to reduce population pressures, rotations among insecticides of different mode of action classes to conserve insecticide efficacy, resistance monitoring, sampling to determine the need for insecticide applications and education to assure proper implementation. More judicious insecticide use is possible with the development of well-founded economic thresholds for more cropping systems. While growers will continue to rely on insecticides as part of western-flower-thrips- and thrips-transmitted virus management, more effective management of these pests will be achieved by considering their management in the context of overall integrated pest management, with IRM being a key component of those comprehensive programs.

Keywords: thrips; insecticide resistance management; *Frankliniella occidentalis*; IPM

1 INTRODUCTION

Although the nature of insecticide use in agriculture has changed dramatically over time since the advent of modern synthetic insecticides, it is most likely that insecticides will continue to be the major tool used to manage insect pests in agricultural crops. This reliance is particularly true for high-value specialty horticultural and ornamental crops (http://www.ams.usda.gov/AMSv1.0/scbgpdefinitions), where the perceived risks of insect damage often encourage growers to make intensive insecticide applications. A byproduct of this reliance on insecticides for pest management has been the continued and mounting development of insecticide resistance among target pests.

By definition, insecticides are designed to kill insects, and therefore their use imposes an intense selective force on target populations. As susceptible individuals are eliminated from a population, the proportion of individuals with resistant phenotypes will increase. Once the frequency of resistant individuals in a population reaches a critical point where the application of an insecticide fails to produce an expected level of control, that population may be termed 'resistant'.[1] It should be noted that individuals are the ones that possess traits for resistance or susceptibility; therefore 'resistant' populations may still possess a certain frequency of susceptible individuals.

The evolution of resistance has long been considered an inevitable outcome of insecticide use,[2] and these concerns have largely been borne out, as the number of cases of insecticide resistance has continued to increase over time.[3] Fortunately, theoretical and empirical evidence shows that the evolution of resistance can be managed, and, with proper insecticide use, it is possible for a resistant population to revert to a susceptible state. A population that has reverted to a susceptible state will still contain a certain frequency of individuals carrying resistant alleles, but that frequency will be low enough not to cause economic damage. However, without proper management, reuse of the same insecticide(s) would quickly lead to a resurgence in resistance.[2]

Managing resistance to insecticides is dependent on a thorough understanding of the population genetics and ecology of the target pest and the target pest's interaction with toxicants, and on the application of this information in a practical manner that growers can successfully implement. Considerable attention

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has been devoted to the development of insecticide resistance management (IRM) programs as a means to preserve and extend the useful life of particular insecticides. The interest in IRM has been spurred on further by (1) the loss of registered products because of human health or environmental concerns and (2) the lengthy process needed to discover, develop and register novel insecticides for commercial use. Although IRM is often presented as a distinct field of inquiry, it should be viewed as an integral component of modern integrated pest management (IPM) systems that cannot logically be separated from an overarching IPM program. There are several basic tenets of IRM that are consistent across programs, although the details of their implementation may vary from system to system.

The western flower thrips *Frankliniella occidentalis* (Pergande) (Thysanoptera: Thripidae) provides a model organism for understanding the complex interactions among the insect's biological attributes that facilitate the evolution of insecticide resistance and the cropping systems that have led to insecticide resistance. Successes in managing insecticide resistance with this difficult pest help to exemplify characteristics of IRM programs that can be incorporated into overall IPM programs.

The western flower thrips is a highly polyphagous herbivore and one of the most important pests of many crops throughout the world.[4–6] Western flower thrips inflict plant damage through oviposition, which produces aesthetic damage to fruiting crops, and through the feeding action of adults and larvae, which scars foliage, flowers and fruits.[7] Most importantly, western flower thrips is able to transmit several species of destructive plant viruses in the genus *Tospovirus* (Bunyaviridae),[8,9] including tomato spotted wilt virus (TSWV) and *Impatiens* necrotic spot virus (INSV), of which it is the most important vector worldwide.

Invasive populations of the western flower thrips began to spread globally as the international trade in horticultural products began to expand during the 1970s.[10] Although it is difficult to establish with certainty, it is likely that populations derived from California greenhouses had already developed resistance to numerous insecticides before becoming invasive.[11,12] Evidence from monitoring studies conducted in Australia soon after the discovery of western flower thrips in 1993 indicated that there was a high degree of tolerance to a range of carbamates, organophosphates and pyrethroids.[13] Regardless of their status in terms of insecticide resistance at the time of introduction, it is clear that insecticide resistance has continued to be a widespread problem with the western flower thrips, as populations have continued to evolve resistance to all manner of new insecticides. The Arthropod Pesticide Resistance Database, maintained by Michigan State University (www.pesticideresistance.org), lists at least 153 documented cases of insecticide resistance in western flower thrips populations from around the world. These cases of resistance involve insecticides in at least seven of the chemical classes currently recognized by the Insecticide Resistance Action Committee (IRAC) (Table 1).

2 MECHANISMS OF INSECTICIDE RESISTANCE

Four general types of mechanism for insecticide resistance have been identified: metabolic detoxification, reduced penetration of toxicants, alterations of target sites for toxicants and behavioral resistance.[14] Most documented cases of insecticide resistance in western flower thrips result from generalized metabolic detoxification pathways, but often multiple mechanisms have been identified as contributing to resistance within populations (Table 1). The propensity for generalized metabolic detoxification to confer resistance is thought to derive from the polyphagous nature of the western flower thrips[11] and the inherent need to detoxify plant allelochemicals. There are three general enzyme systems that herbivorous insects, including western flower thrips, utilize to metabolize toxicants: cytochrome-P450-dependent monooxygenases (P450s), esterases and glutathione *S*-transferases (GSTs).[15] These enzymes have generalized actions that convert hydrophobic compounds to less biologically active hydrophilic compounds.[16] Highly polyphagous species, such as western flower thrips, tend to have a greater abundance and diversity of genes that encode for these enzymes than do species with more specialized feeding regimes, enabling such generalists to contend with a range of different insecticide classes.[15]

Of these three types of enzyme system, P450s appear to be the most important in imparting metabolic resistance in western flower thrips (Table 1). There is direct evidence for oxidative metabolism by P450s conferring resistance to a wide range of insecticides, including carbamates, organophosphates, organochlorines, pyrethroids, neonicotinoids and avermectins.

Enhanced activity of esterases has been associated with insecticide resistance in several populations of western flower thrips, but esterase activity alone does not appear to confer resistance. Rather, esterase activity appears to work in concert with other mechanisms in conferring resistance. Maymó *et al*.[17] found greater esterase activities in western flower thrips from populations that were resistant to either the pyrethroid acrinathrin or to the carbamate methiocarb. However, in this study, no other potential resistance mechanisms were assayed for, and the relatively modest increases in esterase activity ($<$2.5 times that of the reference susceptible population) may indicate the presence of additional resistance mechanisms.[18] In another study, Maymó *et al*.[19] were able to assay individual thrips for both esterase and GST activity, and found increased activity for both types of enzyme for individuals from field-collected populations with low levels of resistance to the organochlorine endosulfan. Jensen[20] found that methiocarb resistance in populations from Danish greenhouses was associated with increased esterase activity. However, bioassays with enzyme synergists showed that inhibition of esterases had less of an effect on suppression of resistance than did inhibition of P450s.

Similar to results that demonstrate esterases to be a component of resistance systems for particular insecticides in western flower thrips, GSTs generally have not been found to be sole mechanisms of insecticide resistance. In one population selected for endosulfan resistance, inhibition of GSTs by the synergist diethyl maleate (DEM) did produce significant reductions in resistance levels, whereas inhibition of P450s and esterases did not reduce resistance levels.[18] However, in most of the other populations examined in that study, resistance was found to be suppressed more by inhibition of P450s than by suppression of other metabolic detoxification systems.

Other non-metabolic resistance mechanisms, including reduced toxicant penetration, and insensitivity to toxicants through altered target sites, including knockdown resistance, have been identified.[21] Reduced penetration of an insecticide through the insect cuticle or gut wall is not considered to be a powerful resistance mechanism, in and of itself.[16] However, it can synergize the effect of other resistance mechanisms. For example, a reduced rate of entry of toxicants into the insect's body may enable metabolic detoxification to occur without the enzyme systems of

Table 1. Representative reports of laboratory, greenhouse and field resistance to insecticides and resistance mechanisms in the western flower thrips *Frankliniella occidentalis*

IRAC MOA class[a]	Insecticide	Location (condition)	Year of detection	Resistance ratio[b]	Mechanism[c]	Reference
1A	Bendiocarb	Columbia, Kansas City, Joplin, St Louis, MO (greenhouse)	1992–1993	0.9–11 (LC_{90})	P450	39, 95
1A	Carbosulfan	Yesha, Israel (greenhouse)	1995	22.2 (LC_{90})	Unknown	96
1A	Methiocarb	Murcia, southeastern Spain (field)	2000–2001	22.3 (LC_{50})	P450	18, 97
1A	Methiocarb	Denmark (greenhouse)	1996	2.1–34 (LC_{50})	P450, esterase, GST, altered AChE	20
1A	Methiocarb	Yesha, Israel (greenhouse)	1995	35.4 (LC_{90})	Unknown	96
1A	Methomyl	San Diego, CA (greenhouse)	1992	43–102 (LC_{50})	Unknown	12
1A	Methomyl	Santa Barbara, CA (greenhouse)	1992	42–180 (LC_{50})	Unknown	12
1A	Methomyl	Columbia, Kansas City, Joplin, St Louis, MO (greenhouse)	1992–1993	3.4–26 (LC_{90})	Unknown	39
1A	Methomyl	Columbia, MO (laboratory)	1992	3.6 (LC_{90})	Unknown	39
1B	Acephate	Kenya (field)	1990	141–244 (LC_{50})	Unknown	40
1B	Acephate	Denmark (greenhouse)	1990	54–96 (LC_{50})	Unkown	40
1B	Acephate	Switzerland (greenhouse)	1990	100 (LC_{50})	Unkown	40
1B	Chlorpyrifos	Santa Barbara, CA (greenhouse)	1992	14–16 (LC_{50})	Unknown	12
1B	Diazinon	Columbia, Kansas City, Joplin, St Louis, MO (greenhouse)	1992–1993	10.4–98 (LC_{50})	P450	39
1B	Diazinon	Kansas City, MO (laboratory)	1989	271 (LC_{90})	P450, altered AcHE	24, 39
1B	Diazinon	Columbia, MO (laboratory)	1992	14 (LC_{90})	P450	24
2A	Endosulfan	Murcia, southeastern Spain (field)	2000–2001	3.6–4.6 (LC_{50})	GST	18, 97
2B	Fipronil	NSW, Australia (field)	2001–2003	35% survival at discriminating dose	Unknown	27
3A	Acrinathrin	Murcia, southeastern Spain (field)	2000–2001	29.8 (LC_{50})	P450	18, 97
3A	Acrinathrin	Almeria, Spain (field)	2003	43 (LC_{50})	P450	98
3A	Acrinathrin	WA, NSW and Queensland, Australia (field)	2003	15–78 (LC_{50})	Unknown	76
3A	alpha-Cypermethrin	WA, NSW and Queensland, Australia (field)	2003	15–45 (LC_{50})	Unknown	76
3A	Bifenthrin	San Diego, CA (greenhouse)	1992	70–106 (LC_{50})	Unknown	12
3A	Bifenthrin	Santa Barbara, CA (greenhouse)	1992	142–275 (LC_{50})	Unknown	12
3A	Bifenthrin	WA, NSW and Queensland, Australia (field)	2003	23–61 (LC_{50})	Unknown	76
3A	Cyhalothrin	Haidian, Beijing, China (greenhouse)	2010	39.67 (LC_{50})	Unknown	44
3A	Cypermethrin	Columbia, Kansas City, Joplin, St Louis, MO (greenhouse)	1992–1993	18.3–273 (LC_{90})	Unknown	39
3A	Cypermethrin	Columbia, MO (laboratory)	1992	232 (LC_{90})	Unknown	39
3A	Cypermethrin	Altinova, Turkey (greenhouse)	2002	2.9–9.6 (LC_{90})	Unknown	99
3A	DDT	Kansas City, MO (laboratory)	1989	6.0 (LC_{90})	Unknown	39
3A	Deltamethrin	Ontario, Canada (greenhouse)	1997	Significantly greater survivorship than in susceptible population	P450?	71
3A	Deltamethrin	WA, NSW and Queensland, Australia (field)	2003	15–70 (LC_{50})	Unknown	76
3A	Esfenvalerate	WA, NSW and Queensland, Australia (field)	2003	15–26 (LC_{50})	Unknown	76

Table 1. (*Continued*)

IRAC MOA class[a]	Insecticide	Location (condition)	Year of detection	Resistance ratio[b]	Mechanism[c]	Reference
3A	Fenvalerate	Kansas City, MO (laboratory)	1989	3.6 (LC_{90})	P450, reduced penetration	22, 39
3A	Formetanate	Murcia, southeastern Spain (field)	2000–2001	23.0 (LC_{50})	P450	18, 97
3A	Permethrin	San Diego, CA (greenhouse)	1992	1182–1217 (LC_{50})	P450	12
3A	Permethrin	Santa Barbara, CA (greenhouse)	1992	42–495 (LC_{50})	P450, *kdr?*	12
3A	Permethrin	Kansas City, MO (laboratory)	1989	2.5 (LC_{90})	Unknown	39
3A	Permethrin	WA, NSW and Queensland, Australia (field)	2003	32–79 (LC_{50})	Unknown	76
3A	tau-Fluvalinate	WA, NSW and Queensland, Australia (field)	2003	167–1300 (LC_{50})	P450?	76
4A	Imidacloprid	Kansas City, MO (laboratory)	1989	14 (LC_{90})	Unknown	39
4A	Imidacloprid	Shandong, China (laboratory)	2009	7.7 (LC_{50})	P450	100
5	Spinosad	Almeria, Spain (greenhouse)	2003	>13500 (LC_{50})	Altered nAChR	26
5	Spinosad	Murcia, southeastern Spain (greenhouse)	2004	>3682 (LC_{50})	Altered nAChR	26
5	Spinosad	Urbana, IL (field)	2001	Greater odds of survival in field test	Unknown	101
5	Spinosad	Haidian, Beijing, China (laboratory)	2006	80.8 (LC_{50})	Altered nAChR	44
5	Spinosad	Mengtougou, Beijing, China (greenhouse)	2010	35.38 (LC_{50})	Altered nAChR	44
5	Spinosad	Hyogo Prefecture, Japan (laboratory)	1998	14 (LC_{50})	Altered nAChR	29
6	Abamectin	San Diego, CA (greenhouse)	1992	20–240 (LC_{50})	Unknown	12
6	Abamectin	Santa Barbara, CA (greenhouse)	1992	67–113 (LC_{50})	Unknown	12
6	Abamectin	Haidian, Beijing, China (laboratory)	2003	45.5 (LC_{50})	P450	43
6	Abamectin	Havat HaB'sor, Israel (greenhouse)	1997	9.0 (LC_{90})	Unknown	96

[a] Insecticide Resistance Action Committee mode of action class (http://www.irac-online.org/eClassification/).
[b] LC_{50} and LC_{90} values of resistant strains compared with the LC_{50} and LC_{90} values for the reference susceptible strains.
[c] P450: cytochrome-P450-dependent monooxygenases; GST: glutathione *S*-transferase; altered AChE: altered acetylcholinesterase target site; altered nAChR: altered nicotinic acetylcholine receptor; *kdr*: knockdown resistance; ?: mechanism not clearly established.

the insect being overwhelmed. Zhao *et al*.[22] demonstrated such a phenomenon in a western flower thrips population that was resistant to the pyrethroid fenvalerate. They found that the rate of entry of fenvalerate into resistant population thrips was one-third of the rate of entry into susceptible thrips. They further showed that metabolism of the fenvalerate that penetrated was more rapid in resistant thrips than in susceptible ones.

Indirect evidence for reduced nerve sensitivity to pyrethroids through knockdown resistance (*kdr*) has been reported for greenhouse populations of western flower thrips from at least two geographic regions.[12,22] In these cases, *kdr* conferred resistance, but it was not the most important resistance mechanism operating in those populations. This conclusion has been supported by direct evidence from a study of the genetics of pyrethroid resistance. Forcioli *et al*.[23] found direct evidence for the presence of *kdr* in a laboratory population of western flower thrips, but this mechanism provided only low levels of resistance to deltamethrin.

Alterations in acetylcholinesterase, which is the target site for organophosphate and carbamate insecticides, have been implicated as a mechanism in cases of resistance to diazinon[24] and methiocarb.[20] However, in these cases, resistance based on acetylcholinesterase insensitivity has been identified as just one of several resistance mechanisms in operation within populations.

Another class of insecticides with a novel mode of action is the spinosyns (IRAC class 5). It appears that the major component of spinosad, spinosyn A, affects an unidentified nicotinic acetylcholine receptor that is not the target site of other classes of insecticides such as abamectin (class 6, avermectins) or imidacloprid (class 4A, neonicotinoids).[25] Recently, there have been a number of cases of rapid resistance development to spinosyns that have been attributed to their overuse by growers who lack efficacious alternatives.[26–28] For example, the overuse of spinosad (Dow Agrosciences) in greenhouse production systems in southeastern Spain led to resistance development within 2 years of spinosad's introduction.[26] The mechanism involved in resistance to spinosyns appears to be related to alterations in the nicotinic acetylcholine receptor, and metabolic resistance does not appear to play a role in resistance.[26,29] In spite of inferring that a similar resistance mechanism was operating in these cases, Bielza *et al*.[30] concluded that spinosad resistance in Spanish populations was monogenic, whereas Zhang *et al*.[29] determined that resistance in a laboratory selected strain from Japan was polygenic.

Although behavioral resistance has not been described for western flower thrips, this may be the result of a lack of testing for it rather than its true absence as a mechanism.[31] The natural thigmotactic behavior of western flower thrips, in which individuals preferentially reside in enclosed, concealed spaces on plants,[32] is thought to reduce the direct exposure of individuals to contact insecticides.[16] Therefore, it is possible that spray applications of insecticides could select for increased cryptic behavior. The problems of delivery of contact toxicants to thrips, caused by the thigmotactic nature of thrips, suggest that insecticides with translaminar or systemic movement through plants may be most effective in targeting thrips.

Regardless of the mechanism(s) that lead to resistance, the rapid speed with which resistance can evolve is likely a function of the *r*-selected life history attributes of western flower thrips and its haplo-diploid mode of sex determination.[4] Western flower thrips has a rapid generation time, which allows multiple generations to occur within a single cropping season.[33–35] Females can be long lived,[36] so that generations are overlapping. Such overlap may enable resistant females to mate with their resistant progeny, further reinforcing selection of resistant alleles in the population.[12] The females are highly fecund, which presents two problems for IRM. Firstly, the high fecundity and rapid development rate allow for population outbreaks to occur rapidly. Consequently, growers are likely to apply insecticide treatments in attempts to avoid outbreaks. Secondly, the high reproductive output of western flower thrips provides more opportunities for the production of resistant progeny. In addition, the haplo-diploid reproductive mode of thrips contributes to resistance development because any alleles for resistance in the hemizygous males are directly exposed to selection. Susceptible alleles will not be carried on in males, and consequently resistance alleles will become fixed more rapidly than in organisms with diploid reproduction.[21,37]

3 CROSS-RESISTANCE

Where one resistance mechanism confers resistance to insecticides with different modes of action (cross-resistance), the range of efficacious insecticides becomes more limited for growers to use. In spite of differences in their modes of action, many classes of insecticides are hydrophobic chemicals that can be converted to less harmful hydrophilic compounds by enzyme activity.[38] Because P450s and other detoxification enzymes have a generalized action that converts hydrophobic compounds to less biologically active hydrophilic compounds, insects with an abundance of these enzymes have the potential to detoxify a range of different classes of insecticides, leading to cross-resistance.[16] For example, enhanced activity of P450s has been implicated as the common mechanism of resistance in certain western flower thrips populations to carbamates (IRAC mode of action class 1A) and organophosphates (class 1B), both of which are acetylcholinesterase inhibitors, and to pyrethroids (class 3A), which are sodium channel modulators.[18,39]

In one of the first documented cases of cross-resistance, populations of western flower thrips from Denmark, Kenya and Switzerland had resistance to the carbamate methiocarb, in spite of never having been exposed to that insecticide. These methiocarb-resistant populations had developed high levels of resistance to organophosphates after repeated treatments with acephate and dichlorvos.[40] Although the mechanism(s) of organophosphate resistance were not determined for these populations specifically, the prevalence of enhanced P450-based resistance to these insecticides (Table 1) suggests that as a likely mechanism. In turn, the generalized action of P450s enables the detoxification of carbamates as well. Zhao *et al*.[39] were able to demonstrate this type of cross-resistance in a laboratory population selected for diazinon resistance. The P450-mediated resistance to diazinon conferred differing levels of cross-resistance to bendiocarb and the pyrethroid cypermethrin. Even though levels of resistance to insecticides with different modes of action may not necessarily be of the same magnitude, growers would still have little choice but to avoid use of those materials showing any level of resistance.

Bielza *et al*.[41,42] have proposed taking advantage of the generalized nature of P450 detoxification as a means of restoring or increasing susceptibility of western flower thrips to insecticides that are detoxified by P450s. This could be accomplished by synergizing the insecticides with certain other pesticides that may not be toxic to western flower thrips but are also metabolized by P450s. In this scenario, the toxicity of the primary insecticide would be increased because of competitive inhibition of enzyme substrates from the synergist. They found that mortality from acrinathrin in an acrinathrin-selected resistant population increased from 0.7% in the absence of a synergist to 93.2% in the presence of the carbamate synergist methiocarb, which by itself only induced 6.1% mortality. This use of synergists could then help overcome resistance or slow the rate of its development to a particular insecticide. However, the successful use of this approach necessitates establishing which resistance mechanisms are present to exploit with appropriate synergists.

The unique mode of action of spinosyns appears to have kept cross-resistance from developing between spinosad and other classes of insecticides in field populations to date.[26,27,43–46] Zhang *et al*.[29] selected a laboratory population for spinosad resistance, and this population displayed low to moderate levels of cross-resistance to prothiophos (class 1B), chlorfenapyr (class 13) and thiocyclam (class 14), in spite of the spinosad resistance being based on altered nicotinic acetylcholine receptors. This cross-resistance may indicate the presence of other undetected mechanisms, rather than just altered acetylcholine receptors, that help to confer resistance to spinosad and consequently enable resistance to these other chemistries.

It is clear from experimental evidence that the existence of any one resistance mechanism does not preclude complementary resistance mechanisms to the same insecticide from being present in the same population (Table 1). The presence of multiple resistance mechanisms to particular insecticides likely increases the probability of cross-resistance to unrelated insecticides occurring. Therefore, it is necessary for IRM programs to assess the extent and nature of resistance within western flower thrips populations and then to determine how best to utilize available insecticides within those conditions.

4 PROACTIVE VERSUS REACTIVE INSECTICIDE RESISTANCE MANAGEMENT

The complex nature of insecticide resistance requires the development of strategies to mitigate the development of resistance or reverse resistance. In effect, IRM programs are intended to maximize the lifespan of efficacy for insecticides. Hence, they should characterize which insecticides should be used, when insecticides should be used and in what sequence or pattern, based on attributes of the cropping system and target pests. Ideally, IRM programs would be put into effect as new insecticides were brought into use, and this has become

commonplace today. This proactive approach has the obvious advantage of minimizing the development of resistance from the outset. The need to establish IRM early in the cycle of use of a new insecticide is clearly shown by the rapid development of resistance by western flower thrips to spinosad in southeastern Spain.[26]

The alternative to a proactive approach is a reactive strategy to contain and reverse resistance once it has been encountered. Clearly, resistance management strategies work best before the onset of resistance,[47,48] but reactive strategies are necessary to counter the development of resistance to insecticides that are already in use. However, if resistance has become severe, reactive programs can be problematic to execute successfully with the western flower thrips because there may be minimal fitness costs associated with some forms of resistance. Bielza *et al*.[49] selected laboratory populations for resistance to the pyrethroid acrinathrin and to spinosad and found that females of these resistant strains had greater fecundity than susceptible females. In addition, survivorship, development rates and longevity of progeny from resistant females were as great, or greater, than for progeny of susceptible females. There are two important implications for the lack of fitness costs with resistance. Firstly, the rate of resistance evolution would be greater than if there were fitness costs associated with resistance. Secondly, the lack of fitness costs would increase the durability of resistance, making attempts to revert populations to susceptibility in the absence of the insecticide problematic.[50] The laboratory populations used by Bielza *et al*.[49] were derived from greenhouses in southeastern Spain with a history of intense pyrethroid or spinosad use. Therefore, it is likely that the results of their study reflect a biologically real phenomenon and are not merely laboratory artifacts.

It is critical for IRM programs to be designed to be comprehensive and not focused on individual insecticide products and their targeting against individual pest species. Therefore, overall insecticide use within particular cropping systems should be considered in the development and implementation of IRM programs. Any applications of a particular insecticide contribute to resistance development in a particular pest species, including western flower thrips, whether that pest is the target of a particular application or not.[51] Spinosad and the related spinetoram are among the most efficacious insecticides against western flower thrips.[52] These insecticides also tend to be compatible with natural enemies of thrips, making them highly valuable in IPM programs.[53–55] However, these insecticides are also among the most effective insecticides against other pests, including Lepidoptera, Diptera and Coleoptera,[56] and this widespread efficacy and versatility increases the dangers of resistance development in western flower thrips, as growers rely on it for a range of pest management issues.[57]

As a corollary to this issue of resistance developing from broad-spectrum use of an insecticide, IRM programs should provide growers with knowledge regarding the effect of insecticides on non-target pest populations, such as whether non-targets are resistant to certain insecticides or whether particular insecticides would disrupt natural controls for pests, as this information would mitigate the risks of secondary pest outbreaks.[58] This type of information is especially critical for insecticides such as pyrethroids, to which western flower thrips populations can be highly resistant (Table 1). The use of pyrethroids against other pests typically leads to outbreaks of western flower thrips as resistant individuals survive and their natural enemies are eliminated in agroecosystems.[54]

5 INSECTICIDE RESISTANCE MONITORING

Although numerous cases of insecticide resistance within western flower thrips populations have been documented, it is still necessary to determine whether control failures are a result of actual resistance or some other problem in the field that needs to be addressed. Control failures may result from factors such as inherently high local populations or poor spray coverage, which reduces insecticide efficacy.[45] Alternatively, when resistance is present it is important to assess the extent of resistance.

Monitoring of populations for changes in susceptibility to insecticides can be accomplished through bioassays that challenge target pests with insecticides to determine susceptibility. For example, tomato and pepper growers in northern Florida observed that spinosad failed to control *Frankliniella tritici* (Fitch), a non-pest thrips, whereas spinosad provided good control of western flower thrips. These patterns were also observed in replicated field experiments,[54,59] which would seem to suggest the development of spinosad resistance in *F. tritici*. However, toxicity tests of feral populations showed that spinosad was still highly toxic to feral *F. tritici* and as toxic to it as to western flower thrips.[60] Ramachandran *et al*.[61] resolved the discrepancy by showing that *F. tritici* recolonized treated areas more rapidly than western flower thrips, thus obscuring the toxic effects. In contrast, the same type of toxicity tests revealed that control failures for western flower thrips with spinosad that were first observed in southern Florida in 2006 were actually the result of resistance.[28]

Monitoring for resistance in a timely manner requires well-designed experiments with sufficient numbers of individuals.[48] Ideally, these would be field-collected individuals, so that there are no differences between genotypic profiles of feral and test populations. Care must also be exercised in the selection of susceptible reference populations. The longer that populations are cultured in the laboratory, the greater their genetic differences from feral populations tend to become, which influences the assessment of resistance levels.[48]

Several different techniques for rapid assessment of susceptibility of field populations of thrips to insecticides have been developed. Each bioassay system has its own positive and negative attributes. Initial testing for insecticide resistance in western flower thrips, done by Immaraju *et al*.,[12] involved the use of Munger cells to expose insects to leaves dipped in insecticide solutions. There was the possibility that thrips could avoid contact with the residues because only the leaf surface was treated, but the cell walls remained untreated. Brødsgaard[40] used a Potter spray tower uniformly to treat the top and bottom surfaces of a glass apparatus similar to a Munger cell. However, the side walls of the cell were swabbed with the insecticide solution to coat the arena completely, which may have led to variations in the amounts of residues across the arena.[13] Herron *et al*.[13] proposed that field conditions could be closely approximated by using a Potter spray tower to treat leaf discs and thrips in arenas simultaneously. However, direct exposure of western flower thrips to insecticide sprays in the field may be limited because of their thigmotactic behavior.[32] A simpler alternative bioassay involves exposing thrips to insecticide-treated bean pods placed inside small plastic cups.[60] The authors argued that this is a robust bioassay because thrips will spend almost all of their time on the bean pods. This bioassay has the additional advantages of enabling rapid testing of field-collected individuals with minimal equipment requirements. Rueda and Shelton[62] developed their thrips insecticide bioassay system (TIBS) to monitor resistance in *Thrips tabaci* Lindeman. With this system, thrips are aspirated into microcentrifuge tubes that

have been coated previously with contact insecticides. Mortality can be determined rapidly, simply by visual inspection of thrips within each tube. Bioassays that incorporate treated plant material for an exposure substrate are likely to be the most versatile for testing chemistries with differing modes of action and are the ones that provide the closest approximation to the manner in which western flower thrips are exposed to insecticides in the field. Still, no one single bioassay will meet every research need, and investigators should be aware of the benefits and drawbacks of different bioassays.[63]

6 INSECTICIDE RESISTANCE MANAGEMENT PROGRAMS

The fundamental goal of IRM is to reduce the frequency of resistance alleles within a population so that the efficacy of a particular insecticide can be preserved.[64] Theoretically, resistance could be suppressed by overpowering resistant genotypes with sufficiently high concentrations of a particular insecticide.[65] This type of tactic would only succeed if resistance alleles were extremely rare within a population. Such a tactic would seem risky for western flower thrips management, given its propensity to evolve resistance, the likelihood that invasive populations carry resistance traits and the potential for cross-resistance. Therefore, the overarching key to minimizing the rate of resistance development is not to overuse any particular insecticide or chemical class of insecticides, and thereby to preserve susceptibility to those insecticides. Consequently, this has led to the most common resistance management tactic of employing rotations among different classes of insecticides. A critical development in the application rotational schemes for IRM is the IRAC Mode of Action code, whereby insecticides are classified according to their chemistry and mode of action and/or target site (see http://www.irac-online.org/). IRAC is an insecticide industry association that promotes 'prolonging the effectiveness of insecticides and acaricides by countering resistance problems' (http://www.irac-online.org/about/irac/). This clearly benefits insecticide producers by protecting their investment in product development, but it also aids growers in maintaining the availability of efficacious insecticides. Mode of action codes enable growers easily to employ rotation schemes for insecticides, once practical ones are developed.

Modeling studies have shown that there is no theoretical advantage to using rotations of multiple insecticides over using one particular insecticide sequentially until it is no longer efficacious in terms of resistance development.[66] However, this has not always been borne out in experimental testing for certain pests, including *Scirtothrips citri* (Moulton).[67] The development of resistance to spinosad observed in Spain and Florida also cautions against sequential product use[26,28] because resistance is likely to develop before new replacement products are available. Resistance management has been termed a 'red queen' race where agriculturists need to 'run' as fast as possible to develop new insecticides to replace those to which resistance has evolved.[68] Early in the synthetic insecticide era, resistance could be combated by the introduction of new chemistries, but the rate of new compound discovery and registration has slowed considerably since the 1970s.[57,69] Given the constraints of developing and registering new materials for commercial use, one long-term goal of IRM can best be viewed as slowing down the rate of resistance development below the rate of discovery and registration of new insecticides.[68] While not necessarily the optimal approach based on theoretical modeling, insecticide rotations are the most practical tactic to help preserve insecticide efficacy. Even in the absence of information on insecticide resistance mechanisms, rotations would still produce benefits for growers over not having an IRM program and placing an overreliance on a single insecticide.[70]

Broadbent and Pree[71] first proposed using rotations against western flower thrips. Their plan called for rotating chemistries after every generation of western flower thrips. Under greenhouse conditions, this would allow for a single chemical class to be used for approximately 2 weeks before switching to a different chemical class. In a similar approach, Herron and Cook[72] advocated a 'three-spray' strategy in which growers were encouraged to apply a single insecticide in three consecutive sprays within a single thrips generation and then rotate to an insecticide from a different chemical class for the next three spray applications. The rationale for this approach is that the intense use of one insecticide within one pest generation would suppress the population, and the selection pressure would be counteracted in the next generation by the use of an alternative chemistry. Robb and Parella[73] proposed a more extensive 4–6 week interval between rotations of chemicals with different modes of action. This interval would allow at least 2–3 generations of the pest to be treated with the same class of insecticide, which theoretically could lead to positive selection for resistance.

An alternative rotation interval has been proposed for fruiting vegetables in Florida, with no more than two consecutive applications of a particular insecticide being made before rotating to a different chemistry.[74,75] Again, an advantage of fewer consecutive applications of the same material is a reduced selection potential. Because no other insecticides provide the efficacy against western flower thrips of spinosyn (class 5) insecticides, growers are encouraged selectively to use spinosyns and rely on less efficacious materials when western flower thrips pressures are low. An additional focus of this plan for pepper production is to use insecticides for western flower thrips or other pests that do not disrupt populations of *Orius insidiosus* (Say), a key biological control agent of western flower thrips.[54,58] Most importantly, growers are encouraged to anticipate pest problems with western flower thrips and to use preventive tactics to help suppress western flower thrips populations.[58]

Because of cross-resistance, Bielza[21] advocated that insecticide rotations be a component of IRM programs, but that these rotations be based on potential mechanisms of resistance rather than strictly based on mode of action classes and the assumption of independent resistance mechanisms. While it is clearly beneficial to base insecticide rotation plans on potential mechanisms of resistance, as discussed above, it may not be possible to predict mechanisms because of the plasticity of resistance evolution across populations. Populations may evolve different mechanisms for resistance to the same insecticide.[20,76] Even in the absence of information on insecticide resistance mechanisms, rotations would still produce benefits for growers over not having an IRM program and placing an overreliance on a single insecticide.[70] Although these rotational plans, whether based on mode of action or mechanism, will reduce the rate of resistance evolution to insecticides, these rotation schemes do not necessarily focus on reducing overall insecticide use in cropping systems. If overall insecticide use then remains high in a cropping system with a simple rotation plan, there will likely be an advance in overall selection for resistance, regardless of the frequency with which insecticide classes are rotated.

Therefore, to improve IRM further, rotation schemes need to be complemented with other tactics that would reduce overall insecticide inputs. One such tactic for more judicious use of insecticides

is to establish realistic economic thresholds for the western flower thrips that growers can easily use. Economic thresholds have been established in some cropping systems but not in many others, in particular in high-value ornamental crops that can tolerate little aesthetic damage or in crops where the threat of Tospovirus transmission is high. In both of these situations, the perceived risks from western flower thrips damage tends to induce growers to have near-zero tolerances for western flower thrips. Unfortunately, intense insecticide use at exceedingly low pest levels often exacerbates problems with insecticide resistance[28] and creating more crop damage in the long run. Realistic damage assessments for western flower thrips have only recently begun to be developed, and, to date, few economic threshold levels have been established for western flower thrips in most cropping systems.

Shipp *et al*.[77,78] developed economic injury levels (EILs), not economic thresholds, for greenhouse-grown cucumbers and peppers in Canada where TSWV has not been considered a threat. In these cropping systems, direct feeding on fruit is the most significant form of damage, but yield reductions could also occur with feeding damage to foliage. Therefore, dynamic economic injury levels were set to account for changes in damage potential according to crop stage, with lower levels during the fruiting stages. They proposed the monitoring of populations with sticky cards and by sampling flowers for adults. Welter *et al*.[79] proposed significantly higher levels for western flower thrips and *Thrips palmi* Karny infesting field-grown cucumbers in Hawaii. They based their thresholds on numbers of thrips on leaves. The higher levels for field-grown cucumbers versus greenhouse-grown ones are based on the greater tolerance of field-grown crops to foliar feeding damage.

Economic thresholds have been developed for fruiting vegetables, including tomato, pepper and eggplant, in Florida.[74,80] Thresholds are based on the numbers of adults collected in flowers and samples, and larvae collected in samples of fruit. In northern Florida, TSWV transmission is the most significant damage caused by western flower thrips, and insecticides alone are ineffective in stopping primary disease spread. Therefore, thresholds are aimed at limiting the aesthetic damage caused by western flower thrips oviposition and feeding damage.[58] Most importantly, these thresholds depend on identification of the thrips species. The more prevalent native species *Frankliniella bispinosa* (Morgan) and *F. tritici* do not cause the damage that western flower thrips do. Therefore, to use the thresholds effectively, thrips species must be identified to distinguish between pest and non-pest species.

A key feature of these threshold systems is that they are based on relatively simple, inexpensive and user-friendly sampling protocols that growers can readily employ.[78] Funderburk[58] recommends flower samples collected in alcohol, which facilitates microscopic examination of specimens for species identification. Many research studies and commercial scouting operations employ yellow or blue sticky traps for estimating western flower thrips abundance. Heinz *et al*.[81] proposed a time-efficient method for counting thrips and other insects on sticky cards, in which only border sections of cards need be examined because that is where insects tend to aggregate. Although colored sticky traps are relatively easy to use for monitoring, estimates from sticky traps do not correlate with thrips abundance on plants.[82,83] Given this discrepancy, care should be exercised in interpreting population estimates from sticky traps alone.

Further reductions in insecticide use can be realized if growers employ preventive pest management tactics. Such tactics will improve IRM and result in more economical crop production. For field-grown vegetable crops, preventive tactics to reduce western flower thrips abundance and Tospovirus incidence include ultraviolet reflective mulches to reduce thrips entry into crop fields.[54,59] Several cultivars of pepper and tomato possess resistance to tomato spotted wilt virus.[8] Cultural controls available for greenhouse situations include ultraviolet absorbing coverings for greenhouses[84,85] and screening of ventilation inlets.[86] Altering environmental conditions, especially between crop cycles, in greenhouses can also be used to reduce western flower thrips populations.[87]

The use of biological control is a viable approach in many cropping systems to reducing overall western flower thrips populations, and it is already a mainstay of thrips IPM in certain systems. In greenhouse systems, augmentation biological control through the release of predatory insects and mites has been very successful, and biological control has been adopted by a high proportion of growers throughout Europe, in particular, because of the advantages it offers growers over alternative pest control tactics.[88,89] A major concern for growers in the maintenance of biological control efforts is conservation of natural enemies. Many insecticides used against western flower thrips or other pests are toxic to natural enemies, so that biological control can be disrupted by poor selection of insecticides or poor timing of applications.[21] In the southern United States, conservation biological control is the cornerstone of western flower thrips management in field-grown pepper. Naturally occurring populations of *O. insidiosus* and competing thrips species suppress western flower thrips populations and the incidence of tomato spotted wilt. However, injudicious use of insecticides, in particular pyrethroids, can release western flower thrips from these natural controls, leading to substantial crop losses.[54,58,80] Therefore, IRM and overall pest management can be enhanced by maximizing the use of biological control and minimizing insecticide use.

One further aspect of improving IRM and overall pest management is education. As reviewed here and elsewhere, a number of effective IRM tactics and overall IPM tactics for western flower thrips have been developed in numerous cropping systems around the world. However, concerted efforts need to be made to educate growers and other clientele about these tactics and how to employ them successfully.[90] Legislating restrictions on pesticide use can often have damaging unintended consequences.[91] The number of insecticide applications that are needed effectively to manage a pest must be balanced against the maximum number of applications that can be made without triggering the rapid evolution of resistance. By providing growers with an education and training framework, growers will be able to understand the implications of various pest management tactics for overall crop health and production.[46] Further, if growers are presented with estimates of the economic consequences of management decisions, they will be more likely to adopt novel pest management tactics. Reitz *et al*.[92] used research trials and commercial demonstration trials to validate that low-pesticide-input programs had economic returns as great as or greater than more pesticide-intensive programs. Growers were more willing to adopt the new programs once they were able to see the economic improvements that the new programs could offer.

7 CONCLUSIONS

Developing and implementing IRM programs for pests such as the western flower thrips clearly is a complex task. The history of the western flower thrips as a crop pest has shown that insecticide resistance can evolve readily, and likely will do so

without proper management. To help minimize the evolution of resistance and the inherent problems in trying to revert populations to susceptibility once resistance has developed, there must continue to be a concerted movement to develop preventive IRM programs for new insecticide chemistries.[93] There is also a need to develop IRM programs to mitigate resistance development for older chemistries that may be used in areas that western flower thrips has recently invaded.[5] Wherever western flower thrips is a pest, IRM programs should be implemented. Waiting to implement IRM until there is sufficient information collected to assess the resistance status within a particular western flower thrips population is not acceptable. Pragmatic programs based on relatively little information can still be valuable,[70] and these can be refined as more information becomes available.[21,94] One of the most critical pieces of information would be ongoing assessments of the resistance status to insecticides used within particular cropping systems, including determination of potential resistance mechanisms. Although long-term resistance monitoring has been practiced in some areas,[13,28] increasing the scope of it geographically and for more insecticides would enhance overall resistance management and the refinement of IRM programs. In addition, it would be desirable to have a broader range of highly effective insecticides to target the western flower thrips to complement the spinosyn insecticides, but the development of such materials cannot be predicted. However, effective insecticide rotation plans have been developed to maximize the effectiveness of spinosyns, with other less effective materials used at appropriate times. These rotation schemes can be complemented with the development of realistic economic thresholds for more crops to help reduce overall insecticide inputs into cropping systems.

In theory, the development of IRM for a single pest is relatively 'easy'. However, the implementation of such a program is complicated by the fact that few crops are affected by a single pest. Most crops are affected by multiple pest species, and growers must contend with each of these effectively. Therefore, growers are faced with a number of decisions that affect their crop management and productivity. To accommodate the complex management decisions with which growers must contend, there is a critical need for overall management programs that integrate management tactics for all pests of concern within a cropping system.[58] These concerns reinforce the need to view IRM as a component of overall IPM programs and not to view resistance management as a goal in itself.

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