Grain storage managers implement an integrated approach to controlling insect pests by using a range of tactics such as sanitation, cooling, drying, and grain cleaning. Chemical treatments, including fumigants and residual insecticides, remain the most effective tools for controlling insect pests and are key elements in integrated approaches. These tools enable grain storage managers to maintain food security, access markets, implement effective quarantine systems, protect the supply chain, and provide consumers with high quality food.

Health, safety, environmental, and economic considerations severely limit the range of chemicals that can be applied to grain. In recent years authorities around the world have reduced the number of chemicals available. Chemicals that can be applied to grain are rare and costly to develop.

In addition to these pressures, insects targeted by the chemicals are rapidly developing resistance to the few alternatives available. The remaining chemicals must be managed carefully to ensure effective grain protection now and in the future.

This chapter briefly summarizes our knowledge of the status of resistance to grain protection chemicals in stored-product insect pests. It describes factors that influence rate of resistance development or selection, including genetics, mechanisms of resistance, gene flow, relative dominance, fitness, and the effects of human activities. The chapter concludes with a discussion of how resistance management tactics can be applied to a real-world situation to show the challenges of managing resistance in stored-product systems.

**Resistance Management in Stored Product Insect Pests**

Pesticide resistance is an increased tolerance to a pesticide that has a genetic basis. As a heritable trait, the development and spread of resistance will be influenced by the selective pressures of pesticide use, the mode of inheritance, fitness costs associated with individuals carrying resistance genes, and movement of pests on geographical scales. Insecticides from a range of chemical groups and several fumigant gases have been used to control insect pests of stored products. In most cases, at least one major pest species has developed resistance to these compounds somewhere in the world, and that same resistance often develops in different parts of the world. Resistance development patterns in stored product insects from one country show potential for resistance development in other countries.

Insecticides have been used mainly as grain treatments (disinfestants and grain protectants); surface treatments for bag stacks, floors, and storage structure walls; and aerosol treatments. Since the mid-20th century these insecticides have been drawn mainly from the organophosphates (OPs) (malathion, chlorpyrifos-methyl, and dichlorvos), the pyrethroids (bioresmethrin, deltamethrin, and beta-cyfluthrin), and from the juvenile hormone analogues (JHAs) (methoprene and hydroprene).
The history of resistance development to insecticides from all of these groups has been well documented in Australia and demonstrates both the propensity of stored products insects to develop resistance and the potential for resistance to develop elsewhere in the future (Table 1). The lesser grain borer, *Rhyzopertha dominica* (F.), is of particular concern given that it is a major pest of stored products and clearly has the potential to develop resistance to OPs, pyrethroids, and JHAs.

Parallel development of insecticide resistances in different countries is well illustrated in the scientific literature. Champ and Dyte (1976) reported that malathion resistance was present in many countries around the world, and resistance to newer insecticides has since been reported from a range of countries as the following examples show. OP-resistant *R. dominica* have been reported from Australia, the United States, and Brazil (Bengston et al. 1975, Zettler and Cuperus 1990, Guedes et al. 1996). Similarly, pyrethroid-resistant maize weevils, *Sitophilus zeamais* Motsch., and *R. dominica* have been reported from Australia and Brazil (Samson et al. 1990, Collins et al. 1993, Guedes et al. 1994, Lorini and Galley 1999).

The principal fumigants used in stored product protection have been phosphine and methyl bromide. Resistance has been detected predominantly in phosphine, with examples from many species from many countries since the 1970s. One key feature of fumigation is that concentration and exposure period can both be altered to maximize fumigant efficacy. This has implications for the detection, measurement, and impact of phosphine resistance (e.g., Collins et al. 2005), with insects carrying resistance genes often controllable in practice.

The global survey of Champ and Dyte (1976) showed that phosphine resistance has been present in many countries for several decades. Subsequent published surveys focusing on specific geographic regions have further demonstrated the extent of phosphine resistance (e.g., Attia and Greening 1981, Zettler et al. 1989, Herron 1990, Zettler and Cuperus 1990, Benhalima et al. 2004).

Different levels of phosphine resistance can occur within a species. In the case of *R. dominica*, for example, at least two levels of resistance appear to exist: weak resistance, with resistant adults about 30 times more resistant than susceptibles when fumigated for 48 hours; and strong resistance, with resistant adults hundreds of times more resistant than susceptible insects (Collins et al. 2002, Lorini et al. 2007). Similarly, at least two levels of phosphine resistance have been reported for *S. oryzae* and *Tribolium castaneum* (Herbst) (Daglish et al. 2002, Jagadeesan 2011). As with resistance to other insecticides, phosphine resistance trends in one country show the potential for resistance development in other countries. The presence of strongly resistant insects in countries such as Australia, Brazil, and the Philippines should be of concern to countries that do not yet have strong resistance. Also, the prevalence of strongly resistant *R. dominica* in Brazil is a warning to countries where strong resistance is rare or has not been detected.

Although biologically derived insecticides have seen some use in agriculture, this has not been the case for stored-product protection. This does not preclude their future use and the potential for insects to develop resistance to these biopesticides should they be adopted. The potential of stored product insects to develop resistance to biopesticides is well illustrated by a study that demonstrated that native populations of the Indianmeal moth, *Plodia interpunctella* (Hubner), could develop resistance to the bacterium *Bacillus thuringiensis* within a few generations (McGaughey 1985). Spinosad is a bacterium-derived biopesticide that has been registered as a grain protectant and is likely to be widely used (Hertlein et al. 2011). Although there is no evidence of the potential of stored product insects to develop resistance to this biopesticide, resistance has developed in other agricultural pests (e.g. Moulton et al. 2000).

**Cross-Resistance and Multiple Resistance**

Cross-resistance is when resistance to a given pesticide causes resistance to another pesticide without the insect having been exposed to the latter pesticide (Scott 1990). For example, *R. dominica* that are resistant to one organophosphate have a tendency to be resistant to other organophosphates. A similar situation occurs with pyrethroid resistant *T. castaneum* and *R. dominica* (Collins 1990, Guedes et al. 1996, Daglish et al. 2003). In the application of pesticides for the control of stored-product insect pests, avoiding the use of pesticides that share cross-resistance is important. Failure to do so hastens the development of resistance.
When an arthropod has more than one mechanism of resistance, it is said to have multiple resistance (Georghiou 1965). For example, certain resistant strains of *P. interpunctella* are resistant to *B. thuringiensis* by altering the target site on which the toxin of this bacterium binds and reducing the number of target sites available (Herrero et al. 2001).

### Mechanisms of Resistance

Four main mechanisms insects can use for resistance to pesticides are described below (Soderlund and Bloomquist 1990, Mota-Sanchez et al. 2002).

**Metabolic resistance** – Insects can develop an increased ability to detoxify and/or metabolize (breakdown) a pesticide by producing higher amounts of enzymes. Enzymes usually used to break down insecticides are cytochrome P450-dependent monoxygenases, hydrolases, or glutathione-$\delta$-transferases. This type of resistance is called metabolic resistance, and it is the most common mechanism of resistance. For example, higher levels of glutathione-$\delta$-transferase have been found in resistant strains of *T. castaneum* (Cohen 1986).

**Target site resistance** – Pesticides work by attaching themselves to target sites. Unless the pesticide molecules attach to these target sites, insects are not affected or killed. Some insects resist pesticides by having genetically altered target sites so that pesticide molecules are unable to attach to them, rendering the pesticides ineffective. This mechanism of resistance is called target site insensitivity. For example, one way *P. interpunctella* is resistant to

### Table 1. Examples of field-derived insecticide resistances detected in Australian stored-product beetles.

<table>
<thead>
<tr>
<th>Type</th>
<th>Species</th>
<th>Insecticide tested</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Benzene hexachlorides (BHCs)</td>
<td><em>Sitophilus oryzae</em> and <em>S. zeamais</em></td>
<td>Lindane$^*$</td>
<td>Champ and Cribb (1965)</td>
</tr>
<tr>
<td></td>
<td><em>Tribolium castaneum</em></td>
<td>Lindane$^*$</td>
<td>Champ and Campbell-Brown (1969)</td>
</tr>
<tr>
<td>Organophosphorus compounds (OPs)</td>
<td><em>Rhyzopertha dominica</em></td>
<td>Malathion$^*$</td>
<td>Greening et al. (1975)</td>
</tr>
<tr>
<td></td>
<td><em>Oryzaephilus surinamensis</em></td>
<td>Fenitrothion$^*$</td>
<td>Collins (1985)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Chlorpyrifos-methyl$^*$</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Pirimiphos-methyl$^*$</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Deltamethrin$^*$</td>
<td></td>
</tr>
<tr>
<td>Pyrethroids</td>
<td><em>T. castaneum</em></td>
<td>Bioresmethrin$^*$</td>
<td>Collins (1990)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cyfluthrin</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Cyhalothrin</td>
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<tr>
<td></td>
<td></td>
<td>Cypermethrin</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Deltamethrin</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Permethrin</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>d-Phenothrin</td>
<td></td>
</tr>
<tr>
<td></td>
<td><em>S. zeamais</em></td>
<td>Deltamethrin</td>
<td>Samson et al. (1990)</td>
</tr>
<tr>
<td></td>
<td><em>R. dominica</em></td>
<td>Bioresmethrin$^*$</td>
<td>Collins et al. (1993)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Bifenthrin</td>
<td>Daglish et al. (2003)</td>
</tr>
<tr>
<td>Juvenile hormone analogues (JHAs)</td>
<td><em>R. dominica</em></td>
<td>Methoprene$^*$</td>
<td>Collins (1998a)</td>
</tr>
</tbody>
</table>

$^*$In commercial use at the time of the cited study.
B. thuringiensis is by target site alteration to the toxin of this bacterium (Herrero et al. 2001).

**Penetration resistance** – Insects have a hard material called the cuticle covering the surface of their bodies. Pesticides that kill insects by contact must penetrate the cuticle and get inside the insect. Some insects have developed barriers against pesticides and can slow the absorption of chemicals into their bodies. This mechanism of resistance is referred to as penetration resistance, and it is not pesticide-specific. When penetration resistance is present alone, it confers weak resistance. For example, resistance to pirimiphos-methyl in certain strains of T. castaneum is by reduced penetration through the cuticle (Walter and Price 1989).

**Behavioral resistance** – In some cases the behavior of insects results in reduced exposure to pesticides. For example, Guedes et al. (2009) found higher rates of flight take-off in a resistant strain of S. zeamais exposed to surfaces treated with deltamethrin. Presumably this behavior has been selected to increase the insect’s chance of survival by reducing the amount of time it spends on treated surfaces.

**Genetics and Ecology of Resistance**

Effective management of resistance requires an understanding of its causative processes. Insecticides act on genotypic variation (mutation, recombination, gene flow) to select for resistant phenotypes. How the selection process operates is determined by the population genetics and ecology of the organism in relation to its environment, including human activity. An understanding of factors such as the inheritance and relative dominance of resistance genes, relative fitness of genotypes in the presence and absence of insecticides, insect movement and mating systems, and human impacts is essential for sustainable resistance management.

Where investigated, insecticide resistance in insect pests of stored products has most often been attributed to a single autosomal gene. For example, in T. castaneum, DDT (Erdman 1970) and lindane/cyocidiene resistance (Beeman and Stuart 1990) are each mediated by a single autosomal gene, and resistance to the organophosphate malathion is also associated with a single gene but is multi-allelic, with alleles for “specific” (carboxylesterase) and “non-specific” resistance (and susceptibility) occurring at the same locus (Beeman 1983, Beeman and Nanis 1986). Single genes are also responsible for resistance to malathion, lindane, and dieldrin in Plodia interpunctella (Attia et al. 1981, Beeman et al. 1982) and DDT/pyrethroid sex-linked resistance in S. oryzae (Champ 1967, Heather 1985).

Multi-gene resistance also occurs. At least two major genes control resistance to organophosphates in O. surinamensis (Collins 1986), pyrethroids in T. castaneum (Collins 1998b, Stuart et al. 1998), and high phosphine resistance in T. castaneum (Jagadeesan 2011) and R. dominica (Collins et al. 2002). Further detailed genetic and molecular analysis of R. dominica (Schlipalius et al. 2002, 2008a) revealed the presence of two loci, rph1 and rph2, responsible for phosphine resistance in this insect. Rph1 controls the “weak” resistance phenotype by providing moderate resistance to phosphine, whereas rph2 by itself confers only very low-level resistance. Rph2 was not discovered in the field until rph1 had become common. When combined in the same individual, mechanisms controlled by rph1 and rph2 synergize to produce a much higher level of resistance known as the “strong” resistance phenotype. Mau (2008) compared the genetics of phosphine resistance in strongly resistant R. dominica strains from three widely separated locations in Australia, and concluded that resistance in each strain was derived independently from others despite genetic analysis being consistent, with two major genes being responsible for resistance in each case.

How genes are expressed in the phenotype is known as dominance. When a pair of alleles is required to express resistance in the phenotype, the allele is a recessive factor. When an allele can phenotypically express itself in the heterozygote as well as the homozygote, it is referred to as a dominant factor. It is important to understand that dominance is not fixed and is dependent on the environment in which it is expressed or how it is measured. For example, resistant homozygotes and heterozygotes may survive a certain insecticide dose, making resistance dominant, but at a higher dose only the resistant homozygotes may survive, making the resistance recessive.

Most knowledge of the dominance of resistance in insect pests of stored products is derived from classical analyses of the inheritance of resistance. Very few resistances are expressed as either fully dominant
or recessive. Most are intermediate, i.e., partially expressed in the heterozygote. For example, resistance to insecticides such as malathion (carboxylesterase) (Beeman 1983), fenitrothion (Collins 1986), pyrethrins (Prickett 1980), and pyrethroids (Collins 1988b, Stuart et al. 1998, Heather 1985) are often semi- or incompletely dominant, whereas resistance to phosphine is incompletely recessive (Bengston et al. 1999, Collins et al. 2002, Daglish 2004).

The bioassay methods used in these analyses, such as exposing insects to insecticide-impregnated papers or to very short exposures of fumigant (FAO 1974; 1975), are intended for rapid diagnosis of resistance but, because they do not reflect field application of chemicals, have limited relevance to resistance management. On the other hand, some analyses (Collins 1986, 1998b) used bioassays that mimicked use of insecticide so that conclusions about the effect of a range of doses on the dominance of phenotypes can be made. This may not be an issue with phosphine, as it has been shown (Daglish 2004) that degree of dominance (and resistance factor) of phosphine resistance in R. dominica and S. oryzae adults was constant over a range of exposure periods up to 144 hours. Whether this finding holds true for longer exposure periods is not known. A second potential problem with laboratory bioassays is that they are overwhelmingly carried out on adult insects and there is a general assumption that dominance will be the same for other life stages. This is not necessarily the case as it has been shown that both relative tolerance and relative dominance vary with life stage in T. castaneum (Collins et al. 1997).

Insects possessing resistance genes are often assumed to suffer a fitness cost (i.e., lowered reproductive success), which explains the initial absence or rareness of the resistance. From a resistance management perspective, a fitness cost would mean that the frequency of resistance would decrease during periods when the pesticide is not used. Despite the development of resistance to phosphine and a range of insecticides in stored product insects, relatively few studies have investigated potential fitness costs associated with these resistances. These studies have variously concluded that there is no fitness cost, there is a fitness cost, or there is even a fitness advantage. Heather (1982) compared the population growth rates of malathion-resistant and susceptible S. oryzae and overall found that resistant populations were no less or more fit than susceptible populations, and nor were population crosses between resistant and susceptible populations. In contrast, Arnaud et al. (2002) reported that malathion-resistant T. castaneum had a higher fecundity and were therefore more fit than susceptible insects. Schlipalius et al. (2008a) concluded that strongly phosphine-resistant R. dominica suffer no fitness disadvantage, after a population of resistant–susceptible cross was reared in the absence of phosphine selection, and the frequencies of resistant, susceptible, and heterozygote individuals determined after 5, 15, and 20 generations. Using a similar approach Jagadeesan (2011) concluded that strong resistance in T. castaneum came with a fitness cost, but weak resistance did not. Several studies in which various physiological or ecological parameters were compared in resistant and susceptible populations have demonstrated fitness costs to insecticide- or phosphine-resistance in various stored product pests (Pimental et al. 2007; Sousa et al. 2009). Clearly, no general conclusions can be drawn about the fitness of resistant stored product insects, and so studies on specific species and resistances are needed.

The fact that studies using different approaches can support contradictory conclusions raises the possibility that expression of fitness in laboratory studies is so situation-specific that different approaches will often lead to different conclusions. Using more than one approach in fitness studies may be advisable to maximize the likelihood of obtaining information that is useful for resistance management.

Understanding genetic structure of populations and gene flow in stored product pests may provide insights into the development and spread of resistance, and the scale on which resistance management should be applied. Studies like these must rely on molecular tools such as resistance markers and neutral DNA markers. No information is available on the frequency of pesticide resistance genes in wild stored product insects, although the discovery of the molecular basis for the inheritance of phosphine resistance in several organisms raises the possibility of resistance markers being developed for phosphine resistance (Schlipalius et al., 2008a; Jagadeesan 2011). Several studies have investigated the levels of genetic differentiation in T. castaneum using neutral DNA markers. Drury et al. (2009) reported relatively low levels globally indicating considerable gene flow, as did Semeao et al. (2010) for the United States and Puerto Rico. Although anthropogenic movement is
likely to contribute to gene flow, Ridley et al. (2011) showed that dispersal through flight is important for this species at least on a district scale. No information is available on population structure and gene flow in *R. dominica*, but Mau (2008) showed that strong phosphine resistance evolved independently in Australian populations from three widely separated geographical origins. The lack of information on population structure and gene flow in stored product pests represents an impediment to understanding how resistance develops and spreads and how it should be managed.

**Resistance Monitoring and Detection**

Resistance monitoring is undertaken for a number of reasons including early warning of resistance, feedback on the success of management activities, diagnosis of control failures, and information on the likely impact of new resistance. Reliable methods of detecting and measuring resistance and an understanding of how results relate to control failures are the foundation of an effective resistance-monitoring program.

The most common method of testing for resistance is to expose the insect to the toxicant and observe and quantify the response, known as bioassay. Standard bioassay methods have been published for testing for resistance to the grain protectants malathion and lindane (FAO, 1974, Busvine 1980) and the fumigants methyl bromide and phosphine (FAO, 1975) in a number of stored product pest species. These methods are based on exposure of adult insects to a “diagnostic concentration” of chemical for relatively short periods of time, 5 to 6 hours and 20 hours, respectively.

The grain-protectant test was designed to provide a result on the day of testing. Diagnostic or discriminating concentrations are developed from the responses of “susceptible” or wild-type strains of insects believed to represent the insect genotype before any selection with the chemical had occurred. The diagnostic dose is usually a single dose used to separate putative resistant from susceptible insects. Choice of diagnostic doses requires careful consideration of the range of responses of populations of target insects and analysis of their response data. Second-level diagnostic doses have been developed in situations where higher-level resistance (a second mechanism) is suspected, or where current resistance levels are too weak to challenge field control (Daglish and Collins 1999). A detailed discussion of bioassay and the statistical analysis of response data is beyond the scope of this chapter. The reader is referred to Robertson et al. (2007) and Stanley (2008) as starting points.

The FAO-published methods provide an international standard that can be used to alert researchers to the presence of resistance in an insect population. They do not reflect how chemicals are used by industry, so they give no indication of the impact of any given resistance on control in the field. For example, the protectant assay exposes insects to chemical impregnated into a filter paper, whereas grain protectants are applied as liquids to grain or industrial surfaces of various types and are expected to remain active for several months.

The phosphine exposure assay is short compared with industry practice of about 5- to more than 20-day fumigations. Resistance tests typically expose only adults, while the treatment is usually aimed at controlling all life stages. (An obvious exception is that treated grain assays must be used to test for resistance to juvenile hormone analogues [e.g. methoprene] because these protectants affect the immature stages and cause negligible parental mortality). For these reasons, other assays that better model field uses have been developed (Collins 1990, Daglish and Collins 1999, Collins et al. 2005). These assays are particularly important in the confirmation and characterization of resistance.

Sampling strategy (reviewed by Venette et al. 2002) should be considered carefully before undertaking a monitoring program. In the early stages of resistance development, resistance gene frequencies are relatively low, and homozygote-resistant insects will be virtually absent in the population (Mackenzie 1996). Thus, the probability of detection of resistance genes will be low (Roush and Miller 1986). If the primary aim of monitoring is discovery of new resistance, then a strategy, such as F2 screen, that maximizes the likelihood of detection could be used (Andow and Onstad 1998). If the primary aim of monitoring is discovery of new resistance, then a strategy, such as F2 screen, that maximizes the likelihood of detection could be used (Andow and Onstad 1998). In later stages of resistance development, when gene frequencies are relatively high, the primary aim of monitoring may be to provide information to a management strategy. In this case, a sampling and detection strategy that provides rapid diagnosis may be more appropriate.
Detection is also influenced by the relative dominance of resistance genes. Most resistance in stored-products insects is semi- or incompletely dominant (Prickett 1980, Stuart et al. 1998, Beeman 1983, Heather 1985, Collins 1986, 1988a) or close to recessive (Bengston et al. 1999, Collins et al. 2002, Daglish 2004) so that the overlap of responses between susceptible and heterozygous genotypes further diminishes the sensitivity of the bioassay method.

A potential solution to the two major drawbacks of traditional bioassay — long response time and low sensitivity — is the development of either biochemical or molecular testing methods. A PCR (polymerase chain reaction) diagnostic has been developed for cyclodiene resistance in *T. castaneum* (Andreev et al. 1994) and genomic methods have been used to identify the major genes responsible for phosphine resistance in *R. dominica* and *T. castaneum* (Schlipalius et al. 2008a, Jagadeesan 2011). The advantages of these techniques are that they can identify resistance in heterozygotes, live or dead, they avoid the need for culturing insects and they provide accurate unambiguous results in less than a day at a reasonable cost (Schlipalius et al. 2008b). The major disadvantage is that this type of test can detect only known resistance genes.

In conclusion, resistance monitoring is an important part of keeping the proportion of susceptible organisms in a population as large as possible. It enables the assessment of pest population status, understanding of potential risks, evaluation of whether a resistance management program is achieving its goals, and the prediction of future trends (Stanley 2008).

**Resistance Management Principles**

Pesticide resistance management is a strategy for applying any pesticide or pesticide class as infrequently as possible to delay the development of resistance to it. Resistance management expects resistance to develop and acts to mitigate the rate at which it develops. This section presents information on possible ways of maintaining the pesticide susceptibility of stored-product insect pests.

A practical resistance-management strategy relies on three major components.

**Information about the system** – Information is required on the state and condition of grain and grain storages in the system and on the occurrence of insect infestation. In addition, there must be information on strengths and frequencies of resistance in insect pest populations. The latter provides early warning of the emergence of new resistances and the occurrence of known resistance. This allows researchers and industry time to assess the situation, avoid control failures, and implement remedial action. Accurate, detailed information permits effective planning and provides feedback on the success of resistance-management tactics.

**Tactics that reduce the rate of selection** – Tactics that reduce the rate of selection are likely to be the most successful in the long term. This can be achieved by reducing the frequency of use of the selecting agent, by reducing the number of insects exposed to the selecting agent, and by maintaining sources of susceptible genes. For example, cooling grain reduces insect population growth, reducing the need to fumigate. Chemical and physical hygiene treatments reduce population numbers, decreasing the number of insects potentially exposed to the selecting agent. The existence of untreated refuges maintains sources of susceptible genes.

**Tactics that destroy resistant insects** – In a situation where resistance has already evolved, tactics that destroy resistant insects are essential for practical resistance management. These can be either higher doses of the current material (e.g. phosphine), alternative chemicals, or physical methods such as heat disinfection. These tactics are used to eliminate resistance foci, that is, instances where resistance has been detected (resistant homozygotes present), and destroy undetected incipient resistance (heterozygotes present). Manipulating chemicals through rotating them in time or separating their use geographically facilitates the destruction of resistant insects.

**Resistance Management Tactics**

**Reducing Selection**

**Minimize applications**

**Theory** – The more often a pesticide is used, the more insects are exposed to selection, and the more
likely that resistance will evolve (Tabashnik 1990). Reducing the use of the pesticide will reduce the rate of selection.

**Practice** – Fumigants, especially phosphine, are used widely in the grain industry, exposing a potentially very large population of insects to selection. In addition, they are often used repeatedly on the same parcel of grain, or in stores where insect populations are maintained in harborage, so that the same population is serially exposed to selection. The aim should be to reduce the overall dependence on these materials and limit repeat fumigations. This will require the use of alternative disinfectants (chemical and non-chemical, such as heat), more effective disinestation systems, expanded use of nonchemical controls, or expanded use of protectants. To avoid calendar-based fumigation, the industry requires better insect detection systems that allow monitoring of whole bulks.

**Storage hygiene – Reduce the number of insects exposed to selection**

**Theory** – Storage hygiene refers to the removal and disposal of all residues of grain, grain dust, dockage, etc., from storages and associated equipment. Grain insect pests can survive for long periods and even multiply on only a small amount of this material. If high levels of cleanliness are maintained inside storages, then the likelihood of insects that carry resistance genes surviving from one storage season to the next is greatly reduced. In addition, if grain residues are removed from the outside of storages and storage equipment, then the risk of infestation from these sources by insects carrying resistance genes is also reduced. Maintaining strict hygiene standards reduces the risk of insect populations becoming resident in a silo and from being repeatedly subject to selection with pesticide.

**Practice** – Good hygiene reduces general infestation pressure and is the basis for effective integrated pest management. High standards of hygiene require an investment in time, training, equipment, and the determination to do a thorough job.

The practice of applying insecticidal sprays to storage structures will increase the likelihood of effectively controlling insects and provides some residual effect but risks selection for resistance to insecticides used. Diatomaceous earth treatments should be used instead of chemical protectants wherever practicable. Diatomaceous earths are not effective where significant numbers of insects are already present in the grain or in high humidity situations, such as ports.

**Grain cooling – Reduce the number of selection events**

**Theory** – Low temperatures can slow insect development and reproductive rates significantly, and inhibit population growth. Reducing the insect population growth rate should reduce the number of treatments such as fumigations required on any parcel of grain and, in some cases, may permit no chemical use.

**Practice** – In many cases, such as tropical and subtropical regions, cooling alone will not ensure insect-free grain but may be sufficient for some segregations such as feed. In practice, feed can come out of any storage and is a potential source of infestation in a common grain path. With effective monitoring, cooling should reduce the number of fumigations required on any parcel of grain. Note that cooler grain may require longer fumigation times or higher fumigant concentrations for effective control. Note that in many situations, storages cannot be cooled economically.

**Provide untreated refuges**

**Theory** – Refuges or areas of untreated habitat (grain, etc.) serve as sources of large numbers of insects, both susceptible and resistant (Onstad 2008). If resistant insects have lower fitness relative to susceptibles, then in the absence of chemical selection, the presence of refuges will result in an increase in the relative frequency of susceptible genes. Early in a resistance episode, susceptible individuals greatly outnumber resistant insects. Refuges also function as a reservoir from which susceptible genes may flow through insect movement and interbreeding into insect populations that are under selection, to reduce the frequency of resistance genes in the populations.

**Practice** – This tactic is often a key part of resistance-management strategies for field crops. This tactic is difficult to implement in the grain industry because it contradicts storage hygiene and market requirements for insect-free grain. Nevertheless, refuges may exist in other parts of the environment. The potential advantages to be gained because of differences in fitness between resistant and susceptible insects may not be realized in the grain storage sys-
tem because differences in fitness between resistance genotypes often are not demonstrated.

A possible variation of this tactic would be to reduce use of a particular pesticide in certain sectors of the industry to create “refuges” from selection. For example, farmers could be encouraged to use non-chemical control technologies including hygiene, cooling, controlled atmospheres, diatomaceous earth, and alternative chemicals (where markets permit).

**Destroying Resistant Insects**

**High doses – Make resistance recessive**

**Theory** – Application of doses high enough to control resistant heterozygotes (insects carrying one copy of the resistance gene or genes) will delay the evolution of resistance because these insects do not survive to reproduce (Roush and Daly 1990). This tactic requires reliable distribution of adequate concentrations of the chemical treatment in a closed system. If resistant homozygotes (insects carrying two copies of the resistance gene(s)) survive such treatments, resistance will rapidly increase in frequency.

**Practice** – This tactic requires implementation very early in resistance development because using high doses that would control only heterozygotes could result in rapid selection for resistance in insect populations where resistant homozygotes are already present.

A practical way to apply this tactic is to aim to control homozygote-resistant insects. This can be done with phosphine because the dosage (concentration and exposure period) of this fumigant can be varied. Fumigation in a silo proven to be sealed will allow concentrations to be held at the required concentration for long enough to ensure destruction of resistant homozygotes (Daglish et al. 2002, Collins et al. 2005) and minimize the opportunity for insects to escape the toxicant. To be effective, this tactic requires optimal application of phosphine and the avoidance of under-dosing. A risk with this tactic is the possible selection for even higher levels of resistance in target species.

**Manipulating chemicals – Rotate in time or separate geographically**

These tactics require two preconditions to be met to be successful. First, the mechanisms of resistance that develop with each of the components should be different and independent (i.e., no cross-resistance). Secondly, the frequency of resistance genes in the target populations must be low and should not occur together in the same individual (Roush 1989). In addition, each tactic relies on its own set of assumptions.

**Theory** – Rotation in time tactic involves the rotation of two or more pesticides to which the insects do not show cross-resistance. Rotations assume, at least at the beginning of the resistance episode, that individuals that are resistant to one pesticide have substantially lower fitness than susceptibles, so their frequency declines between applications of that chemical, and that there is a large gene pool of susceptible insects that will readily mate with resistant insects and dilute the resistance-gene frequency, or both (Tabashnik 1990). The latter relies on the presence of large areas of untreated habitat. Decisions on when to rotate ideally should be made on the basis of the length of insect generations so the period of selection of any pesticide does not extend beyond one generation. Rotations also need to be coordinated over a large area so insects functionally belonging to the same gene pool are not simultaneously selected for resistance to the different pesticides used in the alternation.

**Practice** – Currently, alternative fumigants and grain protectants are limited. Even when potentially available, they are further limited by issues such as environmental and health concerns, cost, and grain-handling logistics.

Most of the conditions described for success of this strategy cannot be met in the grain industry. For example, evidence to date suggests that resistance to phosphine does not decline between applications. Frequency of weak phosphine resistance is often already high in insect populations, and strong resistance genes are present in most regions, so large populations of susceptibles are not available. Further research is needed on these aspects.

Alternative fumigants or grain protectants have value in that they can be used to control undetected incipient resistant populations and to control known resistance outbreaks. In the former, the alternative would be part of a predetermined rotation. In the latter, the alternative would be used when resistance to phosphine has been diagnosed.
Conclusion

The previous discussion of feasible resistance management tactics reveals that grain storage managers have a limited number of options that can be implemented to manage resistance to chemical treatments. Management is restricted, in particular, by the lack of viable alternatives.

A practical resistance management strategy that could be implemented immediately would include:

- Limiting the number of repeat treatments (fumigations) on the same parcel of grain.
- Ensuring highest standards of application. For fumigation this means use of sealed silos so that recommended minimum concentrations and exposure periods are met to avoid under-dosing.
- Strong emphasis on use of nonchemical control technologies including hygiene, cooling, controlled atmospheres, and diatomaceous earths to minimize the use of essential materials such as phosphine across the grain industry.
- Use of alternative chemicals such as protectants and structural treatments (including diatomaceous earth) where acceptable and effective.
- Introduce limited strategic use of alternative fumigants and other chemicals when available.

References


