LIVING WITH RESISTANT STRAINS OF STORAGE PESTS

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Abstract.--Storage pests vary widely in their natural susceptibility to toxicants and species differences within one genus may be greater than those found between susceptible and resistant strains. The proportion of resistant phenotypes may rapidly decline in the absence of selection but the development of resistance does not always involve reduced fitness. Malathion-specific resistance in Tribolium castaneum seems not to involve any loss of fitness in laboratory or field conditions. A method for identifying malathion-resistant genotypes in this species is described. Laboratory studies on biological factors contributing to the fitness of resistant strains are difficult to relate to practical conditions. Such factors are most easily measured under optimal conditions but they may be most significant when the environment is suboptimal. Pleiotropic effects of the genes conferring resistance need to be distinguished from unrelated strain differences. However the true significance of pleiotropic effects on fitness is unlikely to be realised if these are studied only in artificial genetic backgrounds. The most important pleiotropic effects are those which can be used to control the pest as in negatively correlated resistance.

Introduction

Among stored-product pests, strains resistant to pesticides are now widespread. Most of the major pests are involved, as are all the main types of pesticide in use. In a detailed review Champ (1986) lists 31 storage pests resistant to residual insecticides and 9 resistant to fumigants. Since then further instances have been reported, and evidence of additional cases are being presented at this meeting. Those concerned with controlling or preventing infestation of stored foods have to live with this problem, and I want to discuss some of the studies which may help us to do so.

We can meet the problem of resistance by changing the control method. We may get away, at least for a time, by merely changing the pesticide. Better, perhaps, is to seek an alternative control approach in which chemicals are not used, so that resistance to chemical control is irrelevant. In this sense much of this conference is concerned with living with resistant strains. However, there are often practical constraints to the rapid introduction of non-chemical approaches, and many studies on resistance have been undertaken in the hope that they will lead to improved chemical control methods for such strains. Perhaps the ideal aim should be an approach in which the resistance mechanism itself is used against the resistant insects. I do not think this has yet been achieved in practice, but it should be the ultimate aim of studies of insecticide-resistant strains.

Resistant insects are not only able to tolerate doses of pesticide which would kill susceptible ones, they also have the capacity to pass on this ability to their offspring. Resistance is thus essentially a genetic phenomenom as well as a problem in toxicology. Resistance studies need therefore to integrate the approaches of both disciplines and this has not always been achieved in the past.

Heterogeneity of resistant strains

Because resistance is inherited it follows that most resistant strains are heterogeneous. Even if only a single gene is involved a field population may well contain three genetically different types of individual:- resistant homozygotes, susceptible homozygotes, and heterozygotes. The relative proportions of these will depend on the environment the strain has recently experienced. In the presence of the pesticide the proportion of individuals with resistant genes will increase and in the absence of pesticides those with susceptible genes are likely to become more frequent. In other words the gene frequency is likely to change.

The changing genetic heterogeneity of resistant strains is frequently ignored in laboratory studies. When resistance negates practical control measures the first approach is often to test whether alternative pesticides will work. A laboratory culture is established and (sometimes after a few generations of laboratory selection) cross resistance studies are initiated. These studies, which may involve tests with a whole series of alternative pesticides, are normally undertaken with insects from a series of generations of laboratory culture. In each generation of laboratory culture in the absence of pesticide the gene frequency is likely to change. Obviously the value of quantitative comparisons of cross resistance levels is limited when the test insects used for the compounds being compared come from widely separated generations. Yet few studies of cross resistance give any indication of the number of generations of laboratory culture involved. Worse perhaps are those biological studies undertaken with so-called 'resistant strains' which prove to be strains which were resistant when taken into culture some years previously but whose resistance status has not since been evaluated. When such studies show that the responses of the 'resistant' insects are little different from those of susceptibles, there is always the possibility that this is because the frequency of resistant genotypes has dropped to a very low level.

Natural tolerance to toxicants

Many stored-product pests cope well with toxicants occurring naturally in the products they infest. Lasioderma serricorne attacks tobacco despite the nicotine present, and Stegobium paniceum is notorious for its ability to infest plant drugs despite the presence of toxicants like strychnine, aconite or belladonna. An ecological cost may be involved since these insects do better in less toxic foods, but despite this, evolution has favoured the retention of the ability to tolerate toxicants in these species.

Closely related species may also differ widely in their ability to tolerate toxicants, even when these poisons are man-made. Some years ago Johanne Daly and I studied the susceptibility of the adults of seven species of Tribolium to DDT using a topical application method (Dyte and Daly, 1970). The largest species was nearly four times as heavy as the smallest, but even when allowance was made for weight differences, we found a wide range of susceptibility. Tribolium audax, the most tolerant species, was over 1000 times more tolerant than T. confusum which was the most susceptible (Table 1). Using a similar test method a resistance factor of about x 150 for a DDT-resistant strain of T. castaneum was found after laboratory selection. This strain would be about 450 times more tolerant than susceptible T. confusum placing it between T. brevicornis and T. destructor in Table I. Clearly the range of DDT tolerance among different species of the genus is greater than that known between the susceptible and resistant strains of T. castaneum.

Table I. Susceptibility of Tribolium species to DDT

Species	Mean weight (mg)	LD50 µg/mg	Relative susceptibility
T. confusum	2.9	0.025	x 1
T. castaneum	2.2	0.076	x 3.0
T. anaphe	3.6	0.12	x 4.8
T. madens	3.5	0.19	x 7.6
T. brevicornis	8.3	0.82	x 33
T. destructor	5.1	18.4	x 736
T. audax	2.5	26.8	x 1072

We know virtually nothing about how the DDT-tolerant species of Tribolium deal with this toxicant. In T. destructor, and some resistant strains of T. castaneum, DDT is readily synergised by DMC which suggests that a DDT dehydrochlorinase may be involved. However this synergist is virtually ineffective in susceptible strains of T. castaneum, and in other species. It is clear that within this genus there are a number of mechanisms for dealing with this toxicant, and some of them are highly effective.

The natural occurrence of mechanisms for tolerating poisons suggests that such mechanisms need not be a selective disadvantage in the species that possess them. Likewise they need not necessarily be disadvantageous when resistant strains live in the absence of insecticides.

The fitness of resistant phenotypes

Population geneticists expect the frequency of resistance genes to decrease in the absence of pesticide because these genes are initially extremely rare in natural infestations before the pesticide is used. This rarity is taken to indicate that these genes have pleiotropic effects which reduce the overall fitness of resistant genotypes, and that the resistant genes only confer a net advantage when the pesticide is present. This view is supported by some experimental studies e.g. that of Muggleton (1983) who showed that when malathion-resistant strains of Oryzaephilus surinamensis were cultured in the absence of insecticide, the proportion of susceptibles increased in successive generations. However resistance may not always involve very profound biological changes. When we look at the biochemical mechanisms by which resistant insects deal with pesticides we often find they are not qualitively different from those which exist in susceptible strains, merely more efficient. This is the case, for example, with the metabolism of many organophosphorus compounds. The metabolic pathways involved in resistant and susceptible insects are often similar, but the resistant ones use one or more of these pathways more effectively.

In this respect it is of interest to consider malathion resistance in Tribolium castaneum because it has been fairly extensively studied (see Champ and Dyte, 1976 for early references). In this species at least two types of malathion resistance occur which have been referred to as 'malathion-specific' and 'non-specific'. The first type confers a cross resistance only to close analogues of malathion (most of which are not commercial insecticides) and this type of resistance is completely overcome by the synergist triphenyl phosphate. The second type involves cross resistance to a variety of organophosphorous compounds, and is unaffected by triphenyl phosphate. The differing responses to the synergist triphenyl phosphate have been used to distinguish the two types of resistance in field surveys.

Champ and Campbell-Brown found that each of these two types of malathion resistance was governed mainly by a single partially dominant gene which produced dosage-mortality responses in test crosses that were typical of single-factor inheritance. Both genes were in linkage group VI, and the cross-over frequencies with Microphthalmic (Mo) were 19 for the resistance of the malathion-specific type and 18 for the non-specific type. More recently Beeman (1983), using somewhat different methods, has studied the inheritance of malathion-specific resistance in a strain of T. castaneum from Georgia, U.S.A. He too found it to be governed by an autosomal semidominant gene in linkage group VI, though he estimated the gene to be 24.6 map units from Microphthalmic. Subsequent studies by Beeman and Nanis (1986) involving five other strains yielded recombination values between Microphthalmic and malathion resistance ranging from 17.4 to 25.2, and they concluded that a single locus or a closely linked group of loci are involved. These authors reported two incompletely dominant alleles conferring high and low levels of malathion resistance at this locus. Their high level resistance was malathion-specific, but it is not clear whether this was the case with their low level allele, as they defined their alleles only in terms of the level of resistance to malathion. (Strains of T. castaneum whose malathion resistance is not completely suppressed by

triphenyl phosphate are known to occur in the U. S. A. (Champ and Dyte 1976, Haliscak and Beeman 1983))

Beeman and Nanis (1986) examined the relative fitness of the genotypes involved in malathion-specific resistance. They set up experimental populations initiated with homozygous resistant (RR), heterozygous (+R) an homozygous susceptible (++) beetles in Hardy-Weinberg equilibrium proportions. They started with susceptible gene frequencies of 10, 50, or 90%, and cultured the stocks on food that was not ideal in an attempt to force competition between genotypes. During six consecutive non-overlapping generations they measured the frequency of susceptible homozygotes but found no replicates with frequency changes that could not be explained by random genetic drift. This stability is strong evidence that, under the conditions of their experiment, fitness differences between the genotypes were negligible. This result stands in contrast to the previously mentioned findings with Oryzaephilus surinamensis (Muggleton, 1983) .

There is little information on the fitness of resistant genotypes under field conditions but some of the information gathered during the FAO Survey (Champ and Dyte, 1976) seems instructive. In that survey 502 field strains of T. castaneum from 78 countries were tested for malathion resistance, and when this was detected the strains were tested with malathion synergised with triphenyl phosphate (TPP) so as to distinguish malathion-specific from non-specific resistance. Thus for each strain we have an estimate of the frequency of malathion-specific resistant and non-specific resistant individuals (Table II).

Table II Malathion resistance found in Tribolium castaneum in strains collected in 1972-1974 during the F. A. O. Survey showing the percentage of resistant individuals per strain in strains with malathion-specific or non-specific resistance.

Resistant individuals per strain (%)	Number of strains	
	malathion specific	non-specific
100	59	1
91-99	59	2
81-90	47	6
71-80	24	5
61-70	36	7
51-60	46	4
41-50	23	5
31-40	29	10
21-30	33	14
11-20	32	27
1-10	43	144
0	71	277
total	502	502

The estimate of the frequency of non-specific resistant individuals is the most accurate because this is obtained directly from the response to malathion with TPP. The estimate of the proportion of malathion-specific resistant individuals is poorer because this is obtained by deducting the response to malathion + TPP from that obtained with malathion alone. Obviously when the proportion of a strain responding to malathion+TPP is high, the chance of detecting malathion-specific resistance will be reduced. In practice the occurrence of strains with a high level of non-specific resistance was so rare that this potential source of error was unimportant, and the main trend is clear. Not only was malathion-specific resistance much more commom, occurring in 86% of strains compared to 45% with non-specific resistance, but the incidence of strains with a high percentage of resistant individuals was much more frequent in the case of malathion-specific resistance. In fact there was no clear relationship between the number of resistant individuals per strain and the number of strains in the case of malathion-specific resistance, whereas with non-specific resistance the number of strains was strikingly reduced as the incidence of resistant individuals per strain increased, and in over half the strains with this type of resistance the incidence of resistant individuals per strain was 10% or less.

We know little of the history of these strains, and how recently they had encountered insecticidal treatments. However, at the time they were collected malathion was the only organophosphorus compound widely used in or near stored foodstuffs. Selection with malathion should favour the spread of both types of resistance, but the use of other insecticides would have favoured only non-specific resistance. It seems likely that the low frequency of non-specific resistant strains, and in particular the low incidence of individuals with this type of resistance even in strains where it occurred, is likely to have been caused by the rapid loss of resistant individuals of this type when selection was diminished. In other words individuals with non-specific resistance were much less fit than were susceptibles. By contrast, malathion-specific resistant individuals were little if any less fit than susceptibles, and with this type of resistance once relatively high frequencies of resistant individuals had been selected, the gene frequency remained high in the absence of insecticide selection. This evidence from field strains with malathion-specific resistance is in accord with the laboratory studies discussed above in which a laboratory strain whose malathion-specific resistance was derived from an american strain was used. Moreover since the malathion-specific resistant strains in the F.A.O. Survey emanated from over 70 countries it seems likely that the lack of deleterious effects on fitness is a general property of malathion-specific resistance in this species, which is relevant under practical conditions.

In view of this contrasting result with the two types of resistance to malathion in T. castaneum, it is of interest to examine the number of resistant individuals per strain in other species, particularly those which are known to have both specific and non-specific resistances to malathion. Both Tribolium confusum and Rhyzopertha dominica meet this criterion, though at the time of the F.A.O. study non-specific resistance in R. dominica was rare, and only one strain of this type was detected in the Survey. The data for these species is summarised in Table III. In each case resistant strains containing many resistant individuals are much less

common than those containing few, as is the case with non-specific resistance in T. castaneum. It appears that it is malathion-specific resistance in T. castaneum rather than malathion-specific resistance in general which is unusual in having little effect on fitness.

Table III Malathion resistance found in Tribolium confusum and Rhyzopertha dominica in strains collected in 1972-1974 during the F. A. O. Survey, showing the percentage of resistant individuals per strain in strains with malathion-specific or non-specific resistance.

Resistant individuals per strain (%)	Number of strains		
	T. confusum		R. dominica
	malathion specific	non-specific	malathion specific
100	0	0	0
91-99	4	1	1
81-90	1	1	0
71-80	2	0	2
61-70	3	3	2
51-60	6	0	2
41-50	2	1	6
31-40	8	2	5
21-30	6	5	4
11-20	8	8	9
1-10	30	18	24
0	49	80	103
total	119	119	158

White and Bell (1990) have recently described a detailed laboratory study of fitness in malathion specific-resistant Cryptolestes ferrugineus. They were able to show that some fitness defects in a selected resistant strain were not pleiotropic effects of the semi-dominant autosomal gene governing the resistance. However a behavioural characteristic of the larvae which could well be disadvantageous appeared to be related to the resistance gene or genes closely linked to it.

Resistant genotypes in natural infestations

Much of the above discussion is concerned with the relative fitness of phenotypes not genotypes. In Oryzaephilus surinamensis several mechanisms appear to be involved in the inheritance of resistance to organophosphorus insecticides (Muggleton, 1987), and little is known about the genetics of malathion resistance. In Tribolium castaneum, although malathion-specific resistance is largely governed by a single semi-dominant gene, neither the F. A. O. testing technique, nor that used by Beeman and his colleagues (which uses time of exposure as a dosage variable), permit the separation of resistant homozygotes from heterozygotes. There is considerable overlap in the dose-response ranges of these two genotypes, so they cannot be

distinguished by a discriminating dose. There is of course no reason why the fitnesses of the RR and +R genotypes should be similar. Their responses to malathion differ, and their other biolological responses in both toxic and non-toxic environments may also differ. It is obviously desirable that these two genotypes should be distinguished in laboratory and field populations. Moreover until they are, it will not possible to measure the gene frequency of the resistance gene.

Mark Rowland and I have attempted to develop a technique for looking at the frequency of all three of the genotypes concerned with malathion-specific resistance in natural infestations of T. castaneum. Although at present we have only a few preliminary results, the technique works so it seems worth describing. The principle is simple. Adult insects from the field population are individually test mated to beetles from a laboratory culture known to be susceptible. Each of the resultant F1 progenies is then tested with a discriminating dose which knocks down all susceptibles, and from the response in the discriminating dose test the genotype of the field parent can be diagnosed. The three possible crosses are shown in the upper part of Table IV together with the expected F1 response for each field genotype.

Table IV Crosses to monitor malathion-resistant genotypes in natural infestations of Tribolium castaneum.

Field genotype		Laboratory susceptible		F1 progeny	Response to discriminating dose
++	x	++	→	++	100%
+R	x	++	→	½++, ½+R	50%
RR	x	++	→	+R	0%
bb (normal)	x	BB (black)	→	Bb (bronze)	

At first we thought only males from the field sample would be usable as females would have already mated. However this problem was overcome, and the workload reduced, by making the laboratory susceptible strain homozygous for the semi-dominant colour mutation black (BB). Beetles heterozygous for black are bronze in colour and can thus be distinguished from both the black homozygotes and wild-type homozygotes. Most field-collected females had mated previously and thus produced both normal coloured and bronze offspring in the F1, but only the latter which were known to have been sired by black laboratory susceptible males were scored in the discriminating dose test (Table IV lower part). In practice we found it convenient to hold field samples on clean food for a few days before removing active unsexed beetles and placing them individually in vials of flour to which three unsexed adults from the black susceptible culture were added. By not sexing the insects time was saved, and handling reduced, though occasionally we ended up with vials containing four beetles of the same sex. Only bronze colourd individuals were scored in the discriminating dose tests as black F1 progeny were known to be from matings between individuals from the laboratory susceptible stock.

The results in Table V were obtained from two samples in which the F1 progeny produced during the holding period were all susceptible to malathion synergised by triphenyl phosphate. Thus only malathion-specific resistance was present. The gene frequency of the R gene was estimated to be about 26% in one strain and 95% in the other. The table compares the number of each genotype found with that expected from the Hardy-Weinberg equilibrium. The data suggest there was reasonable accord in the larger sample, but that there were too few heterozygotes and too many homozygotes in the smaller sample. However both samples were too small to permit rigorous comparisons. They merely illustrate the type of data that can be obtained.

Table V Occurrence of the three malathion-specific resistance genotypes in two samples of Tribolium castaneum from imports arriving in the U.K.

Import	No. of each genotype ++	+R	RR	Total	Frequency of R gene
Cotton seed expeller	24	8	6	38	26%
(exp*	20.8	14.6	2.6)		
Rice bran pellets	0	9	86	95	95%
(exp	0.2	9.0	85.7)		

*exp = No. expected if R gene in Hardy-Weinberg equilibrium

There are viable semi-dominant black mutations in other stored-product pests e.g. Rhyzopertha dominica (Champ and Genn, 1971) and it would seem that this 'black beetle technique' could be used with other species. Other types of semidominant mutant may be suitable for marking the susceptible strain in some species.

The nature of fitness differences

Different strains of stored-product insects may differ in life history parameters and in other ways. Strains with a long history of laboratory culture may differ from more recently collected field strains, strains of tropical origin may differ from those in temperate regions, and there may also be differences which seem not to be associated with immediately obvious environmental factors (see for instance Collins et. al, 1989, Jacob and Fleming, 1989, and papers cited by these authors). Of particular interest from the point of view of pest management are differences in those biological parameters which affect the net reproductive rate or innate capacity for increase, but other characteristics e.g. behavioural differences can be important. In view of this it is not surprising that resistant and susceptible strains should differ in properties other than their ability to tolerate particular pesticides.

If we are to understand the effects of resistance on fitness we need to distinguish the true pleiotropic effects of the resistance gene(s) from incidental strain differences which may be inherited independently and are thus not an inevitable concomitant of the resistance being studied. This

can be done by studying the inheritance of the resistance, and then repeatedly backcrossing to susceptibles in successive generations until each resistant gene is in a genetic background that is similar to that of its susceptible allele. This was done for example in White and Bell's (1990) study of malathion resistant Cryptolestes ferrugineus. An alternative approach might be to compare RR, R+, and ++ individuals derived from a segregating F2 population. This approach might, for example, be appropriate in a laboratory study to determine whether the three genotypes differed in their response to pitfall traps or refuges. It would of course be necessary for the three genotypes to be identifiable after they had been recovered from the trap or refuge, e.g. by a method like the 'black beetle technique' outlined above.

Unfortunately, the laboratory techniques necessary to distinguish the true pleiotropic effects of resistance genes do not prove that these effects are significant in field populations. Any changes in the background genotype that ameliorate the fitness disadvantages of the resistance gene will have high selective value and are likely to spread in resistant populations. Some of these changes in the genetic background are likely to be removed in the production of the strains with similar background genotype necessary to prove pleiotropy. On the other hand those genes ameliorating fitness defects which will be most readily retained in the resistant population will be those linked to the resistance gene, particularly if the linkage is close. Supergenes with improved fitness are likely to evolve.

There are also problems in relating laboratory studies of pleiotropic effects to the management of resistance in the field. For example say a reduced oviposition rate is a property of a resistant strain. The demonstration that this is a true pleiotropic effect will be most readily done under temperature and humidity conditions which maximise oviposition in the susceptible strain. However if this characteristic is important under practical conditions, it is likely to be most significant when the temperature and humidity of the environment are such that oviposition is rather poor even in normal susceptible beetles. There is also a need to evaluate the significance of pleiotropic effects in the presence as well as the absence of pesticides.

Negatively correlated resistance

When resistance occurs the first studied pleiotropic effect studied is often the cross-resistance to other toxicants. Pleiotropy tends to be assumed rather than proven. Usually a pattern of cross resistance emerges, and if this fits in with other studies, such as differences in the type or extent of metabolic breakdown of the pesticide in the two strains then true cross-resistance (i.e. pleiotropy) is assumed. If the pattern is confused then multiple resistance (i.e. a lack of true pleiotropy) is suspected.

In malathion-specific resistance in Tribolium castaneum there is cross resistance to certain organophoshorus compounds with carboxyesters in the leaving group but not to other organophosphorus compounds (Dyte and Blackman, 1972, Beeman, 1983). This accords with the enhanced metabolism of malathion by carboxyesterases in the resistant strain. Moreover because the synergist triphenyl phosphate not only inhibits the production of

carboxyesterase products but also overcomes the resistance, we can be reasonably confidant that the enhanced metabolism is the cause of the resistance (Dyte and Rowlands, 1968).

In the case of non-specific malathion resistance in this species the situation is less clear but of considerable interest. The strain CTC12 first reported by Champ and Campbell-Brown (1970) has been most studied (see Champ and Dyte, 1976 for earlier references). The level of resistance to malathion is much less than that found in many malathion-specific strains, and there is cross-resistance to malathion synergised by triphenyl phosphate, many other organophosphorus compounds, and many other unrelated types of insecticide. An unusual feature of the resistance to organophosphorus compounds is that the resistance levels to phosphates (P=O compounds) are higher than the levels to the analagous phosphorothioates (P=S compounds). Thus in strain CTC12 the resistance factor for malaoxon was x61 compared to x8 for malathion, whereas in a strain with malathion-specific resistance the resistance factor for malaoxon was x19 compared to x263 for malathion when measured by the same method.

The non-specific resistant beetles metabolised malathion at the same rate as did susceptibles and the various detoxication products of malathion were produced at similar rates in the two strains. However the level of malaoxon found in the resistant strain was less than that in the susceptibles and the resistants produced two metabolites of malaoxon (demethyl malaoxon and methyl thiolophosphate) which were not recovered from the susceptibles. The equivalent metabolite of malathion (demethyl malathion) was not found in either strain.

It appears that this strain is able to demethylate malaoxon but not malathion, and that this is a qualitative difference between the resistant and susceptible strains. Such a mechanism is in accord with the higher resistance levels to phosphates compared to phosphorothioates, because the latter would only be metabolised after conversion to the oxygen analogue. The metabolism of tetrachlorvinphos, a phosphate to which there was a high cross resistance was also studied in the two strains. In the susceptibles this compound was slowly metabolised, the main metabolite being demethyl tetrachlorvinphos. In the CTC12 strain the metabolism was much more rapid and much more demethyl tetrachlorvinphos was produced, the levels of other metabolites being similar to those found in the susceptibles. Thus a similar metabolic pathway was involved though the difference between the two strains was quantitative rather than qualitative.

It is of considerable interest that Champ and Campbell-Brown found that the specific and non-specific resistances to malathion in T. castaneum were largely governed by single semidominant genes which were very closely linked or allelic. When this finding is associated with the toxicological data we have a potential mechanism for negatively correlated resistance. At what is probably the same locus we have one allele which confers high resistance to malathion but susceptibility to tetrachlorvinphos, and another allele which confers resistance to tetrachlorvinphos but a relatively low level of resistance to malathion. The relevant cross-resistance data are summarised in Table VI.

In practice it may be that the relatively low level of malathion resistance found in non-specific resistance is unacceptable, and in any event many pests are now resistant to malathion and many treatments are applied against several species. Even so, cases of negatively correlatedresistance are so rare that it would be of interest if this possible one could be confirmed. I suspect such cases are rare because often toxicologists are satisfied with identifying a probable mechanism of reistance in a particular case, without going on to attempt to use the mechanism discovered against the resistant pest. Only when our knowledge of resistant strains enables us to do this will we be able to readily live with resistant strains of storage pests.

Table VI Levels of resistance to malathion and tetrachlorvinphos in malathion-resistant strains of Tribolium castaneum.

Type of resistance	Treatment method	Resistance factor	
		malathion	tetrachlorvinphos
malathion -specific	impregnated papers	x 260	x 1
	topical	x 263	--
non-specific	impregnated papers	x18	> x 227
	topical	x8	x 55

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VIVRE AVEC DES SOUCHES RESISTANTES DE RAVAGEURS DES DENREES STOCKEES

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RESUME

La résistance aux pesticides chez les insectes des denrées stockées est actuellement largement répandue. La mise en oeuvre de stratégies visant à gérer ces populations de ravageurs résistants commence avec la compréhension des aptitudes des souches résistantes en milieu toxique et non toxique. Il est particulièrement important d'évaluer comment le développement des aptitudes physiologiques à résister aux pesticides risque de modifier les caractéristiques biologiques de l'espèce, sous l'angle du potentiel biotique de l'insecte et des méthodes de lutte autres que chimiques. Les approches indépendantes de toxicologues et des généticiens doivent être prises en compte. Les effets pléiotropiques des gènes confèrant la résistance doivent être distingués des différences dues à des souches génétiquement différentes. Cependant, la signification exacte des effets pléiotropiques sur ces aptitudes a peu de chances d'être comprise si on se contente des études entreprises sur une base purement génétique et artificielle.