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Review

# Arthropod pest resurgence: an overview of potential mechanisms

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The phenomenon of insecticide-induced resurgence of arthropod pests has long been known to occur in response to a reduction in natural enemy populations, releasing the pest population from regulation. However, studies of resurgent populations infrequently examine other mechanisms, although numerous alternative mechanisms such as physiological enhancement of pest fecundity, reduction in herbivore–herbivore competition, changes in pest behaviour, altered host-plant nutrition, or increased attractiveness may also cause, or enhance the probability of, resurgence. Additionally, many studies that have identified natural enemy mortality as the primary cause of resurgence do not document a priori regulation by natural enemies and, therefore, are correlative in nature. In this paper, a universal definition of resurgence is proposed and criteria for determining the occurrence of resurgence are listed. Both of these are essential to rigorous evaluation of this important phenomenon. Alternative mechanisms for resurgence and potential areas of future research are identified. It is argued that insecticide resistance is not a mechanism underlying resurgence; rather, it simply enhances the probability that resurgence may occur. The similarity of resurgence to some insect outbreaks is discussed. In some cases natural outbreaks differ only in the initiating factor. The importance of resurgence management to plant protection is that resurgence is totally contradictory to the intended outcome of insecticidal application. This conflict strengthens the need to identify specifically the causal factors for each case of resurgence in order to manage this detrimental phenomenon effectively.

**Keywords:** resurgence; natural enemies; insecticides

Despite early recognition as an important problem in pest management, resurgence of arthropod pest species remains a widely misunderstood aspect of pest management programmes. This may explain the diversity of definitions that have been proposed for insecticide-induced resurgence (*Table 1*). Part of the problem may be that, in many studies, no attempt has been made to determine the underlying mechanism(s) responsible for increases in pest species abundance after insecticide application (Reynolds, 1971). Instead of determining the cause(s) of resurgence many studies simply document the occurrence of resurgence by identifying a failure of the insecticide to control the target pest, documenting unexpectedly high pest densities, or simply comparing unquantified pest and natural enemy abundances in treated plots compared to control plots (Kinzer *et al.*, 1977; Shepard, Carner and Turnipseed, 1977; Morrison, Bradley and van Duyn, 1979). Also lacking is a cohesive database for researchers to evaluate occurrences of resurgence. Despite these limitations, resurgence of insect pests is commonly believed to occur solely as a result of insecticide-related elimination of natural enemies. A causal relationship between natural enemy decline and pest resurgence has even been included in several definitions of pest resurgence (*Table 1*). Without question, natural enemy mortality is one mechanism responsible for insecticide-induced resurgences. However, many other mechanisms may cause resurgences, or increase the likelihood that they will occur. Nevertheless, when alternative causes have been identified, only rarely have they been examined critically or accepted.

This paper is not an exhaustive review of pest resurgence. Rather, our primary objectives are (1) to point out the ambiguities and variability in published definitions of resurgence, (2) to provide an overview of resurgence mechanisms in order to gain a perspective of the multitude of factors that come into play, (3) to delineate the relationships among factors responsible for insecticide-induced resurgences, naturally occurring herbivore outbreaks, insecticide resistance and secondary pest outbreak, (4) to establish the role of resurgence in crop management and (5) to relate the concept of resurgence to ecological and evolutionary theory. All these objectives rely on the accurate identification of the causal mechanisms of resurgence. Only through a mechanistic approach can the phenomenon of resurgence be understood and attempts be made to limit the occurrence of this detrimental side effect of insecticide use. We hope that this treatise will lead to more rigorous multifactorial experimental approaches to the study of the mechanisms of resurgence and dispel the idea that resurgence is caused *solely* by natural enemy mortality.

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In order to provide a framework for this review, we start by identifying and defining resurgence, then we review potential mechanisms that act either to cause or to enhance resurgence. Our discussion of specific mechanisms begins with a review of natural enemy mortality because of its prominent position in the resurgence literature. After reviewing mechanisms, we discuss the relevance and importance of resurgence to related ecological and evolutionary processes, and to plant pest management.

## What is insecticide-induced resurgence?

We define resurgence as an increase in *target* arthropod 'pest' species abundance to a level which exceeds that of a control or untreated population (*Figure 1*) following the application of an insecticide (acaricide). Given the presumed toxicity of the insecticide (acaricide) this increase is unexpected. Some definitions (*Table 1*) and reported occurrences of resurgence include an initial decline in pest abundance immediately following insecticide application (*Figure 2*). This decline is typically followed by an increase in the pest population to a level higher than before application. Pest populations may also resurge without this initial decline. This may occur, for example, in cases where pesticides have sublethal effects on a pest.

A secondary pest outbreak is closely related to, and often confused with, resurgence. In fact, Metcalf (1986) defined secondary pest outbreak as a 'type II resurgence'. For clarity, we define secondary pest outbreak (SPO) as the increase, after insecticide application, of a non-target species. The presumption underlying SPO is that before insecticide application the non-target species had been either regulated, excluded, or otherwise maintained at sub-economic levels. Despite the obvious differences between resurgence and SPO, the causal mechanisms responsible for both phenomena may be similar.

### Identifying resurgences

We propose several criteria that would demonstrate whether an increase in pest abundance is related to insecticide- or acaricide-induced resurgence. Adherence to these criteria should permit a more rigorous experimental approach to the identification of causal

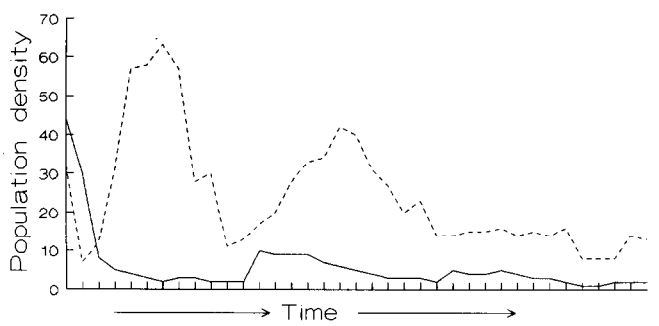


Figure 1. Properly controlled example of insecticide-induced resurgence. Insecticide treatment occurred at time zero. In this case a control population of the pest species (——) remains at the lower level than a population that has been treated with an insecticide (– – –). Adapted from Huffaker and Kennet (1956), *Figure 1*, p. 199

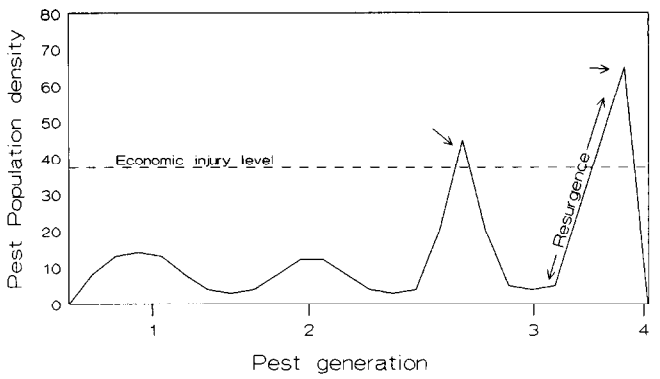


Figure 2. Hypothetical example of insecticide-induced resurgence. Left arrow: first treatment required to stop economic injury; right arrow: second treatment required to depress resurgent pest population; dashed line: economic injury level. Note that following insecticide application there is an unexpected increase in the pest population and that resurgence occurs across generations of the pest species. Adapted from Bottrell (1979), *Figure II-4*, p. 12

mechanisms of resurgence. The first criterion is that increases in pest populations must follow insecticide (or acaricide) treatment. We recognize that the use of other chemicals (i.e. fertilizers, herbicides and fungicides) may also contribute to pest density increases via mechanisms similar to those induced by insecticides. However, these agrichemicals differ in that their use is not intended to cause direct mortality to the resurging pest species.

The second criterion is that the response of the pest population to insecticide application must be an increase in its abundance. Therefore, examples of 'resurgence' in which increased crop loss as the sole criterion by which inecticide-treated and untreated pest populations are compared (Hussein and Rahman, 1981) should not be considered as documented cases of resurgence. That is, crop damage or loss does not necessarily indicate an increase in pest species abundance.

Third, for studies of resurgence to be valid, unsprayed pest populations that in all respects other than the insecticide treatment, are comparable to the treated population, must be used. Reports that merely document population increases following an insecticide treatment do not necessarily identify an insecticide-induced change.

## Natural enemy mortality and alternative mechanisms of resurgence

Because many studies have identified the elimination of natural enemies as the causal factor for resurgence, and this is routinely found to be true (Downham, 1989; Croft, 1990), many important gains have been made in the identification of insecticides that cause natural enemy mortality (Brandenburg and Kennedy, 1983) or pest resurgences (Newsom and Smith, 1949; Croft, 1977; Hoy and Dahlsten, 1984; Downham, 1989; Elzen, 1989). In contrast, there has been only limited investigation of alternative effects of these insecticides. In this section we outline what is known about mechanisms responsible for pest resurgences and their implications for further research into the mechanisms responsible for this phenomenon.

Table 1. Definitions of insecticide-induced resurgence of arthropod pests

| Definition[a] | Source |
|---|---|
| . . . rapid pest population increases following insecticide/acaride application. Populations that have led to increased damage to host plants and necessitated retreatment in order to bring about the desired level of . . . control | Riley (1988) |
| . . . Increase in [pest] abundance following insecticide treatments | Bentley *et al.* (1987) |
| . . . An abnormal increase in [a] pest population following insecticide treatment, often far exceeding the economic injury level | Chelliah (1986) |
| . . . A sudden and dramatic upward shift in the general equilibrium position [of an insect population following pesticide application] so that it lies well above the economic injury level . . . typically result from alterations in action of both density independent and density dependent factors that regulate the general equilibrium position | Kogan (1986) |
| The rapid reappearance of a pest population in injurious numbers, usually brought about after application of a broad-spectrum pesticide has killed the natural enemies which normally keep a pest in check | Staring (1984) |
| . . . A significant population increase in treated compared with untreated fields | Reissig *et al.* (1982a) |
| . . . A statistically significant increase in the [pest] population or [pest] damage in insecticide-treated plots over that of untreated plots | Heinrichs *et al.* (1982) |
| The population of the target species may quickly recover from the pesticide action and for variety of reasons may rise to new and higher levels | Smith and van den Bosch (1967) |
| . . .Rapid increases of pest insect populations after treatment with chemical insecticides | Shepard *et al.* (1977) |
| Pest populations may be increased by the application of insecticides which kill both the pests and their natural enemies | DeBach (1974) |
| . . . The abnormally rapid recovery of a pest population that was initially suppressed effectively by insecticide treatment | Newsom (1974) |
| . . . Treatments may give satisfactory initial control but in absence of biological controls (which are more susceptible to an insecticide) the pest population may subsequently increase to even higher levels than before treatment | Graham-Bryce (1987) |
| Abnormally rapid return to economic abundance of a pest that was initially suppressed by a pesticide which also destroyed that pest's natural enemies | Bartlett (1964) |
| . . . When a pest returns (after a broad spectrum insecticide application) it is able to multiply without restraint from natural enemies, so that far worse pest problem is created than was present before the pesticide was applied | van Emden (1974) |
| A situation in which a population, after having been suppressed, rebounds to numbers higher than before suppression occurred | Pedigo (1989) |
| . . . Two types: I. Pests whose population were initially suppressed by the insecticide application but which rebounded to excessive levels within a relatively short time. II. Potential pests that developed into serious pests after insecticide application to control other target species | Metcalf (1980) |
| The rapid increase of the target pest population following application of an insecticide, often to a level higher than existed prior to the control measure . . . | Bottrell (1979) |
| The rapid numerical rebound of a pest population after use of a broad spectrum pesticide, brought about usually by the distraction of natural enemies which were otherwise holding the pest in check | van den Bosch and Messenger (1973) |
| . . . Post-treatment numerical increase in pest populations far above those occurring in conventionally-treated plots | Gerson and Cohen (1989) |
| The rapid reappearance of a pest population in injurious numbers, usually brought about after the application of a broad-spectrum pesticide has killed the natural enemies which normally keep a pest in check | Oudejans (1983) |
| . . . Levels [of the herbivore] were significantly higher after application [of the insecticide] than before application | Jones (1990) |

[a]Various parameters are included in these definitions which limited their usefulness in determining the causes of resurgence; factors such as host plant damage, economic considerations, and inclusion of natural enemies limit the applicability of the definition to all cases of resurgence

## Differential mortality of natural enemies and pests

The elimination of natural enemies as the result of insecticide spraying has long been believed to be the primary factor responsible for pest resurgence (Ripper, 1956; Van de Vrie, McMurtry and Huffaker, 1972; Croft and Brown, 1975; Metcalf, 1975). Even early insecticides (e.g. heavy metal compounds, elemental sulphur and petroleum oil formulations) that are very selective, caused resurgences. Yet, due to the selective nature of these insecticides, target insect resurgence from the disturbance or elimination of natural enemies

was rare (Glass, 1975). Nevertheless, mortality of natural enemies due to insecticide application was documented even before organic pesticides came into widespread use (Bartlett, 1964). With the increasing use of pesticides and the introduction of DDT and other new classes of pesticides after World War II, there was a parallel increase in reports of pest resurgence apparently resulting from natural enemy mortality. By 1956, Ripper had tabulated examples of more than 50 herbivore species that increased in abundance after being sprayed with insecticide. In many of these examples, such an increase was often explicitly or implicitly linked to natural enemy mortality. Since then, many additional studies have documented a negative correlation between pest and natural enemy population densities following the application of insecticides (e.g. Kinzer *et al.*, 1977; Shepard *et al.*, 1977; Morrison *et al.*, 1979; Downham, 1989; Croft, 1990).

Unfortunately, many of these studies are flawed because elimination of natural enemies by insecticides or acaricides can be confirmed as the cause of resurgences only if it can be demonstrated that the same natural enemies regulate the pest under field conditions. If either effective control of the pest species by natural enemies in pre-spray (or control) plots, or a density-dependent relationship between the pest and natural enemy were documented together with differential mortality of natural enemies, then a differential mortality can be causally linked to resurgence.

Another problem is that many conclusions have been drawn based solely on laboratory data. In the laboratory, insecticide-induced differential mortality of natural enemies has been well documented (Waage, Hassell and Godfray, 1985). However, Waage points out that the absence of refugia, the inability of natural enemies to escape laboratory confinement and the difference in the persistence of insecticides in the laboratory and the field illustrate the need for additional field studies.

## Direct effects of insecticides on natural enemies

The most common and obvious direct effect of insecticides on predators or parasitoids is increased mortality. Many studies have demonstrated a differential mortality of natural enemies and their prey (Croft and Brown, 1975; Croft and Morse, 1979; Elzen, 1989; Croft, 1990). Generally, natural enemies are more susceptible to insecticides than their phytophagous host/prey (Newsom, 1967; Abdelrahman, 1973; Plapp and Bull, 1978; Coats, Coats and Ellis, 1979; Rajakulendran and Plapp, 1982; Weires *et al.*, 1982; Braun *et al.*, 1987a; Croft, 1990). For example, Taylor (1954) found that DDT treatments killed a tachinid larva within its host although the host survived. Croft and Brown (1975) noted that adult parasitoids are usually more susceptible to pesticides than the host stage they attack, and immature parasitoids are less susceptible to insecticides than are the adults. Additionally, some studies have shown that parasitized hosts may be more susceptible to insecticides than unparasitized hosts (Ahmad and Forgash, 1976; Fix and Plapp, 1983; Culin and Dubose, 1987). Computer simulations that incorporate insecticide dosage, immigration and natural enemies into resistance models indicate that much of this difference in susceptibility can be linked to use of high doses of insecticides, which limit the evolution of insecticide-resistant natural enemies and promote the evolution of resistant pest populations (Tabashnik and Croft, 1982).

Differences in susceptibility to insecticides between herbivores and natural enemies have been attributed to (1) differences in the detoxification abilities of herbivores and natural enemies (Taylor, 1954; Huffaker, 1971; Croft and Morse, 1979; Flexner, Lightheart and Croft, 1986), (2) rapid concentration of pesticides (due to biological magnification) in predatory species that feed on contaminated prey (Reda and El-Banhawy, 1988), (3) increased exposure of adult parasitoids to pesticide residues due to higher mobility than their herbivorous prey (McClure, 1977; Bostanian *et al.*, 1984; Hoy, 1984) and (4) natural enemies' physiological inability to develop resistance as easily as their host species (Croft, 1972).

Even sublethal doses of insecticides can negatively affect the behaviour and physiology of natural enemies. For example, insecticides applied in a soybean system can prolong the development time and reduce the fecundity of predators (Lawrence, Kerr and Whitcomb; 1973; Walker and Turnipseed, 1976). Pesticides can similarly negatively affect parasite fecundity (Smith and Grosch, 1976). Some insecticides and acaricides also can repel some parasitoids and predators (Bartlett, 1965), increase their walking speed (Hoy and Dahlsten, 1984), reduce food intake (O'Brien, Elzen and Vinson, 1985), decrease flight activity (Elzen, O'Brien and Powell, 1989) and reduce foraging behaviour (Jiu and Waage, 1990). Similarly, sublethal exposure to insecticides may lower learning ability in parasitoids in the same way that permethrin lowers the learning ability of honeybees (Mamood and Waller, 1990). The consequences of such behavioural changes can be significant. Hull and Starner (1983) showed that changes in predator behaviour as a result of the use of a synthetic pyrethroid can cause outbreaks of phytophagous mites. In summary, insecticides can have a variety of effects on natural enemies, which may result in decreased effectiveness and an associated increase in pest abundance.

## Indirect effects of insecticides on natural enemies

Indirect effects of insecticides on natural enemies also may be important. Insecticides may interfere with the performance of natural enemies by directly altering the quality of the prey, or indirectly altering the quality of prey as a consequence of insecticide-induced changes in the host plant. Examples of indirect effects include the repulsion of phytoseiid predators exposed to malathion-treated spider mite eggs (Hussey and Huffaker, 1976), and the protection of egg sacks of cottony cushion scale sprayed with arsenicals from the predacious vedalia lady beetle (Smith, 1929). Insecticides may also alter the host plant of the pest and have a negative indirect effect on the pests' natural enemies. For example, insecticide applications have been reported to reduce the attractiveness of host plants to parasitoids (Elzen, 1989), to cause dispersal of phytoseiid mites from permethrin-treated foliage (Braun *et al.*, 1987b), and to cause a change in the foraging pattern and a reduction in search time on brussel sprout foliage treated with

permethrin and malathion by a braconid parasite of aphids (Jiu and Waage, 1990).

Insecticides may also eliminate alternative hosts (prey), or other essential food sources of natural enemies. For example, Kiritani (1979) cites a study where the elimination of egg parasitoids was brought about both by the direct lethal effect of insecticides and by the elimination of an alternative host, *Naranga aenesceus*, during the period when eggs of its primary host were absent in rice fields. Reduced availability to natural enemies of alternative foods such as honeydew may result from a decline in a homopteran population due to an insecticide application (Coppel and Mertins, 1977). Similarly, although the insecticide applied to control codling moth did not kill a secondary mealybug pest species, it did poison the honeydew produced by them. This poisoned honeydew killed adult lacewing predators that fed on the mealybug, presumably through dermal contact (Doutt and Hagen, 1950). Tabashnik and Croft (1982), using computer simulation of data collected from apple orchards, speculated that a primary cause of predator mortality following pesticides application is a reduction in number of the prey. This prey reduction limits the food available to natural enemies and, thus, causes natural enemy decline.

The direct toxic effects of insecticides may differ between species of natural enemies. For example, the application of systemic insecticides results in a reduction in predacious hemipteran populations in cotton and soybean fields whereas coccinellids remain unaffected (Ridgway *et al.*, 1967; Rummel and Reeves, 1971; Morrison *et al.*, 1979). This dichotomy may result from differences in feeding habits among the predators. Hemiptera predators (e.g. *Orius* spp. and *Geocoris* spp.) may feed on plant tissues at some stage of their life cycle (Stoner, 1970; Salas–Aguillar and Ehler, 1977; Kiman and Yeargan, 1985) whereas the coccinellids are solely predacious.

## Resurgence and the dynamics of pest–natural enemy interactions

Although negative effects of insecticides on natural enemies are an obvious mechanism responsible for resurgence, these effects may not be the underlying cause of resurgence. An underlying assumption of the differential mortality mechanism is that the disruption of natural enemies releases pest populations from regulation. This scenario in turn assumes that the insecticide-induced mortality of pests is not great enough to slow population recovery following pesticide applications. However, Hassell (1990) has hypothesized that insecticide-induced mortality of parasites acts to reduce the effect of the insecticide-induced depression of the pest population, increasing the likelihood of pest resurgence. This reduction in the population depression is due to the release of the pest population from regulation by its natural enemies. Therefore, even with significant reductions in the pest population, resurgence may occur.

Insecticide-induced reductions of pest populations may also disrupt predator–prey (parasitoid–host) interactions by reducing prey density to a point where natural enemies can no longer locate prey effectively. For example, Ehler (1989) reported that in unsprayed plots, a parasitoid showed a density-dependent response to its host, whereas in sprayed plots the response was density independent. Disruptions of this kind may lead to local extinction of natural enemy populations. Hanna, Heatherington and Judenko (1952) reported a decline in populations of coccinellids due to a lack of sufficient mealybugs to support them. Similarly, reduction of pest populations following insecticide application may result in spatial or temporal escape of pests and a patchy distribution of the prey/hosts. Numerous studies have addressed the importance of prey (host) spatial heterogeneity in enemy attack (e.g. Hassell and May, 1974; Murdoch and Oaten, 1975; Murdoch and Reeve, 1987). Cappuccino (1988) found that spatial patterns exhibited by two aphid species, *Uroleucon nigrotuberculatum* and *U. tissoti* on goldenrod, resulted in differences in the relative vulnerability to several natural enemies. Discontinuous prey/host distribution also could result from differences in levels of resistance within the pest population, in the dispersal response of prey (Penman and Chapman, 1983), or in uneven pest recolonization of the treated area. As a consequence the frequency with which natural enemies encounter hosts is similarly reduced (e.g. Bierzychudek, 1988). Reduced encounter rates of the host/prey can result in natural enemy emigration out of treated areas of increased natural enemy mortality (Coppel and Mertins, 1977).

Resurgence can be facilitated by several life history characteristics of both the pest and the natural enemy. Higher pest reproductive rate (relative to that of its natural enemy) may allow the pest population to escape suppression by natural enemies. For example, among the entomofauna of cabbage only an aphid population resurged in response to several insecticides, whereas lepidopteran and flea beetle populations were suppressed (Root and Skelsey, 1969). A similar conclusion can be drawn for 87% of the resurging pest species reviewed by Ripper (1956) that had a higher reproductive rate than most of their natural enemies. These pest groups included mites, scale insects, aphids, thrips, leafhoppers and whiteflies.

Other life history traits, such as voltinism, dispersal ability and feeding habits, may also influence the length of time required for pest and natural enemy populations to recover after insecticide application. In addition, recovery time may differ between pest and natural enemy. For example, Kapetamakis, Warman and Cranham (1986) showed that spider mites in apple orchards increased to outbreak levels within 2 months after spraying, whereas it took several more months for the population of a predatory mite to recover. In other systems, it took as long as 6 years for populations of natural enemies to recover fully from insecticide treatment (Bartlett, 1964; Smith, 1970; Kiritani, 1977).

## Alteration of plant quality

The ability of insecticides (acaricides) to alter the nutritional quality and chemical constituents of plants is fairly well documented (Chapman and Allen, 1948; Saini and Cutkomp, 1966; Wheeler and Bass, 1971; McClure, 1977; Chelliah and Heinrichs, 1980; Jones and Parrella, 1984; Mellors, Allegro and Hsu, 1984). These plant changes can, in turn, affect the feeding and

reproductive behaviour of herbivorous insects (Saini and Cutkomp, 1966; Jones and Parrella, 1984; Mellors *et al.*, 1984). Whether the alteration of plant quality by pesticides can affect herbivore reproduction and behaviour in ways which promote resurgence of insect populations is not as clearly understood. However, changes in plant quality, such as increased nutritive value (McClure, 1977; Jones, 1990), increased plant growth (Chelliah and Heinrichs, 1980), increased plant attractiveness (Chelliah and Heinrichs, 1980), and possibly reduced plant defence (although to our knowledge, the latter mechanism is undocumented) could indirectly promote or enhance resurgence.

Alteration of plant quality by carbofuran, a carbamate insecticide, is one of the best-documented examples of direct insecticide-induced changes in plants. In the absence of phytophagous insects, carbofuran has been found (1) to increase growth and yield in burly tobacco (Pless, Cherry and Morgan, 1971), (2) to increase grain yield in corn (Daynard *et al.*, 1975), (3) to enhance growth in soybeans (Wheeler and Bass, 1971; Mellors *et al.*, 1984) and (4) to synergize the action of the growth-stimulating effects of indole-3-acetic acid (IAA) applied to pea stem segments (Lee, 1977). Lee (1977) found that metabolites of carbofuran inhibited the enzymatic degradation of IAA and hypothesized that IAA persisted longer in the plant tissues, thus enhancing plant growth.

Insecticide-induced changes in plant quality have been implicated in resurgence of *Nilaparvata lugens* (the brown planthopper) on rice (Chelliah and Heinrichs, 1980; Buenaflor, Sexena and Heinrichs, 1981; Heinrichs and Mochida, 1984). For example, decamethrin was found to decrease the ratio of carbohydrates to nitrogen and to increase the levels of free amino nitrogen in a susceptible rice strain (Buenaflor *et al.*, 1981). In the same study, a resistant rice strain was found to be less prone to these biochemical changes following treatment with decamethrin. In contrast, use of perthane did not cause resurgence of the brown planthopper or changes in carbohydrate/nitrogen levels. In addition to these changes, Chelliah and Heinrichs (1980) found that whereas decamethrin and methyl parathion enhanced the growth of rice plants, perthane did not.

Many other insecticides also alter plants in ways that may benefit herbivores. One of the earliest examples of plant growth enhancement was the response of numerous crop plants to DDT exposure (Chapman and Allen, 1948; Saini and Cutkomp, 1966). Methomyl similarly increases soybean plant growth (Wheeler and Bass, 1971). Finally, Leigh (1963) found that the growth and fruiting rates of cotton treated with insecticides were enhanced. These changes often have important consequences: for example, application of malathion or permethrin to rough lemon leaves increased fecundity and reduced mortality of the mite *Panonychus citri* (McGregor) feeding on these leaves. This was presumed to be a response to improved plant quality due to the effects of the insecticides on the plant (Jones and Parrella, 1984). Permethrin also caused resurgence in the two-spotted spider mite on treated *Prunus cerasus* L., for similar reasons.

Although the above studies demonstrated that enhanced plant quality is a potential mechanism for resurgence, a cause-and-effect relationship has yet to be established. One difficulty is that it is not always obvious whether the physiological, behavioural or reproductive responses of pest species to plants altered by insecticides have an impact on pest population increases. A population-wide effect is essential in the study of the mechanisms responsible for resurgence, as stated in the criteria discussed in the Introduction.

### Induction of insect detoxification enzymes by plant secondary chemicals and/or insecticides

Mixed function oxidases (MFOs) comprise a group of enzymes (such as aldrin epoxidases and glutathione transferases) that are thought to function primarily in the metabolism and detoxification of xenobiotics such as plant defensive chemicals or insecticides (Brattsten and Wilkinson, 1973; Krieger, Feeny and Wilkinson, 1971). Various insecticides (as well as plant allelochemicals) can influence whether detoxification pathways such as those involving MFOs are activated in pest species (Terriere, 1968; Brattsten and Wilkinson, 1973; Feyereisen and Durst, 1978; Yu and Terriere, 1978). Induction of these enzymes in insects as a result of exposure to host plant secondary chemicals may, in some instances, enhance their ability to cope successfully with certain insecticides (Berry, Yu and Terriere, 1980; Yu, 1982a, b; Berry *et al.*, 1988, 1989, 1990).

Mortality of various pest species exposed to a variety of insecticides can be reduced as a result of feeding on certain host plants or particular allelochemicals of their host plants (*Table 2*). However, the induction of MFOs is unlikely to be a primary cause of pest resurgences. Instead, MFOs may increase the likelihood that resistance will develop in the pest population (see following section on insecticide resistance). Increased survival of pests due to the induction of MFOs may also enhance the direct effects of the insecticides if they cause increased fecundity (see below).

### Direct and indirect enhancement of fecundity

Direct stimulation of fecundity as a consequence of exposure to a pesticide is known to occur in some pest species (Abdallah, 1968; Dittrich, Streibert and Bathe, 1974; Dittrich, Hassan and Ernst, 1986; Dittrich, 1987). Insecticides may extend the longevity of females, physiologically stimulated changes in females that enhance egg production, or increase survival of progeny. Some mites produce more eggs per female, and their progeny have a greater female-to-male ratio when adult or nymphal females are exposed to sublethal doses of DDT or carbaryl (Dittrich *et al.*, 1974; Dittrich, 1987). Topically applied carbaryl enhances fecundity of pecan leaf scorch mites and pecan aphids (Dutcher, 1983; Dutcher and Payne, 1983). Application of decamethrin and methyl parathion directly to the integument of female brown planthoppers causes an increase in egg production that is independent of any host plant effects (Chelliah, Fabellar and Heinrichs, 1980; Reissig, Heinrichs and Valencia, 1982b; Heinrichs and Mochida, 1984). For example, diazinon does not alter plant growth or attractiveness, but does increase reproductive rate when directly applied to the brown planthopper (Chelliah and Heinrichs, 1980).

Table 2 Selected examples of reduced insecticide-induced mortality resulting from the induction of enzymes by particular host plants or host plant allelochemicals

| Herbivore | Insecticide to which exposed | Inducing foodplant | Enzyme induced | Reference |
|---|---|---|---|---|
| Southern armyworm (*Prodenia eridana*) | Carbaryl | Methylbenzenes | | Brattsten and Wilkinson (1973) |
| Gypsy moth (*Lymantria dispar*) | Dimlin, carbaryl, acephate, *Bacillus thuringiensis*, diflubenzuron | Douglas fir (*Pseudotsugas menziessi*) | Aldrin epoxidase, glutathione transferase | Berry *et al.* (1988, 1989, 1990)<br>Keating *et al.* (1988) |
| Variegated cutworm (*Peridoroma saucia*) | Acephate, methomyl, malathion | Peppermint | Aldrin epoxidase | Berry *et al.* (1980) |
| Fall armyworm (*Spodoptera frugiperda*) | Aldrin, carbamate, diazanon, methamidophos, methyl parathion | Corn, cowpea | Aldrin epoxidase, glutathione-*S*-transferase | Yu (1982a, b) |

Changes in fecundity may occur as a result of contact with treated surfaces. Age-specific fecundity, reproductive rates and mortality-corrected reproductive rates of *Panonychus citri* increase when mites are reared on excised lemon leaves, topically treated with sublethal doses of malathion and permethrin (Jones and Parrella, 1984). For the brown soft scale, methyl parathion has been implicated as the cause of increased fecundity (Hart and Ingle, 1971).

Indirect stimulation of fecundity of pest species, due to the increased nutritive value of their host plants, also occurs (see section on altered plant quality). What is not well documented is a cause-and-effect relationship between insecticidal exposure and enhanced plant quality and/or increased fecundity. Indirect enhancement of fecundity may be due to increased feeding, improved nutritional quality of the host plant, increased ability of the pest species to feed on its host or reduced competition among herbivores. For example, *Tetranychus urticae* exhibits higher fecundity when reared on trifoliate leaves of bean plants that have been dipped in a 0.05% DDT emulsion. The $F_1$ females also exhibit faster development on treated than on untreated plants. Topical application of DDT to the mites does not produce these results, suggesting that the benefits of nutritional changes in the plants caused by either DDT or its metabolites are responsible for the observed differences in fecundity. The significant increase in the mite population sprayed with DDT also suggests that the nutritive value of the host plants of the mites may be enhanced (Saini and Cutkomp, 1966).

In other studies it is less clear whether the effects of insecticides on feeding behaviour are direct or indirect. Mellors *et al.* (1984) found that, following the initial mortality caused by carbofuran, treated plants supported faster population growth of the two-spotted spider mite (*T. urticae* Koch) than did control (insecticide-free) plants. It is not clear whether the observed effects were due to feeding by mites directly on carbofuran and its metabolites, increased feeding rates on the plants, or the influence of nutritional changes in the plant.

Clearly, not all species exhibit increases in fecundity following insecticide application, nor do all increases in fecundity lead to resurgence. For example, although an increase in reproduction was found in *Stilophilus granarius* treated with DDT, this effect was more than compensated for by insecticide-induced mortality (Kuenen, 1958). Thus, Kuenen suggested that less-resistant populations of this beetle were unlikely to exhibit resurgence. However, more-resistant populations may have a greater potential to exhibit resurgence, since the higher mortality suffered by susceptible populations would counter any enhancement of fecundity. This further suggests that, although resistance is not required for resurgence to occur, resistance may enhance resurgence (see sections on alteration of plant quality and resistance).

### Effects of insecticide exposure on insect behaviour

There are no studies showing that insecticide-induced behavioural effects cause insecticide-induced resurgence. Without analyses of behavioural responses before insecticide application in non-sprayed (control) and sprayed plots, the induction of abundance-enhancing behaviours cannot be attributed to insecticides. However, behavioural changes have been suggested as causes of resurgence, such as increased motor activity (Davis, 1952b; Penman, Chapman and Hesson, 1981; Iftner and Hall, 1983; Penman and Chapman, 1983) and enhanced ovipositional behaviour (Kinzer *et al.*, 1977; Chelliah and Heinrichs, 1980). Behavioural avoidance of sprayed plants by natural enemies is also a potential causal mechanism (Jiu and Waage, 1990).

Hyperexcitability of males in response to pheromones is a sublethal effect of insecticides affecting some insects.. This insecticide-induced behavioural change may act to enhance population growth in some species if hyperexcitability results in more rapid location of calling females. By reducing age to first mating, time to first reproduction is reduced and, thus, population growth may be enhanced. Hyperexcitability of males in response to pheromones has been recorded for *Grapholita molesta* (Linn and Roelofs, 1984), *Trichoplusia ni* (Linn and Roelofs, 1985), and *Pectinophora gossypiella* (Haynes, 1988). Although this is an intriguing mechanism, there is no direct evidence that this type of hyperexcitability has resulted in an increase in mating or population growth. For all these species, treated males could detect lower concentrations of pheromone that untreated control males. Despite this, the males were less able to locate potential mates.

Increased feeding in response to insecticides has been demonstrated in a braconid parasitoid (O'Brien *et al.*, 1985), blowflies (Long and Murdock, 1983) and planthoppers (Heinrichs and Mochida, 1984). Of course, in order for an increase in feeding rate to be relevant to resurgence it must result in greater fecundity, or natality. Heinrichs and Mochida (1984) documented increased fecundity and increased feeding rate in treated planthoppers, but did not establish a cause-and-effect relationship between increased feeding rate and increased fecundity.

Host finding by adult herbivores also may be altered by exposure to insecticides. Cotton plants treated wth aldicarb or monocrotophos were preferentially selected as ovipositional sites by *Heliothis zea* and *H. virescens* significantly more often than were untreated plants (Kinzer *et al.*, 1977). However, it is not clear whether this preference resulted in increased feeding and abundance on treated plants. Chelliah and Heinrichs (1980) found that rice plants treated with insecticides were more vigorous and attractive to the brown planthopper. The preferential selection by the brown planthopper of plants treated with decamethrin and methyl parathion may be due to the altered growth and chemistry of the plants. Adults orientated more strongly to the lush, enhanced plant growth in insecticide-treated areas. Odour from insecticide residues alone had no effect on orientation. The increased attraction to the plant and aggregative feeding of the brown planthopper may have contributed to an increase in host nutritive value of fed-on shoots (e.g. free amino acids increased up to 30-fold) compared with shoots that were uninfested or supported low numbers of planthoppers (Cagampang, Pathak and Juliano, 1974). These changes in behaviour may have contributed to enhanced colonization and survival of the brown planthopper in the field and thus may have led to resurgence.

In contrast, other studies indicate that insecticides (acaricides) sometimes fail to induce behaviour that may lead to resurgence. In a recent examination of the effects of insecticides on mite motor activity, Jones (1990) found little support for the theory that the increased activity had a population-enhancing effect. Additionally, in a general review of the sublethal effects of neurotoxic insecticides on behaviour, Haynes (1988) found little direct, unambiguous evidence that behavioural changes could enhance population abundance. Clearly, further research may differentiate between the taxa or life histories that are subject to insecticide-induced behavioural changes and those that are not.

## Reduction of competition

The detrimental effect of insecticides on non-target biota (Pimentel, 1961; Newsom, 1967) may result in the elimination of phytophagous species that ordinarily compete for resources with a target pest species. The release from competition resulting from the differential effects of insecticides may, in turn, allow or facilitate the resurgence of pest populations. At low population densities (in the absence of competition) maximal reproductive potential may be realized, enabling the population to rebound to levels beyond those before spraying or those in unsprayed areas. For example, Root and Skelsey (1969) examined how applications of insecticide altered the structure of the arthropod community associated with cole crops. The application of carbaryl was followed by outbreaks in the aphid population coinciding with a reduction in densities of herbivore competitors. Similarly, reduction of intraspecific competition also may facilitate resurgence. For example, DDT and pyrethroid application can cause dispersion of spider mites that were previously aggregated (Penman and Chapman, 1983; Brandenburg and Kennedy, 1987) and thus reduce competition and enhance population growth. Relaxation of intraspecific competition has been suggested as the mechanism responsible for higher egg production per female in spider mites (Davis, 1952a, but see Jones, 1990).

## Insecticide resistance as a mechanism for resurgence

One nearly ubiquitous result of insecticide application is that some target organisms will survive insecticide exposure whereas others will not (i.e. insecticide resistance will occur). With respect to our definition of resurgence, therefore, we ask whether resistance alone can provide a mechanism of resurgence, or, if other mechanisms, alone or in combination with resistance, are responsible? By our definition, in order for an increase in pest abundance to be classified as resurgence, there must be a pest population increase in the treated plot to a level greater than that in untreated areas.

Given three hypothetical pest populations, one pesticide resistant, one susceptible, and one of unknown resistance (control), we can demonstrate the relationship between insecticide resistance and resurgence (*Figure 3*). Theoretically, pesticide applications to the resistant and susceptible populations, should respond in a predictable manner. The resistance population would decline slightly owing to mortality of susceptible individuals, and the susceptible population would decline to

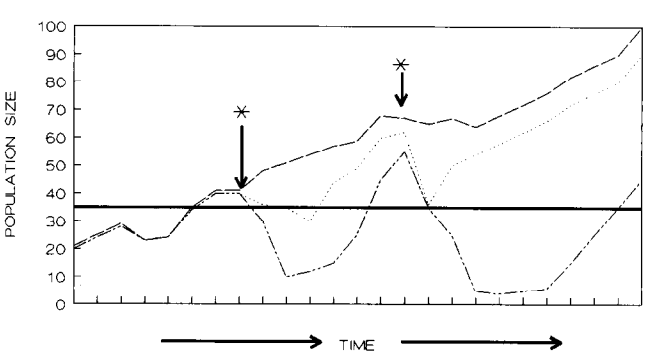


Figure 3. Hypothetical representation of the responses of susceptible (– – – –) and resistant (. . . . .) pest populations to application of a resurgence-inducing insecticide compared with an untreated control population (– –). Asterisks denote pesticide applications to resistant and susceptible populations; bold horizontal line = economic injury level. Note the resistant population response to the insecticide: the resistant population level never exceeds that of the control since it does not increase any faster than does the control population. Also note that there is a delay in the occurrence of resurgence (the point where the pest population exceeds the level prior to insecticide application) of the susceptible population compared with the resistant population.

a greater degree because it contains more susceptible individuals. However, the resistant population *would not* be expected to increase to a higher level than the untreated (control) population, that did not receive an insecticide application, unless other parameters that increase the fitness of the resistant population (such as the sublethal effects described earlier) are also affected by the insecticide treatment. Instead, the resistant population would suffer a decline due to mortality of less resistant individuals and would then increase at a rate similar to that of the untreated population (as in *Figure 3*). Therefore, resurgence by definition would not occur unless other factors cause an unexpected increase in the resistant or susceptible populations. Short-term selection for resistance, by itself, cannot cause resurgence.

However, resistance may also enhance the probability that resurgence will occur. For example, following an application of insecticides to a population containing resistant individuals, population levels will be higher than in a similar population of susceptible forms (*Figure 3*). Since the population upon which mechanisms of resurgence will act is larger, the resistant population is more likely to resurge than a susceptible population. This enhancement may be even more pronounced when factors that cause resurgence are coupled (genetically or otherwise) with factors causing resistance.

## Resurgence: an ecological or evolutionary process?

In previous sections we examined resurgence and its causal mechanisms largely as an ecological process occurring within one to several generations. Here, we ask two questions: (1) are the mechanisms that cause insect outbreaks and insecticide-induced pest resurgences the same and (2) can resurgence be an evolutionary process, representing a change in gene frequencies over time? The distinction between ecological and evolutionary processes is important, not only in the types of mechanisms involved, but also in the implications each process has for population dynamics and pest management. It is important to realize that resurgence is necessarily an ecological phenomenon, occurring as a result of insecticide application, and not an evolutionary process. Nevertheless, we argue that a variety of mechanisms causing resurgence may ultimately lead to evolutionary changes, most notably the selection of forms resistant to insecticides.

### Resurgence and insect outbreaks

Berryman (1987) defined an outbreak as 'an explosive increase in the abundance of a particular species that occurs over a relatively short period of time'. From this perspective, resurgence is an outbreak that is characterized by the nature of the causal agent, i.e. insecticides, which typically reduce rather than enhance survival. The counter-intuitive relationship between insecticide use and survival sets resurgence apart from the more common outbreaks that occur in managed and unmanaged ecosystems.

Resurgent populations are most analogous to 'pulse gradient outbreaks' (*sensu* Berryman 1986, 1987; see *Figure 4*), and appear to reach outbreak levels for similar reasons to those in 'typical outbreaks', i.e. escape from predation/parasitism, improvement of host plant quality, environmentally induced changes in behaviour and physiology, etc. An analysis of the factors that cause and/or enhance resurgence (see previous sections) indicates that in resurgent populations pesticides may (a) cause some or no mortality of the target pest but have an overall positive influence on fitness in any of several ways, such as by increasing fecundity, (b) cause some or no mortality of the target pest but have an overall positive influence by inducing changes in the food plant that are favourable to the target species, or (c) cause some or no mortality of the target pest but have an overall positive influence by negatively affecting other factors that cause target species mortality, such as natural enemies and competitors. Thus, although insecticides (acaricides) are anticipated to cause some mortality in resurgent populations, they may not have an overall negative effect on population increase and subsequent abundance.

Our discussion of the mechanisms responsible for resurgence has been limited to those factors most frequently proposed in published studies. Much more research is needed to determine which of these factors cause or enhance resurgence. As our understanding of agroecology grows, we will identify other potential mechanisms that should be evaluated, or we may gain a different perspective on a previously known mechanism. An example of each is provided.

Benedict, Chang and Bird (1991) reported that the microbial phytoflora of a crop plant can play a significant role in (a) the inhibition of plant pathogenic microbes, (b) the quality and quantity of volatile plant allelochemicals produced by plants, (c) the reduction of insect herbivory and thus plant damage, (d) the oviposition behaviour of insects, and (e) intraspecies communication due to changes in pheromone. Similarly, microbial associates of the plant in the rhizosphere

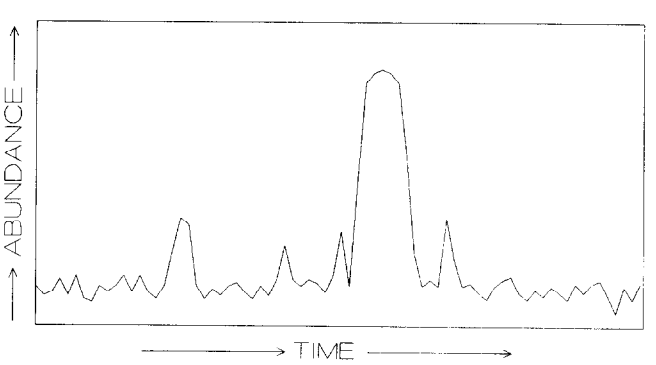


Figure 4. Hypothetical representation of eruptive pest (herbivore) outbreak (*sensu* Berryman, 1986) in an environment considered moderately favourable for pest species. This type of outbreak resembles the population fluctuations associated with resurgence but differs in its initiating factor(s); eruptive outbreaks occur in response to temporary environmental disturbances, whereas resurgence by definition requires application of an insecticide. Adapted from Berryman (1986), *Figure 4.12*, p. 71

directly and indirectly affect plant nutrition and potentially the defensive capability of plants, and thus the survival, development and fecundity of associated herbivores (Ingham and Molina, 1991; Johnson and Bentley, 1991; Jones and Last, 1991; Rabatin and Stinner, 1991).

Potentially, any insecticide that alters or inhibits these and other microbe–plant–herbivore interactions in such a way as to benefit the target species may cause resurgence. Any differential mortality of micro-organisms due to pesticides that favour microbial species that ultimately benefit the target species, might lead to resurgence. Mortality of soil micro- and macro-arthropods, which typically feed on ectomycorrhizal fungi, may enhance the survival of these fungi and their ability to provide critical nutrients to plants. Enhanced plant nutrition may, in turn, enhance herbivore fitness and hypothetically could lead to resurgence. Whether such changes occur within the timeframe necessary for the development of resurgence also has yet to be determined.

The ecological perspectives gained from insights on emigration and immigration in agroecosystems may add a different view of a previously considered mechanism such as changes in plant quality. For example, we can describe resurgent population abundance as $P = (r + i) - (m + e)$, where $r$ = birth (reproductive) rate, $m$ = mortality rate, $i$ = immigration rate and $e$ = emigration rate. This simple relationship assumes that the average abundance of the target species is the same in sprayed and prespray or untreated populations and that the $r$ of immigrants is the same, on average, as that of resident individuals. Thus, in circumstances in which $r$ and $m$ are unaffected by pesticides, resurgence may still occur if $i$ is increased or $e$ is reduced, or both occur in sprayed populations. This scenario would require that pesticides cause changes in the habitat that attract individuals of the target species and/or retain them once in the area, compared with presprayed or unsprayed areas.

One proximate cause that might lead to such changes could be the effects of pesticides on the leaf surface waxes. Insecticides typically contain surfactants, emulsifiers and solvents, which are known to disrupt or modify waxy layers of insects and presumably those of plants, since the actions of these chemicals is unlikely to be specific to insect lipids. Recent research has demonstrated the importance of allelochemicals within plant wax layers in orientation, oviposition and feeding behaviours (Juniper and Southwood, 1986). Thus, greater volatilization of these compounds, due to the disruptive actions of pesticides, might increase the attraction of the plant and thus increase immigration and/or residence time (see sections on Insect Behaviour, and Altered Plant Quality). Clearly, this scenario will depend on the specific pesticide used, as well as the plant and target species involved, as the outcome may be detrimental to the target species.

Similarly, as many physical and chemical defence structures such as trichomes and spines are on leaf surfaces, pesticides may impair these defences and enhance herbivore survival. Finally, the interactions with micro-organisms, described above, could also lead to changes in orientation/attraction and tenure time in sprayed areas.

## Implications of resurgence and related phenomena to crop protection

The goal of crop protection from pests is to minimize pest-induced losses. Current integrated pest management (IPM) programmes attempt to achieve this goal through the use of economically sound and environmentally safe management practices. The use of insecticides is one of the most common ways of managing pests, primarily because of its many advantages over other methods of control, such as their high effectiveness, rapid action, usefulness under many situations and low cost (Metcalf, 1982). For this reason, the practice of integrated pest management relies most often on strategies in which insecticides are applied only as required, as determined by sampling and the use of economic thresholds (Pedigo, Hutchins and Higley, 1986). This approach depends on the accurate prediciton of expected losses, because insecticides must be applied before loss occurs; prediction of pest abundance and damage therefore form the basis of effective strategies.

The rationale for many studies of resurgent pest populations is the presumption that an understanding of the phenomenon can help prevent crop losses. However, this goal is rarely stated explicitly in studies of resurgence (but see Eveleens, van den Bosch and Ehler, 1973). Since resurgence results in an unexpected increase in pest abundance compared with untreated populations, it is likely that the pest will cause greater-than-expected crop losses. However, because crop losses specifically attributable to resurgence are rarely measured (but see Hussein and Rahman, 1981) the crop loss and resurgence relationship is often unknown. Despite the scarcity of data, the undesirability of any possible crop loss has stimulated continuous research to find ways to eliminate or reduce the probability of pest-induced crop losses attributable to insecticide applications.

Researchers have suggested several methods for reducing the occurrence of resurgence, and one of the most common suggestions has been the substitution of insecticides that do not cause resurgence. For example, Heinrichs *et al.* (1982) and Chelliah *et al.* (1980) recommended that insecticides that cause resurgence of the brown planthopper should be identified and not recommended for insect control in Asian rice. They argued that altering rates of application is not a realistic method of reducing the possibility of resurgence. However, other researchers suggest that altering application rates or patterns of coverage may eliminate resurgence. For example, McClure (1977) concluded that the scale, *Fiorinia externa*, on hemlock could be controlled by a thorough and timely insecticide application, whereas applications covering only parts of trees or applied when natural enemies are abundant could lead to scale resurgence. Similarly, Hall (1979) suggested that an accurate and even distribution of pyrethroid insecticide at low dosages would control the primary pests of apple and minimize distribution of predators of European red mite populations, thereby avoiding secondary outbreak of the phytophagous mite.

Because of the frequently presumed importance of natural enemies as the cause of resurgence, many

researchers have suggested strategies to conserve natural enemies as a method of avoiding resurgence. For instance, Reissig *et al.* (1982b) suggested evaluating relative toxicity of insecticides to brown planthopper and its natural enemies in order to avoid compounds detrimental to the natural enemies. Furthermore, they suggested that field tests may indicate whether selective dosages, modified formulations, different application techniques, or altered timing of insecticide applications would be useful in reducing pest abundance while increasing the effectiveness of important predators. Bentley *et al.* (1987) suggested that the use of rates of acaricides lower than those suggested on the label in almond orchards, produces a higher predator–prey ratio in mite populations. However, in most cases researchers are less specific in their recommendations and instead offer generalizations such as (use) 'insecticides only when necessary to reduce the possibility of resurgence' (e.g. Shelton, Wyman and Mayor, 1981).

Beyond factors associated with insecticide application, many factors influence the degree of resurgence (i.e. the increment of increase in population abundance) including weather (Dintenfass, Bartell and Scott, 1987), crop management practices (Reissig, Heinrichs and Valencia, 1982a), the composition of the natural enemy community (Shepard *et al.*, 1977), and host-plant growth and nutrition (Heinrichs and Mochida, 1984). Many of these factors can be manipulated in a crop system at least to reduce the degree of resurgence. For example, Reissig *et al.* (1982a) found that the amount of brown planthopper resurgence in rice decreased in plots of resistant rice cultivars compared with those of susceptible rice cultivars. They suggested that resistant cultivars can be used to reduce resurgence, but cautioned that the population build-up on moderately resistant cultivars may increase the planthopper to damaging levels.

Problems associated with insecticide-induced pest outbreaks may be best solved through a complete analysis and subsequent alteration of the crop management system or by following non-insecticidal tactics of an IPM programme. For example, following outbreaks of tobacco budworm and spider mites in cotton, a re-evaluation of control practices led to a new, more economical system based on short-season varieties and traditional cultural practices (Adkisson *et al.*, 1982). These same practices may also reduce the likelihood of resurgence. On the other hand, existing crop management programmes may do a reasonable job of pest management, even tolerating losses related to resurgence, and possible programme innovations may result in increased pest outbreaks. Such a problem has been forecast in apple orchard systems where research indicated that newly labelled pyrethroid compounds would disrupt existing spider mite management programmes (Croft and Hoyt, 1978). In addition, research in almond orchards concluded that navel orangeworm was best managed by winter removal of unharvested almonds, which harbour this pest, and by early nut harvest during the growing season instead of relying on newly labelled pesticides (Bentley *et al.*, 1987).

Some factors related to the resurgence phenomenon cannot be manipulated; nevertheless, potentially, monitoring these factors may be useful in predicting the probability of resurgence. For example, current weather patterns are often used by extension entomologists to forecast various kinds of pest outbreaks and these same patterns may be shown to be associated with resurgence. Furthermore if patterns of pest abundance among habitats can be related to the incidence of resurgence, then this could be a useful diagnostic tool.

Shepard *et al.* (1977) found that newly established soybean fields are more susceptible to pest outbreaks than continuous plantings of soybeans and they suggested that the probable reason was the increase through time of natural enemy populations in cultivated areas. If natural enemy reduction by insecticide is the cause for resurgence, as Shepard *et al.* (1977) suggested, then insect pests in continuous soybean cultures would be less likely to resurge following an insecticide application. Presumably this is either because the natural enemies in continuous soybean production areas are more likely to be resistant to, or able to withstand insecticide applications, or because pesticide applications to more established populations of natural enemies are less likely to result in significant disruption of the predator–prey balance. This hypothesis has yet to be experimentally tested.

## Conclusions

Insecticide-induced resurgence has been reported since chemicals have been used as the primary tool for pest suppression. Despite this, there are few conclusive studies demonstrating a cause-and-effect relationship between any particular mechanism and resurgence (but see listed literature concerning the brown planthopper). Often, the studies that do exist simply present correlations and address only one of many potential mechanisms. The lack of evaluation of alternative mechanisms for resurgence, particularly in agroecosystems, has often led to the support of widely accepted (but generally unproven) conclusions about the mechanisms causing resurgence (such as the differential mortality of target species and their natural enemies). Conversely, a mechanistic approach to the study of resurgence should lead not only to rigorous investigation of multiple mechanisms but also to better management of pest populations.

The differential mortality of natural enemies and their herbivore hosts has been reported repeatedly, yet its relationship to resurgence has not been proved because of numerous methodological problems or lack of appropriate data. Similarly, in many cases the causal role of differential mortality between natural enemies and the target pest has been presumed almost as if it were self-evident. Despite these criticisms, differential mortality undoubtedly has been a causal or contributing factor in many insecticide-induced resurgences. If differential mortality is to be accepted as a causal (or the primary) mechanism for resurgence, a number of features of the natural enemy/pest population interactions must be determined. First, regulation of the pest population by natural enemies (or at least a functional relationship) must be demonstrated before accepting that a reduction in the abundance of natural enemies will lead to an increase in the abundance of a pest population (i.e. resurgence). A removal experiment in which natural enemies are removed and the

pest population is measured over time would give, at the very least, cursory evidence that this mechanism alone might lead to resurgence. However, even with such evidence, concurrent interactions such as physiological changes in the pest species or host plant may be as important in causing resurgence as the increase in natural enemy mortality.

Secondly, a broad experimental perspective is needed when attempting to determine cause and effect in cases of resurgence. For example, in the case of *Heliothis* spp. feeding on cotton (Kinzer *et al.*, 1977), although natural enemies were found to suffer greater mortality due to exposure to insecticides, it was the enhanced attractiveness of insecticide-treated leaves to ovipositing females that made the greatest contribution to the development of resurgence. It is necessary, therefore, that research consider other potential mechanisms such as effects on competing species of herbivores, nutritional enhancement of plants, direct stimulation of pest fecundity, and other factors reviewed here.

We urge that the existence of prerequisites, for a given resurgence mechanism, be clearly established before the role of the mechanism itself is evaluated. For instance, when differential mortality is induced, it is necessary to demonstrate that following exposure to insecticides the pest population exhibits a higher rate of population increase than the natural enemy population(s) (may be due either to selection for life history characteristics or a greater head start for the pest population relative to natural enemy populations). Only rigorous testing of this and similar preconditions can provide direct support to the idea that the reduction of natural enemy populations is the primary mechanism causing resurgence.

Thirdly, long-term studies of resurgent pest populations are necessary to identify clearly the mechanism of resurgence. Unfortunately, there are few long-term studies of resurgence in which multiple factors have been examined. Two exceptions are the ongoing studies of the resurgence of the brown planthopper feeding on rice, and the resurgences of spider mites. In the brown planthopper system, numerous factors, not just natural enemy mortality, are responsible for planthopper resurgence (Chelliah and Heinrichs, 1980; Heinrichs *et al.*, 1982; Reissig *et al.*, 1982a, b; Ooi, 1986). Likewise, numerous studies of spider mite resurgences (Huffaker and Spitzer, 1950; Gerson and Cohen 1989; Jones, 1990) have failed to support the initial interpretation (Steiner, Arnold and Summerland, 1944) that reductions in natural enemy populations alone are responsible for pest increases.

Documentation of resurgence mechanisms must also include rigorous evaluation of multiple causes for resurgence. For example, although the proximate cause for resurgence may be the physiological changes in plants exposed to insecticide, the ultimate causation may be a combination of the plant-mediated enhancement of the pest species fecundity, coupled with an increase in ovipositional preference for insecticide-treated plants in conjunction with a reduction in natural enemy mortality. The latter changes, i.e. changes in fecundity or behaviour, may be the factors that need to be manipulated to reduce the occurrence of resurgence.

Because of the multifactorial nature of the phenomenon of resurgence, it is often difficult to distinguish between mechanisms. For example, direct physiological enhancement of fecundity, behavioural changes that lead to increased immigration or decreased emigration, and enhancement of host-plant quality, may all result in increased abundance on treated plants compared with untreated plants. Only close monitoring of herbivore population structure, movement and reproduction, with simultaneous plant tissue analysis can distinguish between these mechanisms.

We have argued that resistance alone cannot cause resurgence, and that short-term selection for resistant forms *alone* is not an example of resurgence. However, insecticide resistance may play a role in resurgence. Resistance and resurgence can be tightly coupled and may work synergistically. The occurrence of insecticide resistance should magnify the effects of resurgence, and resurgence should increase the rate at which genetically based resistance evolves.

It is clear that resurgence is a complex phenomenon. Furthermore, there appears to be no coherent body of data from which a researcher can draw, because much of the literature on this topic is scattered in articles dealing with other aspects of pest control, ecology, and insect–plant interactions. In addition, the primary objective of many studies in which resurgence is reported is the evaluation of an insecticide or the development of control technology. We urge that researchers go beyond simply reporting the occurrence of resurgences or documenting preconceived mechanisms for resurgences. There are a number of important issues that, if addressed, will help define and relate resurgence to population ecology, allowing resurgence to be viewed within a broader ecological context. Given that not all populations of resurgent species undergo resurgence and that resurgence does not occur in all habitats, either resurgent individuals must phenotypically or genotypically differ from non-resurgent individuals, or habitats in which resurgence occurs must differ from those in which no such change is observed. To determine whether one or both of these outcomes are truly prerequisites for resurgence, more data are needed than are currently available.

It has yet to be determined whether basic patterns in life-history parameters are common among resurgent species, or if commonalities in insecticide (acaricide)–target species–agroecosystem interactions exist. Similarly, it is not known why certain species in a given agroecosystem resurge, whereas others in the same habitat, at the same time, do not. Are certain groups of insects (e.g. leaf feeders vs. piercing sucking insects, Homoptera vs. Heteroptera, early-season vs. late-season feeders, etc.) more likely to undergo resurgence? Is resurgence more likely among target pest species that feed on crop plants rich in (detoxification enzyme-inducing) allelochemicals? Is there some commonality (with regard to type, mode of action, mode of entry, etc.) among those insecticides that typically cause resurgence? The answers to these and other questions will begin to define the true nature of resurgence and lead to better management of this problem.

Insecticide-induced pest resurgence, although widely occurring and detrimental to crop production, is accepted as a drawback to the use of pesticides (Metcalf, 1986). One reason why it is simply accepted is that, often, by the time that resurgence is observed in a crop situation,

it is too late to determine the mechanisms causing it. Prespray information is essential to determine what changes have occurred. Additionally, multiple changes often occur simultaneously, requiring extensive experimental studies to separate the effects of each factor. Some mechanisms, such as the interaction of resistance and resurgence, require multidisciplinary research which, although becoming more common, has in the past been infrequent. The dogma associated with the underlying mechanisms of resurgence is hard to ignore and thus much of the research on this phenomenon has reinforced the simplistic idea that reduction of natural enemies is the sole mechanism responsible for resurgence. It is necessary that extensive research, such as has been (and presumably is being) undertaken in systems such as the brown planthopper–rice system be extended to many different pest–crop systems in order to understand the complexity of the phenomenon of resurgence.

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