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# *Outbreaks of colony-forming pests in tri-trophic systems: consequences for pest control and the evolution of pesticide resistance*

*Vincent A. A. Jansen, Inst. of Evolutionary and Ecological Sciences, Section Theoretical Biology, P.O. Box 9516, NL-2300 RA Leiden, the Netherlands (present address: NERC Centre for Population Biology, Imperial College at Silwood Park, Ascot, U.K. SL5 7PY) and Maurice W. Sabelis, Dept of Pure and Applied Ecology, Univ. of Amsterdam, Kruislaan 320, NL-1098 SM Amsterdam, the Netherlands*

## Population dynamical explanations

Ironically, pesticide applications in agriculture may result in the opposite of the intended reduction of the pest population. Following an initial treatment, the target population may resurge rapidly to greater than pre-treatment levels. In addition, populations of non-target species that were previously far below economic thresholds may increase greatly after applications and develop into pests of economic importance. Pesticide-induced outbreaks have been reported for very different groups of phytophagous arthropods and for several types of pesticides (Ripper 1956, Barbosa and Schultz 1987, Penman and Chapman 1988, Roush and Tabashnik 1990). This raises the question whether there are general principles underlying these outbreaks.

One explanation for pesticide-induced outbreaks is that, for reasons rooted in their evolutionary past, phytophagous pest species are less susceptible to pesticides than their natural enemies. Hence, pesticide treatment will have a more drastic effect on natural enemies than on pests, resulting in outbreaks. However, this explanation ignores population dynamical effects on predator-prey balances. Furthermore, measurements of susceptibility to direct pesticide doses reveal that natural enemies are generally *not* more vulnerable to pesticides than their prey species (Hoy 1990) and may even show a trend towards lower predator susceptibility (Croft and Brown 1975). Thus, empirical support is lacking and (as we argue below) there is no good reason to expect that predatory arthropods would have been exposed to a narrower range of toxicants than their prey.

A more sound explanation follows from consideration of the coupled population dynamics of predator and prey (May 1985, May and Dobson 1986). After pesticide application the densities of phytophagous arthropods are reduced. As soon as the harmful effects of the pesticide abate, conditions are favourable for herbivores: they suffer less predation and possibly experience reduced competition. Hence the numbers of pests and potential secondary pests will increase at a rate close to their maximum growth rate.

Even if natural enemy densities are not directly affected, their densities will decrease after pesticide application since they are largely deprived of their main or only food source: phytophagous arthropods. Populations of predators can only increase after prey have attained sufficiently high densities. The increase in predator numbers therefore typically lags behind that of the pests. By the time the predators have returned to the pre-application densities, the pest populations have had the opportunity to increase unchecked for some time. The importance of food limitation to differential rates of recovery after pesticide application has been confirmed in a number of simulation studies (Tabashnik 1986 and 1990); even when the immediate mortality is similar for predator and prey, predator populations are more severely suppressed by pesticides than are pest populations (Waage et al. 1985).

Pesticide induced outbreaks are often considered as transient events (Ripper 1956); after pesticide use is stopped, the ecosystem is expected to recover and predator-prey balances to restore. However, this need not be the case. Persistent outbreaks can result from the simultaneous existence of two stable states; one in which the predator controls the pest, and another in which the predator exists at very low levels, or is absent, and the pest is not controlled. After a small disturbance the system will return to its original state. For larger disturbances, however, the system may stabilize at either state, depending

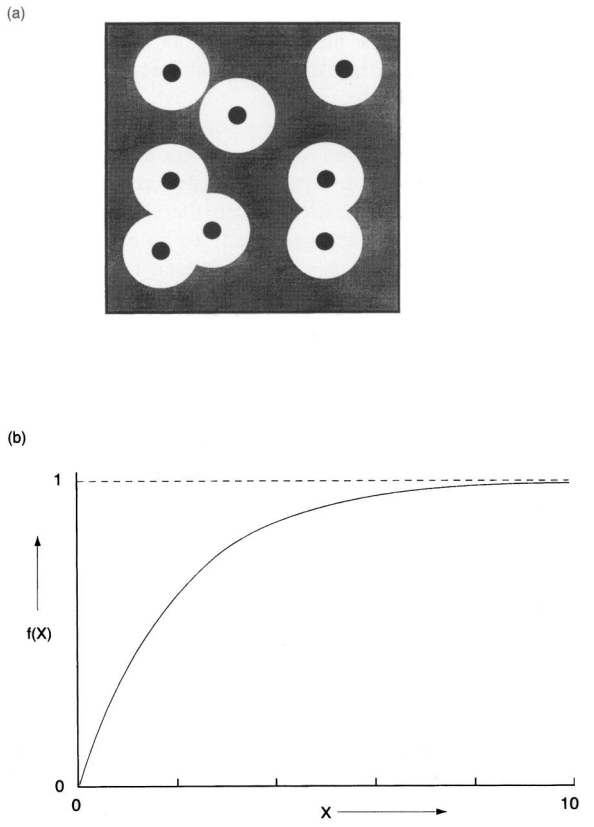


Fig. 1. (a) A number of patches spaced randomly. Each patch (black disk) has an attractive region (white disk). Attractive regions can overlap partly.
(b) The fraction of space filled by patches ($F(X)$) versus the density of patches ($X$) when each patch has an attractive region of $d_x = 1/2$. Patches are assumed to be placed randomly and to have an attractive region of size $d_x$. Without overlap $X$ patches would fill $d_x X$, as is the case for low $X$; for large densities the fraction of space filled goes asymptotically to 1. The probability for a dispersing individual to successfully locate and colonize a patch is $F(X)$ times a constant $a_x$. Assuming that the number of searching individuals is proportional to the number of patches, the total number of discovered patches is $a_x F(X)$ times the number of patches.

on the precise details of the disturbance. A reduction of both predator and prey densities can flip the system from one stable state to the other. Bistability is a rather well known phenomenon in predator-prey models in which either the prey growth is negative at low prey densities or the predators' functional response has a sigmoid form (Noy-Meir 1975, Southwood and Comins 1976, May 1977). The stable states then normally are steady states. It is perhaps less known that bistability can also occur in models in which some of the stable states are limit cycles, under conditions less strict then needed for bistable steady states. When an uncontrolled pest population exhibits cycles, pesticide application can reduce predator populations beyond recovery and cause the pest to exhibit repeated outbreaks (Godfray and Chan 1990).

Pest outbreaks have usually been explained by reference to predator-prey models in which changes in the plant population are assumed to occur on a much longer timescale. However, for colony forming pests the time scales of plants and colonies become so similar that plant dynamics no longer can be ignored. Models for colony-forming pests should therefore take the form of food chains with three trophic levels. Tri-trophic food chains generally tend to be bistable. This is independent of the precise model formulation, as can be shown by an analysis of the normal forms of tri-trophic models (Klebanoff and Hastings 1994) and has been demonstrated in various tri-trophic models (Jansen and Sabelis 1992, Jansen 1994, McCann and Yodzis 1995). In one of the stable states three species coexist while in the other stable state the third trophic level is absent and the first and the second trophic level exhibit sustained oscillations. Sufficiently large disturbances can bring about a change from one stable state to the other. Our main aim is to discuss the consequences of this bistable behaviour for the evolution of pesticide resistance, and we will briefly discuss population dynamical consequences for pest control, such as failure in the establishment of natural enemies and adverse effects of using resistant plant varieties on biological control.

## A simple tri-trophic model

The potential effect of pesticides on plant-inhabiting arthropod predator and prey populations is usually inferred from models ignoring the population dynamical interactions with plants. This approach is based on the assumption that the generation times of arthropods are smaller than those of plants by orders of magnitude. Changes in plant density will hardly be noticeable on the timescale of the arthropods' lifetime.

However, when the arthropod herbivores form colonies, this timescale argument no longer holds. What then matters is the characteristic timescale of the interaction between the herbivore colony and the plant, or between the predators and herbivores within a colony. Such timescales exceed the arthropod generation times and come close to the lifetime of leaves from a perennial plant or the generation times of annual plants. In these cases it is more appropriate to consider the tri-trophic interactions, by incorporating the dynamics of leaf abundance of perennials or the dynamics of annual plants in the model.

We will present a simple model for a pest that forms colonies. The variables in our model represent densities of patches, rather than of individuals. The variable $R$ represents the density of vacant plant patches, these should be thought of as leaves or clusters of leaves. Plant patches grow logistically with carrying capacity $c$. A herbivore that discovers a vacant plant patch can start a colony and thus transform this patch into a herbivore patch. The density of herbivore patches is described by $N$.

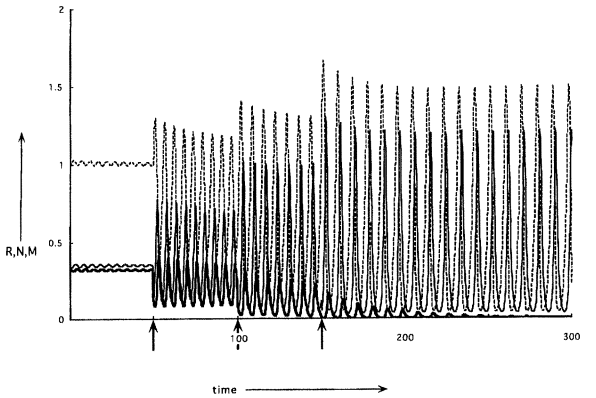


Fig. 2. A simulation run using the tri-trophic model presented in the text. The simulation starts in the neighbourhood of a stable three species equilibrium. At the positions of the arrows the herbivore and predator populations are both reduced by 60%. After the second spray the predators already decline over every cycle, the third spray reinforces this effect. Eventually, the predators disappear while the plants and herbivores show sustained oscillations. Dashed line: density of vacant plant patches, thin drawn line: density of herbivore patches, thick drawn line: density of patches with herbivores and predators. Parameters: $r=1$, $c=3$, $d_r=1$, $a_n=3$, $d_n=0.01$, $a_m=290$, $k=1$, $\nu=1$.

New herbivore patches are formed with rate $a_n N F(R)$ where $F(R)$ gives the fraction of space filled when the density of vacant plant patches is $R$ and $a_n$ is a proportionality constant (Fig. 1). Herbivore patches disappear when the herbivores have depleted all plant material in a patch (with rate $kN$) or are transformed in a patch with herbivores and predators when discovered by a predator. Analogously to the discovery of empty patches by herbivores, predators discover herbivore patches with rate $a_m M F(N)$. Here, $M$ represents the density of colonies with herbivores and predators, $a_m$ again is proportionality constant. Patches with herbivores and predators disappear with rate $\nu M$. The system of equations reads (Hogeweg and Hesper 1978):

$$\frac{dR}{dt} = rR\left(1 - \frac{R}{c}\right) - a_n N F(R)$$

$$\frac{dN}{dt} = a_n N F(R) - kN - a_m M F(N)$$

$$\frac{dM}{dt} = a_m M F(N) - \nu M,$$

where

$$F(X) = 1 - e^{-d_x X}.$$

The model presented above describes an undisturbed system. The effect of a pesticide treatment is modelled as a single, instantaneous reduction of herbivore and predator densities; thus their respective pesticide induced mortalities occur only at the moment of pesticide application. Between sprayings the pesticide does not affect the system and the population dynamics is described by the equations given above.

Tri-trophic models can have two simultaneously stable states, one in which three species can coexist and one in which the predators are absent. The disturbance caused by the application of pesticides can therefore cause the predators to decrease and eventually to go extinct, leaving the pest to show sustained outbreaks (Fig. 2). The crucial factor for bistability is that the two species limit cycle is stable against invasion of the predators unless large numbers are introduced. The loss of predators produced by the application of pesticides therefore is permanent.

Why this type of bistability is a common feature of tri-trophic systems can be easily understood: when few predators are introduced while plants and herbivores oscillate, these predators have to survive the troughs in herbivore density. With relatively short peaks in prey density the losses suffered during the troughs can not be made up for, and the predators will decrease over every cycle and eventually go extinct. When, however, sufficient predators are introduced they can effectively control the herbivore density and thus stabilize the herbivore-plant interaction. This can result in coexistence of plants, herbivores and predators. Pesticide applications can bring about the reverse: a permanent change from a state with plants, herbivores and predators to one with repeated herbivore outbreaks.

Admittedly, there is little empirical evidence that such bistable tri-trophic systems exist, but the theoretical arguments for their existence are very general. One possible example is the interaction between plants, spider mites and predatory mites. When not controlled by natural enemies or pesticides, spider mites can reach densities that lead to the defoliation of the plants they are living on. After defoliation the densities drop dramatically (Burnett 1979). When the plants regenerate new leaves, such outbreaks are repeated. A small number of natural enemies of the spider mites may not survive in such an environment (Burnett 1979). Yet, a state where the three trophic levels can coexist is feasible (Van de Klashorst et al. 1992).

## Evolutionary consequences

Resurging pests will trigger more pesticide treatments by beleagured farmers, which in turn leads to strong selection for resistance. This is true for both the target pest species and other species that happen to live in the treated areas. In this way many phytophagous arthropods have developed resistance against pesticides. However, the development of resistance in field populations of arthropod predators is relatively scarce and seems to develop much more slowly than among their prey (Croft and Brown

1975, Croft and Morse 1979, Hoy 1990, Tabashnik 1990). Of the 447 reported cases of resistance, less than 3% were predators and parasites (Georghiou 1986).

Although there exist several hypotheses that explain the slower development of pesticide resistance in predators (see May (1985) and Mallet (1989) for reviews) only the mechanism of dilution of the resistance gene population by immigration and removal of susceptible genes (Comins 1977, May 1985, Van der Laan and Hogeweg 1988) provides a sound mechanistic explanation. This hypothesis can explain the slower development of predator resistance when immigration and mixing are sufficiently high and resistance genes are sufficiently recessive. However, the condition of mixing will rarely be met in small colony forming arthropods since their populations often consist of local mating groups. For example, many species of mites (Sabelis and Nagelkerke 1988, 1993, Sabelis 1990) and parasitoids (Godfray 1994, Hardy 1994) form colonies consisting of the progeny of one or a few founding mothers, causing sibmating to be more likely than under random mating. Under these circumstances the progeny of invading individuals will mostly encounter sibs and their genes will hardly mix with those of the resident population. Yet, predatory mites develop resistance slower than their herbivorous prey. In these populations another mechanism must operate that can slow down the development of resistance.

Such a mechanism can be most easily demonstrated in systems that are bistable. As shown, many herbivore-predator systems in which the herbivore forms colonies are intrinsically bistable. When a resistant herbivore invades a regularly sprayed area from a larger population outside, it will be quickly selected for and replace the resident population. The dynamical properties of the resistant herbivore's population will be, up to the change in mortality due to pesticides, quite similar to those of the susceptible population, i.e. the resistant population is also capable of showing sustained outbreaks.

Let us next follow the fate of a resistant predator that migrates into the area under pesticide treatment. Repeated sprayings will sooner or later bring the system in a state in which the herbivores show sustained outbreaks and the predators on average decrease. Although this resistant predator will find unfavourable food conditions in its newly chosen habitat, it is still better off than predators that do not carry the resistance gene. In due time the resistance gene will replace the predators' susceptible gene. However, the resistant predator population still has to cope with the troughs in herbivore density and may decrease from outbreak to outbreak. The crucial point now is that when a certain fraction from both the predator and prey population is sent back into the surrounding population, the number of exported resistant herbivores will be larger than the number of exported resistant predators. This is a straightforward consequence of the fact that after spraying the herbivores fare better than the predators. Thus the resistant herbivores will have a considerably greater effect on the resistance-gene frequency of the background populations than the resistant predators.

The rate with which the resistance gene will be selected in areas under treatment largely depends on the initial frequencies, i.e., the frequency of the resistance gene in the background population. The mechanism described above gives predator genes a systematic disadvantage in increasing their frequencies in the background population. This generally slows down the development of resistance in predator populations relative to populations of their prey.

## Perspectives for pest control

The existence of multiple stable states easily goes unnoticed in biological systems. Whenever there is a (semi-) permanent change there usually is a multitude of potential reasons for it (see Alam et al. (1971) for a fine example); hence there is little need for yet another explanation that is produced out of the theoretician's hat. However, multiple stable states can be a straightforward consequence of very plausible assumptions. Tri-trophic models illustrate this point: when the timescale of herbivore colony dynamics is of the same order as the plant's generation time, there is the possibility of herbivore-plant limit cycles. Predators' life is difficult when prey densities are low over longer time periods. Hence, predators introduced in small quantities will not survive these fluctuations, whereas predators introduced in large quantities can stabilize the plant-herbivore interaction and establish themselves permanently. Using pesticides in such a system of herbivore and predator can result in eradication of natural enemies.

Pest management requires a proper understanding of the population dynamics of pests and their natural enemies. Repeated use of pesticides in a system of herbivores and their predators can deteriorate the food conditions to such an extent that predators go extinct. This is not only due to mortality from pesticide action, but also to the impoverished food conditions for the resistant predators surviving pesticide spraying. The same holds for pesticides that are less harmful to natural enemies, since these will also cause a reduction of prey densities. For evaluation of the effects of pesticides on ecosystems it is not sufficient to measure the susceptibility of predators, because this ignores the population processes after spraying. In addition, predator survival should be estimated or measured in field trials. A similar argument holds for the introduction of genetically improved predators: these will only be successful if the improved predator finds enough prey to survive on. This might explain the variable outcomes of field releases of resistant predators (Hoy 1985, 1990). The existence of multiple stable states in tri-trophic systems also has implications for the use of plants that are genetically modified to reduce herbivore feeding: although the use of resistant plants has

no direct impact on predators, it can modify the supply of phytophagous prey so as to cause predator extinction and thereby provoke pest outbreaks. A better understanding of the population dynamics of systems involving more than two trophic levels might prevent the failure of costly research programmes on plant resistance breeding and biological control.

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