Ecological Applications, 12(6), 2002, pp. 1872–1882

THE RISKS OF BIOCONTROL: TRANSIENT IMPACTS AND MINIMUM NONTARGET DENSITIES

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Abstract. The biocontrol of insect pests may pose a risk to native insects if the biocontrol agent attacks nontarget species. Potential biocontrol agents are screened before release to determine their acceptance of nontarget species and the suitability of nontarget species for their development. Here we show that, even though a biocontrol agent has very low acceptance of a nontarget species, it may nonetheless have a large impact on the nontarget population. This impact does not require the nontarget species to be a suitable prey capable of supporting the biocontrol agent population, but instead may be a transient impact that occurs soon after the agent is released. Because the population of biocontrol agents is likely to increase rapidly in response to the high density of its target pest, the resulting high density of the agent population may dominate its low acceptance of the nontarget species, causing a strong decline or even local extirpation of the nontarget. We demonstrate this possibility using models of host–parasitoid dynamics that incorporate a broad range of assumptions about the life histories of hosts and parasitoids, and that demonstrate how various common aspects of host–parasitoid biology are likely to reduce this risk considerably. The predictions of the models are reasonably approximated with a simple formula, which potentially provides a simple method for assessing the risk of transient impacts, but which should only be applied loosely (in a qualitative manner) and in the context of a fuller understanding of other factors affecting risk in the system in question.

Key words: biocontrol, nontarget risks; biological control; extinction risk; host–parasitoid dynamics; host-range testing; insect biocontrol; nontarget effect; parasitoids as biocontrol agents; population models and biological control.

INTRODUCTION

There has been much debate about the potential impact of biocontrol on nontarget species (Po-Yung 1988, Howarth 1991, 1992, Civeyrel and Simberloff 1996, Simberloff and Stiling 1996*a*, *b*, Strong 1997). Many examples seem to show that nontarget species suffer a negative impact from biocontrol agents, although the quality of evidence varies from anecdotal to relatively quantitative (Howarth 1983, Bennett 1993, Barratt et al. 1998, Follett et al. 2000, Stiling and Simberloff 2000), and the paucity of detailed studies makes it difficult to assess the frequency and severity of nontarget effects (Lynch et al. 2000, Lynch and Thomas 2000). While some authors believe that biocontrol has caused nontarget extinction in the past, and that extinction continues to be a risk (Howarth 1983, Simberloff and Stiling 1996*a*, *b*, Strong 1997), many are more skeptical, critical of the quality of the evidence in general, and believe extinction or serious population reduction is impossible given the precautions of modern-day biocontrol (e.g., Van Lenteren and Martin 1999).

Here we investigate the potential risk from parasitoid biocontrol agents to nontarget hosts even when the nontarget hosts are much less preferred by and suitable for the biocontrol agent. We focus on scenarios where host-range testing, interpreted in isolation, would suggest low risk, but ecological considerations may imply otherwise. Specifically, we consider cases in which nontarget hosts cannot support the agent population in the long term, but instead suffer during a transient period of ''spill-over'' parasitism shortly after the introduction of the agent. This period does not last long, but such transient effects may cause extinctions, such as that of *Bessa remota* in Fiji (Tothill et al. 1930, Roberts 1986, Howarth 1991). Extinction is possible because the target host may be very common, so transient peaks in biocontrol-agent abundance may be very large—large enough to compensate for a low propensity for the agent to locate and accept the nontarget.

The observation that large peaks in agent abundance after introductions are likely to impact on nontargets is not new, and there are a number of examples from weed biocontrol (Harris 1988, 1990). Weed biocontrol

Manuscript received 31 August 2000; revised and accepted 18 February 2002; final version received 27 March 2002.
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agents have been known to damage crop plants, including those unrelated to the target weed, when at high densities soon after introduction, particularly when host-plant populations have collapsed. Examples include two agents introduced against prickly pear in the 1920s in Australia, *Cactoblastis cactorum* and *Chelinidea tabulata* (Harris 1988), which both fed on various crops (particularly melons); *Teleonemia scrupulosa*, an agent introduced against *Lantana camara* in Uganda in 1963, but which fed on sesame (Davies and Greathead 1967, Harris 1988, Pemberton 2000); *Zygogramma bicolorata*, which was introduced against *Parthenium hysterophorus* in India in 1984, but which fed on sunflower during the initial years of establishment (McFadyen 1998); and *Trichilogaster acaciaelongifoliae*, released in South Africa in 1982 against *Acacia longifolia*, but which spilled over to *Acacia melanoxylon* (a species used commercially) in areas where stands of the two plant species overlapped and there was competition between ovipositing females on the preferred host (Dennill et al. 1993, 1999). Native species may also have been affected by such spillovers—this is known to be the case for *Teleonemia scrupulosa* (Pemberton 2000), for control agents of purple loosestrife *Hylobius transversovittatus* (Blossey et al. 1994) and *Galerucella calmariensis* (Corrigan et al. 1998), and for an agent of water hyacinth in the United States, *Neochetina eichhorniae*, which has caused minor damage to ornamental *Canna* spp. and to *Pontederia* spp. when weed populations collapsed (Center 1982, Harris 1988). Critical to many of these cases were huge peaks in biocontrol agent numbers, a collapse in numbers of the target pest, and a degree of transient expansion of the host range of the biocontrol agents due to unusually high levels of competition among the agent insects. It is quite likely that such phenomena are general in biocontrol beyond weed control, where damage to nontarget crops has been the principle reason these phenomena have been noticed.

Here we take a formal look at the risk factors associated with transient dynamics occurring shortly after biocontrol agents are introduced. We focus on the use of parasitoids as biocontrol agents, because these are common agents whose possible transient effects have not often been considered. Although there is less evidence for transient effects in insect biocontrol than in weed biocontrol, we suspect that this does not reflect the true risks of transient effects in insect biocontrol. Whether or not complete local extirpation via transient impacts is likely in insect biocontrol, the potential for serious, population-level nontarget impacts that are difficult to predict from pre-release host-range testing deserves investigation. Moreover, the possible instability of host–parasitoid population dynamics may make local extirpation more likely (Murdoch 1991, 1994, Murdoch et al. 1985, Taylor 1991).

Although we shall not directly address large-scale population changes such as global extinction, it is important to consider our analysis of local effects in a broader context. Local populations of target and agent may be stable and persistent, or they may be unstable, with the global persistence of target and agent being ensured by immigration and emigration (i.e., metapopulation dynamics; Hanski and Gilpin 1991). In either case, transient impacts on nontargets will influence their broad-scale abundance. Although we investigate the conditions in which the probability of local extirpation of a nontarget will be high, we do not argue that complete extinction over large areas is likely.

Modeling the effects on nontargets, which involves the population dynamics of agent, target, and nontarget species, could push us towards quite complex models. However, using complex models with a complex set of solutions and predictions might not be as useful as one might expect, because complex models can be limited in scope, and the data required to parameterize complex models are often impractical or impossible to collect. Furthermore, this approach might obscure some quite simple and transparent take-home messages that can be shown by simple models. Therefore, we focus on simple models for the dynamics of biocontrol agent, target, and nontarget.

We use these simple models to test the following formula for predicting transient nontarget effects:

1) Estimate the minimum density of the target species that will occur following introduction of the biocontrol agent.
2) Calculate the difference between the target species density before agent introduction and the minimum density following introduction, and estimate the number of agents that would be produced if this number of target individuals were parasitized.
3) Calculate how many nontargets would be killed if the nontarget population were attacked by all of these agents in the same generation. This approximates the lowest transient density to which the agent will depress the nontarget.

This formula does not depend on detailed knowledge of the population dynamics of the system in question. It is simply the prediction one would make if a biocontrol program resulted in an instantaneous (one generation) explosion of the biocontrol agent population, and an equally sudden collapse of the target pest. We show that, somewhat surprisingly, this formula also often works when it takes multiple generations for the agent population to build to sufficient densities to suppress the target population. Thus, we have a simple rule of thumb for estimating the risk of transient nontarget effects.

Below, we first analyze a very simple model of biocontrol agent, target, and nontarget dynamics to demonstrate the application of our formula for estimating nontarget effects. Then we present a collection of more complicated models incorporating differing characteristics of agent, target, and nontarget interactions, show-

ing when this formula works, when it must be modified, and when it fails. While any true test of the formula must involve real studies on nontarget effects, the required studies do not yet exist. Therefore, we use the collection of models to establish the plausible utility of the formula.

The Simplest Case and Approximate Solution

To illustrate the formula for predicting transient nontarget effects, we begin with a simple discrete-time model for three species derived from the two-species Nicholson-Bailey model (Hassell 1978). Both the target and nontarget experience self-regulation (i.e., intraspecific competition), but not interspecific competition. The agent attacks hosts with a type I functional response, so that the proportion of hosts escaping attack is given by the zero term of a Poisson (random) distribution.

These assumptions give the following model:

$$P_{t+1} = c_H H_t \exp\{r_H(1 - H_t/K_H)\}(1 - \exp\{-a_H P_t\}) + c_N N_t \exp\{r_N(1 - N_t/K_N)\}(1 - \exp\{-a_N P_t\}) \quad (1a)$$

$$H_{t+1} = H_t \exp\{r_H(1 - H_t/K_H) - a_H P_t\} \quad (1b)$$

$$N_{t+1} = N_t \exp\{r_N(1 - N_t/K_N) - a_N P_t\}. \quad (1c)$$

Here, P_t is the density of the biocontrol agent in generation t, and H_t and N_t are the target and nontarget host densities. For the target and nontarget, respectively, r_H and r_N are the intrinsic rates of increase, a_H and a_N are the searching efficiencies of the biocontrol agent, c_H and c_N give the conversion of parasitized hosts into the next generation of agents, and K_H and K_N are the carrying capacities. In general, we assume that the searching efficiency of the agent for the target is greater than for the nontarget, $a_H > a_N$; this constraint reflects our intention to investigate cases where nontarget risk will not be revealed by host-range testing. Nontarget carrying capacity is also kept low, as we assume that the target is more common than the nontarget before the introduction of the biocontrol agent, so $K_H > K_N$ (in fact, $K_H = 10K_N$ for all our models). In essence, we have deliberately excluded cases in which the nontarget alone supports breeding populations of the agent. Note that, given this, the exact carrying capacity K_N, and conversion rate c_N for the nontarget were both found to have almost no impact on the degree of nontarget effect, and we do not alter these parameters in the investigations below. Nonetheless, as this analysis is not intended to be universal, we do not claim that the level of these parameters will never have an impact on nontarget effects.

This, and subsequent models, were simulated using simple spreadsheet methods (on Microsoft Excel) to iterate the difference equations, and macros where necessary to repeat simulations a large number of times to obtain graphs of minimum nontarget density against other parameter ranges. The first 45 generations after introduction of the agent were simulated. Results were not sensitive to extending the length of the simulation, because in this and all subsequent cases (unless stated otherwise) the target and agent had by this point either gone extinct, reached a coexistence equilibrium, or entered a stable limit cycle with peaks and troughs equal to or less extreme than those of the transient conditions following introduction.

We used this basic model to consider the application of the approximation formula to cases in which the biocontrol agent causes the (local) extinction or near extinction of the target. The formula for predicting the transient nontarget effect for the case $c_H = 1$ (that of a solitary parasitoid in situations of full suitability of the targets as hosts) is applied as follows:

1) The density of the target before the introduction of the agent is K_H. As the target is driven to extinction or near extinction locally, the minimum density of the target is zero.
2) The density of the agent that would result if all targets were parasitized is just the carrying capacity for the targets, K_H, since each parasitized target gives rise to one agent.
3) The proportion of nontargets that would survive the simultaneous attack by the K_H agents is $\exp(-a_N K_H)$. Therefore, the predicted minimum density to which the nontarget is depressed, N_{min}, is

$$N_{min} = K_N \exp(-a_N K_H). \quad (2)$$

Thus, the greater the initial density of targets, K_H, and the greater the searching efficiency of agents for the nontarget, a_N, the greater the depression of the nontarget. Furthermore, K_H and a_N combine multiplicatively. If, for example, the searching efficiency for the nontarget were 1/100th of the searching efficiency for the target, this low searching efficiency would be countered by a target species abundance 100 times that of the nontarget.

The above formula, Eq. 2, is appropriate for the basic model of a target–agent–nontarget interaction because this model is unstable, with the target going (locally) extinct relatively quickly (followed by the agent parasitoid). Although the approximation was written to describe the instantaneous attack on all nontargets in the same generation, we find that it predicts the minimum nontarget density even when the agent population builds, and the nontarget population declines, over multiple generations (Fig. 1). Altering the searching efficiency of the agent for the target, a_N, leads to a change in the time scale at which events occur, but does not greatly alter N_{min}. When plotted against the carrying capacity of the target, K_H, or the searching efficiency for the nontarget, a_N, the minimum nontarget density is described well by Eq. 2, except at very low values of K_H (Fig. 2)

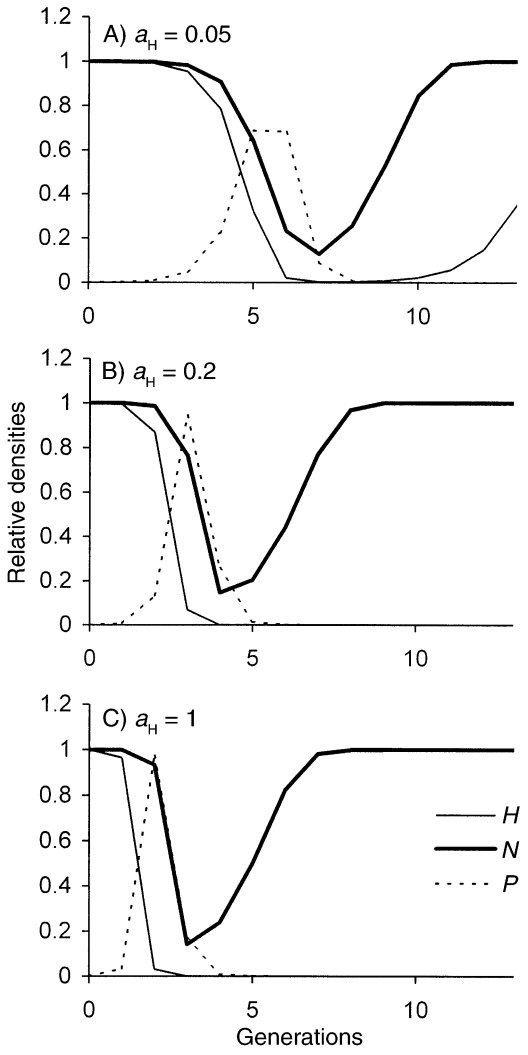

FIG. 1. Transient dynamics of a modeled non-persistent target–agent interaction, and the nontarget impact (Eq. 1), for different a_H, where a = searching efficiencies of the agent, on H, the target host. Other parameters are $a_N = 0.02$, $r_H = r_N = 1$, $K_N = 10$, and $K_H = 100$ (where N = the nontarget host, r = the intrinsic rate of increase, and K = carrying capacity). Results are shown as relative densities: H (relative density at time t) = H_t/K_H, N (relative density of non-target host) = N_t/K_N, and P (relative density of biocontrol agent) = P_t/K_H.

or of a_H (not shown). In these cases, because the density of the target or the searching efficiency of the agent for the target is so low, the agent is only just able to sustain itself on the target. Furthermore, the agent–target equilibrium is monotonically stable, and populations quickly converge to equilibrium and persist, rather than undergoing large fluctuations and local extinction. In these cases, where stability and persistence is achieved by target self-regulation, the approximation may underestimate the minimum nontarget density, thereby overestimating the impact on the nontarget.

The formula can be expanded to include parasitism functions other than that used in the Nicholson-Bailey model:

$$N_{min} = K_N f(c_H d K_H) \quad (3)$$

where $f()$ is any function determining the proportion of hosts escaping parasitism in relation to agent density, and $c_H d K_H$ is a term calculating the peak density of agents in the nontarget habitat. We have added the term d to highlight the possibility of a spatial separation between target and nontarget areas. Parameter d can be interpreted as the relative density of agents in nontarget habitats, compared to target habitats, and so portrays the degree of overlap and/or dispersal of the agent between the populations. Because the effect of simple spatial separation is obvious, we assume for simplicity that target and nontarget populations overlap fully ($d = 1$) in the rest of the analyses.

The more general form of the approximation formula (Eq. 3) can be tested by altering the function $f()$, rather than by changing c_H or d. By explicitly stating Eq. 3 in general terms for any function $f()$, we emphasize that in real host–parasitoid interactions this term is very unlikely to be as simple as that of the Nicholson-Bailey model, so some information on the nature of the parasitism function is necessary to make predictions. Changes to the parasitism function can be dealt with by adapting the approximation accordingly. As we

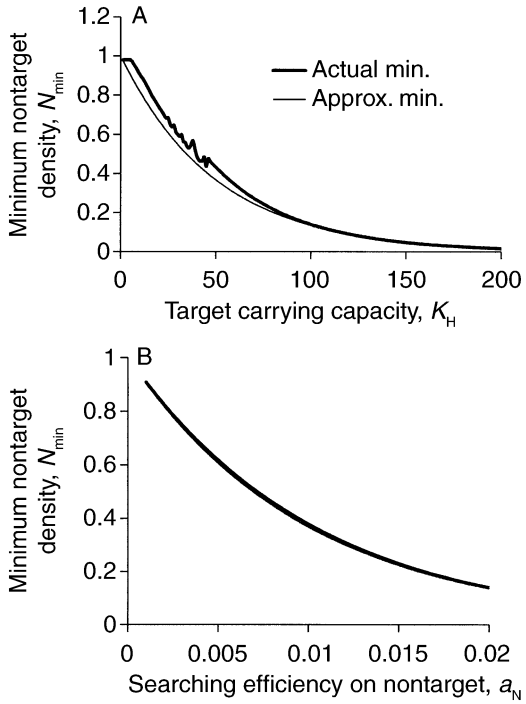

FIG. 2. Performance of the approximation formula for minimum nontarget density (relative to carrying capacity) over changes in two model parameters, (A) carrying capacity of target K_H and (B) searching efficiency on nontarget a_N. Other parameters are $a_H = 0.2$, $a_N = 0.02$, $r_H = r_N = 1$, $K_N = 10$, and $K_H = 100$. See Fig. 1 for definitions of parameters.

show below, in the complete absence of information about the parasitism function, the use of Eq. 2 from the Nicholson-Bailey model generally gives the worst case approximation, because it is likely to overestimate the transient impact of the agent on the nontarget species.

Parasitoid Interference

We examined the effects of parasitoid interference and similar processes (such as pseudo-interference; Hassell and May 1973, 1974, Free et al. 1977, May 1978, Walde and Murdoch 1988, Pacala et al. 1990, Pacala and Hassell 1991, Jones et al. 1993, Taylor 1993) using the following function (Hassell and Varley 1969, Hassell 1978):

$$f(P) = \exp(-aP^{(1-m)}). \quad (4)$$

When $m = 0$, this gives the basic Nicholson-Bailey case of random searching. As m increases from zero, increasing interference among parasitoids takes place, causing a diminishing increase in the risk of a host being attacked with increasing parasitoid density. The corresponding model for target, nontarget, and agent population dynamics is obtained by replacing terms of the form $\exp(-aP)$ in Eq. 1 with $\exp(-aP^{(1-m)})$. From Eq. 3, the formula to approximate nontarget effects when the target is driven to extinction is

$$N_{\min} = K_N \exp(-a_N K_H^{(1-m)}). \quad (5)$$

Fig. 3A shows that raising the level of interference (increasing m) decreases the impact of agent on nontarget. Furthermore, the approximation formula (Eq. 5) works well. Note that with increasing m, the agent–target dynamics become stable, with the agent suppressing the target to a new low equilibrium (Fig. 3B). Therefore, the success of Eq. 5 does not break down when the agent–target interaction is highly stable, even though the approximation was derived under the assumption that the target was driven extinct. Of course, the formula could be modified, using the true minimum density of the target species rather than zero (extinction). But estimating the minimum density of the target may be difficult, and the marginal improvement of the approximation formula would be small provided the equilibrium density of the target after agent introduction is low.

Fig. 3B also serves to illustrate the relative importance of long-term nontarget impacts caused by a continuous flow of agents from the agent–target interaction to parasitize the nontarget. In this case, because biocontrol is successful, the equilibrium level of the agent population is low, so the long-term impact is minimal. In general the long-term impact will depend on the equilibrium level of the agent and its searching efficiency for the nontarget, both of which should be low in successful, specific biocontrol agents. Also, because equilibrium levels are generally inversely proportional to searching efficiencies (when the agent is at least partially successful at depressing its host), the searching efficiency of the agent for the nontarget relative to that for the target should be a good predictor of long-term nontarget impact. In contrast, for transient risks, searching efficiency for the target may not be a key factor, because the importance of the searching efficiency may be overwhelmed when the population size of the target is much greater than that for the nontarget.

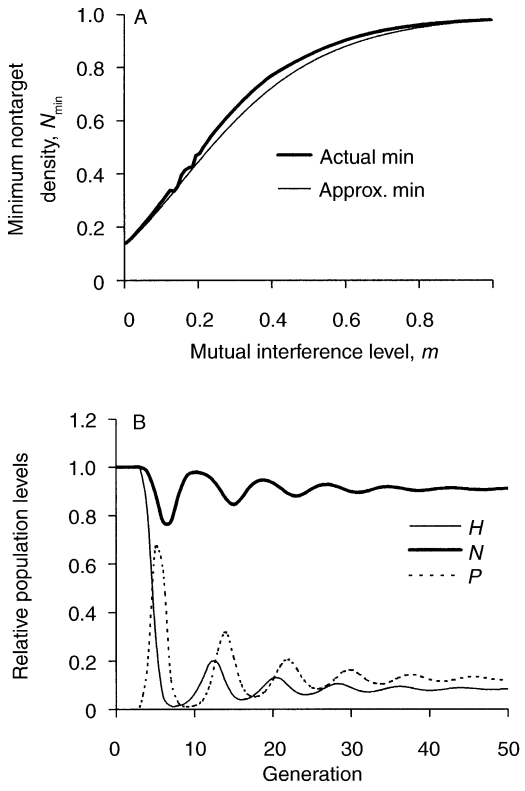

Fig. 3. The effect of mutual interference on minimum nontarget densities (relative to carrying capacity). (A) Performance of the adapted approximation (Eq. 5) over changes in the level of mutual interference. (B) An example of a stable scenario possible with mutual interference ($m = 0.4$), for which the approximation also works well. Other parameters are $a_H = 0.2$, $a_N = 0.02$, $r_H = r_N = 1$, $K_N = 10$, and $K_H = 100$. See Fig. 1 for definitions of parameters.

A mention should be made of physical refuges in which target and nontarget hosts are safe from parasitism, because refuges are considered important factors in host–parasitoid ecology (Hawkins and Gross 1992, Hawkins et al. 1993, Hochberg and Hawkins 1994). Depending on the details of entry and exit from a refuge, the dynamical consequences of a refuge can be very similar to mutual interference (Hassell 1978, Hochberg and Hawkins 1994, Lynch et al. 1998). Assuming a fixed proportion of the nontargets are in a refuge, by adapting the parasitism function $f()$ appropriately (see Hassell 1978), the impact of the biocontrol agent on nontarget density is well predicted by the approximation (results not shown).

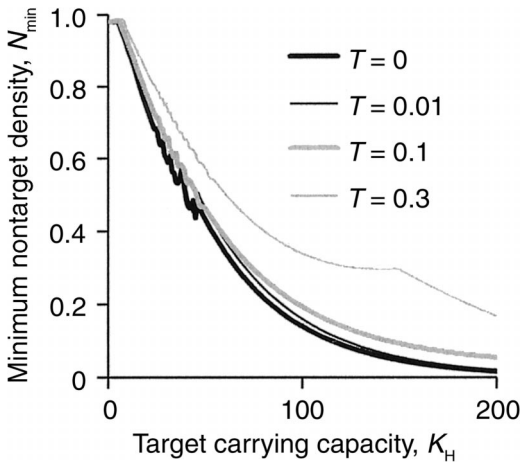

FIG. 4. The effect of a type II functional response on minimum nontarget densities. T is the handling time of the agent expressed as a proportion of its active life span (or the phenomenological equivalent). Higher T represents a more extreme functional response. Low, biologically reasonable values of T do not cause much deviation from the approximation. Other parameters are $a_H = 0.2$, $a_N = 0.02$, $r_H = r_N = 1$, $K_N = 10$, and $K_H = 100$. See Fig. 1 for definitions of parameters.

TYPE II FUNCTIONAL RESPONSE

When the searching efficiency of a parasitoid decreases with increasing host density, a type II functional response occurs (Hassell 1978, Begon et al. 1996). Type II functional responses can be caused by the time it takes for parasitoids to handle hosts, by the limitation of parasitoid egg numbers, or by egg maturation rates. Type II functional responses are common features of host–parasitoid population biology (Hassell 1978). How do type II functional responses influence transient nontarget effects?

When the biocontrol agent has a type II functional response on both target and nontarget, the proportions of target and nontarget hosts that are parasitized are, respectively,

$$f_H(P, H, N) = \exp\{-a_H P/(1 + T(a_H H + a_N N))\} \quad (6a)$$

$$f_N(P, H, N) = \exp\{-a_N P/(1 + T(a_H H + a_N N))\}. \quad (6b)$$

Parameter T can be thought of as the proportion of the active life span of a parasitoid spent handling one host and consequently not searching for new hosts, although the use of this parameter does not necessarily imply a handling-time-based mechanism for the functional response. We assume that equal amounts of time are spent on individual hosts of the target and nontarget species, but due both to the differing densities and searching efficiencies, the total time spent handling target individuals will be much larger. Thus the target population density will be the main factor determining the rate of saturation of the functional response. The corresponding population model is obtained by replacing $\exp(-a_H P)$ and $\exp(-a_N P)$ in Eq. 1 with f_H and f_N above.

Fig. 4 shows that T needs to be >0.1 before the nontarget effects under a type II functional response differ much from those obtained under a type I functional response. When handling times are this long, taking up major fractions of parasitoid life span, the minimum nontarget densities are elevated. While functional responses this extreme based on a handling-time mechanism are very rare (Hassell 1978), other mechanisms may occasionally produce relationships sufficiently severe to make minimum densities deviate from the approximation. The approximation is still likely to be accurate in the majority of cases, though, and again at least reveals the worst-case scenario.

ORDER OF PARASITISM

So far, we have assumed parasitism occurs at the same time as, or after, host density dependence. It is important to establish whether our basic result still holds when parasitism occurs first, as is likely to be the case for egg parasitoids such as *Trichogramma* spp. The Nicholson-Bailey model for agent–target–nontarget dynamics (Eq. 1) can be reformulated to take account of such a situation:

$$P_{t+1} = c_H H_t(1 - \exp\{-a_H P_t\}) + c_N N_t(1 - \exp\{-a_H P_t\}) \quad (7a)$$

$$H_{t+1} = H_t \exp\{r_H - r_H(H_t \exp\{-a_H P_t\}/K_H) - a_H P_t\} \quad (7b)$$

$$N_{t+1} = N_t \exp\{r_N - r_N(N_t \exp\{-a_N P_t\}/K_N) - a_N P_t\}. \quad (7c)$$

The target and nontarget densities are now given in terms of egg abundance in each generation. The term for density dependence has been adapted such that only those hosts surviving parasitism as eggs or early-instar larvae go on to contribute to competition. Therefore, H_t in the term calculating mortality from density dependence in Eq. 1b has been replaced by $H_t \exp(-a_H P_t)$. Also, because parasitoids now complete their development on host eggs, they are not affected by density dependence, so competitive mortality has been removed from the parasitoid equation.

Applying the formula for predicting nontarget effects when the target is driven to very low densities or extinction,

$$N_{min} = K_N \exp(-a_N c_H K_H) \times \exp(r_N - r_N \exp\{-a_N c_H K_H\}). \quad (8)$$

The first part of this expression is the same as that obtained previously (Eq. 3), while the second part accounts for the density-dependent rebound in the population following the removal of eggs before density dependence. The larger the population growth rate of the nontarget, the greater is this rebound effect, leading to less of an impact on the nontarget by the biocontrol agent.

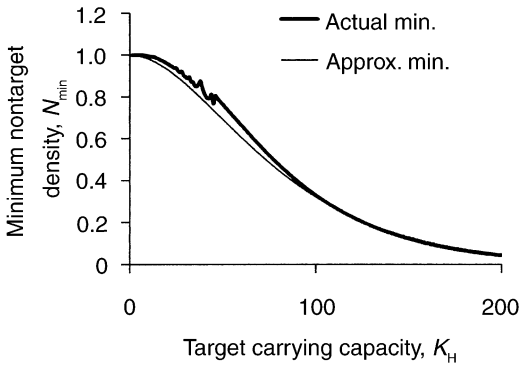

FIG. 5. The effect of parasitism of eggs or early instar larvae (before host density-dependent competition) on the target and nontarget hosts. A new approximation (Eq. 8), adapted to this situation, performs well. Other parameters are $a_H = 0.2$, $a_N = 0.02$, $r_H = r_N = 1$, $K_N = 10$, and $K_H = 100$. See Fig. 1 for definitions of parameters.

The effectiveness of this adapted approximation is illustrated in Fig. 5. Early parasitism appears to reduce the nontarget effect considerably. For most values of K_H in our example, minimum densities are around twice as high as in the previous model (Fig. 2). This means that, all other things being equal, egg parasitoids or early larval parasitoids are less likely to produce nontarget effects than agents attacking later stages. The smaller size, and hence lower dispersal abilities and searching efficiencies, of egg parasitoids will likely add to this result. However, because we are dealing with host densities in terms of eggs, it is worth remembering that the target-host carrying capacity, K_H, is measured in terms of the number of eggs, not adults. Therefore, it will take a much larger numerical value, and this will act to increase the measured effect on the nontarget. Consequently, we have no strong expectations about whether egg parasitoids produce smaller or larger nontarget effects than parasitoids of other stages.

POPULATION REGULATION OF THE NONTARGET

As we have seen above, parasitism of eggs or early larvae allows the nontarget species to absorb the mortality caused by the agent via compensating density dependence later in its life cycle. Other factors may alter the nature of impact absorption by the nontarget in a similar way. Thus, the mechanisms of population regulation for the nontarget will probably have an effect on the level of nontarget impact.

Returning to the original model (Eq. 1), the influence of the population growth rate of the nontarget can be examined by increasing the nontarget's intrinsic rate of increase, r_N, above that of the target r_H (Fig. 6A). For higher intrinsic rates of increase, the minimum density that the nontarget experiences is higher, indicating a smaller impact on the nontarget. This occurs because, when the biocontrol-agent population increases for multiple generations, the successive impacts on the nontarget each generation are mitigated in part by the nontarget's greater intrinsic rate of increase, resulting in a greater nontarget population density in each successive generation. However, note that our analysis only extends to intrinsic rates of increase of $r_N = 2.5$ (i.e., a net reproductive rate of 12.18), because beyond this point the single populations of the nontarget are unstable (and soon even chaotic), making the quantification of impact difficult. Furthermore, such chaotic behavior resulting from large r values is also believed to be rare in nature (Hassell et al. 1976).

We also explored the form of intraspecific density dependence affecting the target and nontarget species. The relationship between the per capita population growth rate and the density of a population is the defining character of intraspecific density dependence (Begon et al. 1996). Severe relationships, where the per capita population growth rate decreases quickly with density, are often described as ''scramble competition,'' while more gradual changes are usually referred to as ''contest competition'' (Begon et al. 1996). A range of forms of competition can be modeled in host–parasitoid interactions with a theta-Ricker function (Bernstein 1986), where the proportion of hosts surviving competition is given by $\exp(-r(H_t/K)^\theta)$. The parameter θ governs the type of competition, with large

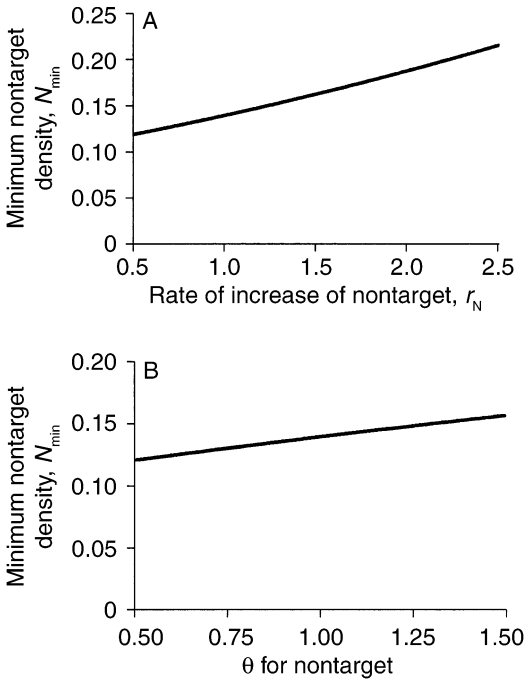

FIG. 6. The effect of (A) population growth rate and (B) form of competition in the nontarget host on minimum nontarget densities. High values of r (intrinsic rate of increase) and of θ (a parameter determining the form of competition) imply higher minima. Thus, both high growth rates and scramble competition allow nontarget populations to absorb the impact of parasitism, thereby reducing the nontarget effect. Other parameters are $a_H = 0.2$, $a_N = 0.02$, $r_H = r_N = 1$, and $K_N = 10$. See Fig. 1 for definitions of parameters.

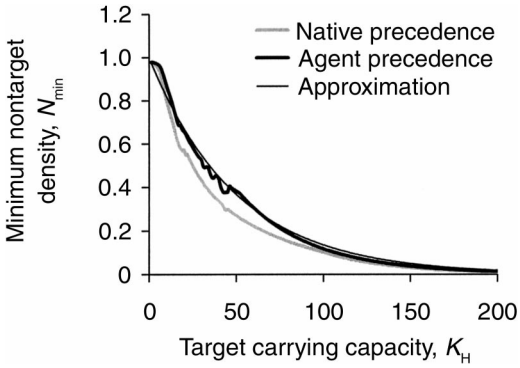

FIG. 7. The performance of the original approximation (Eq. 2) where the nontarget host is regulated by a specialist parasitoid. If the introduced parasitoid has precedence over the native parasitoid in multiply parasitized hosts (agent precedence), the approximation works well, whereas in the reverse situation (native precedence) minima are lower than the approximation suggests.

values of theta corresponding to more scramble-like competition.

Scramble competition ($\theta > 1$) always reduces the nontarget impact, whereas contest competition ($\theta < 1$) magnifies the impact, in comparison with the null ($\theta = 1$) model (Fig. 6B). In general, while both nontarget population growth rate and competition type do have consequences for minimum densities, these changes are relatively subtle, and they do not affect the utility of the approximation formula.

Thus far we have assumed that the nontarget is regulated by intraspecific competition. To investigate the case in which a native specialist parasitoid regulates the nontarget, we constructed a model based on Eq. 1 but added a fourth equation for a specialist parasitoid that attacks only the nontarget host. This fourth equation is identical in form to Eq. 1a, but interference for the new parasitoid was added so that the system consisting of the specialist parasitoid and nontarget host is persistent. We consider two possibilities for the interaction between the two parasitoid species: (a) the biocontrol agent always emerges from multiply-parasitized hosts (''agent precedence''), and (b) multiply-parasitized hosts give rise only to the resident parasitoid (''native precedence''). The results for both cases are shown in Fig. 7. In the case of agent precedence, the nontarget impacts fall in line with the approximation. What is not shown, however, is the impact of the biocontrol agent on the resident parasitoid, which suffers from the introduction via mortality within multiply-parasitized hosts. Where the native parasitoid has precedence, it does not suffer such a severe impact. However, as the native parasitoid is less affected, it can continue to generate mortality of the nontarget host over the transient period, which is added to that of the agent. Therefore, the nontarget minima are slightly lower than the approximation would suggest (Fig. 7), as the mortality caused by the two species acts in concert.

AGENTS WITH DYNAMIC HOST RANGES

The host ranges of certain biocontrol agents are likely to be outcomes of flexible aspects of their behavior. For example, acceptance of a less suitable host may depend on encounter rates with more acceptable hosts, as well as many other factors (Godfray 1994). Also, the tendency to search in particular microhabitats for hosts may depend on the expected local densities of target and nontarget hosts. For example, microhabitats with very low host densities may temporarily be completely ignored (Papaj et al. 1994). The decision to leave individual patches of habitat is also likely to depend on relative and absolute densities of hosts of different quality, and also on densities of conspecific parasitoids (Godfray 1994, Vos et al. 1998, Visser et al. 1999). These decisions are apparently made on the basis of both experience and local information regarding host densities and levels of superparasitism, and from direct encounters with conspecific searching parasitoids. Understanding such behaviors using models calculating fitness optima, and by postulating decision-making rules (e.g., ''rules of thumb''), has become an area of much sophistication (Godfray 1994, Roitberg 2000). These types of parasitoid behaviors will change the attack rates of biocontrol agents on target and nontarget hosts. We suspect that, for some species at least, in situations with many agents and few targets, many more (less suitable) nontarget hosts may be attacked.

Here we examine how dynamic changes in the realized host range of the biocontrol agent could influence the nature of nontarget impacts. We make a very simple assumption about how acceptance of the nontarget changes. Specifically, when the expected fitness of a parasitoid agent exclusively utilizing the target host goes below a certain level, the agent will begin to accept the less suitable nontarget hosts (the zero-one rule; Godfray 1994). This assumption produces two distinct searching efficiencies by the agent for the nontarget: a low searching efficiency when the density of target hosts is high, and a high searching efficiency when the density of target hosts is low.

Mutual interference is an important interacting factor here, so it was included in the model. The importance of mutual interference arises because changes in the level of mutual interference not only have a direct effect on the peak numbers of parasitoids, but also on the number of target hosts present at the time of this peak, due to the tendency of parasitoid interference to promote stability. Stable scenarios never have the huge troughs in target abundance that will leave agents with very few preferred hosts. This will strongly influence the realized host range of the agent at the time of greatest potential impact on the nontarget. Again, the way we model mutual interference can also be taken to en-

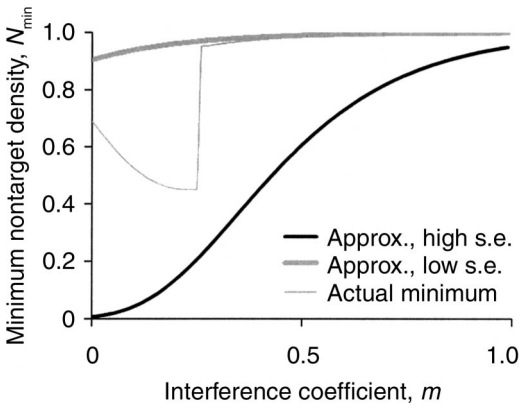

FIG. 8. The effect of dynamic host range in systems with interference. The changes in minimum nontarget density with changes in the mutual interference coefficient are shown. The two alternative searching efficiencies (s.e.) on the nontarget are used to generate the appropriate approximation for this type of system (Eq. 11) (''Approx., high s.e.'' and ''Approx., low s.e.''). Under a dynamic host range scenario, shifts from the low to the high searching efficiency occur when the expected fitness of a parasitoid individual parasitizing only the target host falls below a threshold (ρ). For low values of m (around 0.3), there are violent fluctuations in agent and target densities, leading to generations when the agent has the higher searching efficiency for the nontarget. When this occurs, the predictions from the approximation formula underestimate the minimum nontarget densities. Other parameters are $a_H = 0.2$, $r_H = r_N = 1$, $K_N = 10$, $K_H = 100$, $a_{high} = 0.05$, $a_{low} = 0.001$, and $\rho = 0.005$. See Fig. 1 for definitions of parameters.

compass a number of other phenomena, such as risk aggregation and refugia.

To model changes in nontarget acceptance, we started with Eq. 4 for the parasitism functions. Two alternative searching efficiencies were applied for the agent searching for the nontarget depending on the results of the following inequality, which defines the fitness (expected number of progeny) of an agent when utilizing the target population only:

$$[c_H(H_t/P_t)](1-\exp\{-a_H P_t^{(1-m)}\}) > \rho. \quad (9)$$

The threshold ρ defines the critical fitness below which the nontarget is accepted. When this inequality is satisfied, the parasitism function for the nontarget is

$$f(P) = \exp(-a_{low}P^{(1-m)}) \quad (10a)$$

whereas when the inequality is not satisfied,

$$f(P) = \exp(-a_{high}P^{(1-m)}). \quad (10b)$$

The searching efficiency of the agent for the target is always a_H, as in Eq. 4.

The results with representative parameter values are shown as a plot of minimum nontarget density against the strength of parasitoid interference, m (Fig. 8). The figure also shows the approximation calculated for each of the two searching efficiencies:

$$N_{min} = K_N\exp(-a_{(low)}K_H^{(1-m)}) \quad (11a)$$

$$N_{min} = K_N\exp(-a_{(high)}K_H^{(1-m)}). \quad (11b)$$

For the values used, moderate or high values of m ensure enough stability so that there are few periods of very low target density. Thus, conditions are rarely extreme enough for the biocontrol agent to attack the nontarget at high searching efficiency. In these cases the approximation based on the low searching efficiency is accurate. Where m is low, however, the dynamics are predominantly unstable, with either diverging oscillations or sustained cycles of some sort. These dynamics imply periods of low target abundance coupled with high agent abundance. The first peak in agent numbers is often large, and conditions are such that the high searching efficiency for the nontarget is applied in many (but not all) generations over this period. This means that the impact on nontargets resulting from the first peak is large, but not as large as it would be if the high searching efficiency were applied continuously. Unfortunately, there is no simple way to predict the nontarget effect when parasitoids change their searching behaviors in response to target and nontarget densities, and given that studying shifts in searching behavior under different conditions that parasitoids will likely face in nature is difficult, we see no simple resolution of this problem. These situations are doubly complex because the cycles that sometimes occur when m is low lead to different timings of the peaks in agent and target abundance, so that nontarget utilization, and therefore nontarget impact, may be higher in subsequent peaks in agent density than it is in the first peak (only the effects of the first peaks are shown in Fig. 8, though). Therefore, dynamic host-range changes on the part of biocontrol agents may complicate the prediction of transient nontarget effects generally.

DISCUSSION

Even relatively unsuitable, little-preferred nontarget hosts may be at risk of severe population reduction, and perhaps local extinction, from the introduction of a parasitoid biocontrol agent during transient periods just after agent introduction. This is because the biocontrol-agent population is likely to increase rapidly on its initially-abundant target host, and the high population density of the agent and declining population density of the target could result in much parasitism of the less preferred nontarget host. This could happen in spite of an absence of any potential for the nontarget population to sustain the agent over the long term.

The predicted transient impact on a nontarget host is greatest in the most basic host–parasitoid model we considered (Eq. 1), where a huge peak in agent density following introduction leads to the rapid local extinction of both target and agent. Similar phenomena still occur where target–agent interactions are locally persistent, but factors that promote local stability and persistence tend to reduce the magnitude of the nontarget effects. Subsequent models, investigating a range of phenomena common in host–parasitoid dynamics, showed how a number of mechanisms are likely to

reduce these risks in any particular host–parasitoid system.

In many cases, the depth of the troughs in nontarget densities following the introduction of the biocontrol agent can be described by a very simple approximation formula. The most basic version of this formula (Eq. 2) is based on data that are in principle easy to obtain: the density of the target, the density of the nontarget, the ability of the biocontrol agent to locate and propensity to accept the nontarget, and the efficiency of conversion of parasitized targets into agents. Although in models these factors are described by parameters that can be considered abstractions, and might be difficult to estimate in the field in a strict way, any quantitative data relating to these factors would help narrow the range of expectations of the severity of transient nontarget effects.

By investigating a range of models, we demonstrated that the generalized approximation formula (Eq. 3) does well under many circumstances. Not only can the formula potentially be used to give some indication of the worst-case local consequences for a nontarget, it also gives insight into the important ecological characteristics that influence nontarget effects. Even where the approximation 'failed,' closer examination of the failure revealed new ecological factors that may be important in predicting nontarget effects, particularly those that mitigate nontarget effects relative to the worst-case expectation.

Our analyses have focused on transient effects that occur soon after the introduction of a biocontrol agent. We focused on transient effects for four reasons. First, this problem has been neglected by theorists who have in the past focused mainly on community assemblage and stability issues in host–parasitoid systems. Second, transient effects may have a large impact on nontarget species even if they are very much less preferred by the biocontrol agent than is the target species. This highlights the potential difficulties confronting any prerelease screening of a potential biocontrol agent. Third, we show that even on marginal hosts introductions may cause extinction on the local level, which although not significant in themselves, may translate, via metapopulation dynamics, into broad-scale declines in nontarget abundance. Fourth, transient effects are the most difficult to study in the field, because they occur rapidly and only once in a given location. By highlighting the potential magnitude of transient nontarget effects, we want to emphasize the need to have monitoring programs in place before biocontrol programs are launched. They also present an opportunity for the use of short-term laboratory experiments (with a single parasitoid, a suitable host, and a marginal host) in a way that could usefully illuminate the risks of biocontrol.

Acknowledgments

This research was funded by EU project FAIR5-CT97-3489 (''ERBIC''), which supported L. D. Lynch, and NERC Centre for Population Biology at Silwood Park and grants from USDA-NRI, which facilitated the collaboration of A. R. Ives. We would like to thank Bob Holt, Keith Hopper, Rob Knell, and William Snyder for useful discussions, Fred Gould for gathering weed-related examples, and two anonymous reviewers for very helpful comments.

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