

The dynamic effects of an inducible defense in the Nicholson–Bailey model

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Abstract

We investigate the dynamic effects of an inducible prey defense in the Nicholson–Bailey predator–prey model. We assume that the defense is of all-or-nothing type but that the probability for a prey individual to express the defended phenotype increases gradually with predator density. Compared to a defense that is independent of predation risk, an inducible defense facilitates persistence of the predator–prey system. In particular, inducibility reduces the minimal strength of the defense required for persistence. It also promotes stability by damping predator–prey cycles, but there are exceptions to this result: first, a strong inducible defense leads to the existence of multiple equilibria, and sometimes, to the destruction of stable equilibria. Second, a fast increase in the proportion of defended prey can create predator–prey cycles as the result of an over-compensating negative feedback. Non-equilibrium dynamics of the model are extremely complex.

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1. Introduction

Inducible defenses—protective structures or mechanisms that prey organisms develop only in the presence of predators—are often spectacular examples of adaptive phenotypic plasticity. They may involve plastic changes in morphology, behavior or life-history, and are thought to evolve in systems that meet the following four conditions (Tollrian and Harvell, 1999a): (i) predation risk is variable in space or time; (ii) prey can estimate predation risk from a reliable cue; (iii) prey can effectively reduce predation risk by expressing a defensive phenotype; (iv) the defensive phenotype incurs a fitness cost, which makes it disadvantageous in the absence of predation risk. In recent years, inducible defenses have become a popular model system in

evolutionary ecology. For example, they have been used to study the trade-offs involved in phenotypic evolution and to understand fitness optimization in variable environments (see, Tollrian and Harvell, 1999a).

In addition, inducible defenses are receiving increasing interest from population ecologists (reviewed by Lima, 1998; Werner and Peacor, 2003). Intuitively, by creating a negative feedback between predator density and prey vulnerability they have the potential to stabilize predator–prey cycles. Several authors have incorporated inducible defenses (or related mechanisms such as prey refuges or antipredator behavior) into predator–prey models, and many models have, indeed, shown them to have a stabilizing effect (e.g., Ives and Dobson, 1987; Gyllenberg et al., 1996; Ruxton and Lima, 1997; Vos et al., 2004). Other studies, however, have yielded more ambiguous results (e.g., McNair, 1986; Kokko and Ruxton, 2000; Luttbegg and Schmitz, 2000; Ramos-Jiliberto et al., 2002), indicating that the relationship between inducibility and stability is not always straightforward.

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The majority of previous models have been based on differential equations (but see, Gyllenberg et al., 1996; Kokko and Ruxton, 2000; Luttbegg and Schmitz, 2000), that is they assume overlapping generations. Such an approach is justified for many well-studied systems with inducible defenses, for example in ciliates (Kuhlmann et al., 1999) or freshwater cladocerans (Tollrian and Dodson, 1999). However, inducible defenses are also found in systems with non-overlapping generations (or at least strongly seasonal life cycles). Examples include the larvae of mayflies and their stone-fly predators (Peckarsky et al., 1993), tadpoles and larval dragonflies (e.g., McCollum and Leimberger, 1997), wolf spiders (Persons et al., 2002), spiders and grasshoppers (Schmitz et al., 1997), rose hip flies and their parasitoids (Hoffmeister and Roitberg, 1997), or voles and weasels (Ylönen, 1989). For such systems, a model framed in difference equations seems more appropriate. Here, we develop and analyze a predator–prey model with an inducible defense and non-overlapping generations. We do not attempt to describe any specific interaction. Instead, we build a very simple model which we hope captures important aspects of real-world systems.

Our model is based on the classical Nicholson–Bailey model. This is one of the simplest discrete-time predator–prey models, and it has played a prominent role in the search for stabilizing mechanisms in predator–prey and host–parasitoid interactions (e.g., Hassell, 1978). In its basic form, it is always unstable and does not even allow persistence of the system (i.e., permanent coexistence of the interacting species). However, it can be stabilized by a variety of mechanisms (e.g., Hassell and May, 1973; Beddington et al., 1975; Hassell, 1978; Adler, 1993; Godfray et al., 1994; Doebeli, 1997). For example, Hassell and May (1973) showed that persistence and stability are possible in a predator–prey system if a constant proportion of the prey is protected from predation. Here, we use this ‘constant-proportion defense model’ as a null model, and we extend it by assuming that inducibility of the defense causes the proportion of protected prey to increase with predator density.

Important properties of inducible defenses are determined by trade-offs and constraints (Tollrian and Harvell, 1999b). Trade-offs are caused by defense costs, such as reduced fecundity of defended individuals. Constraints may arise if the prey has limited information about its environment or the induction mechanism is subject to developmental noise. Here, we incorporate trade-offs and constraints into a simple threshold model of defense induction. More precisely, we assume that the prey can express two alternative phenotypes: one phenotype is non-defended and fast-growing, whereas the other one is defended (‘induced’) but grows more slowly. The prey use a cue, such as a kairomone (i.e., a predator-released chemical), to estimate predator-density, and they develop the defended phenotype if the estimated predator density exceeds a threshold. Unreliability of the predator cue as well as developmental noise generate stochastic variation in the response of individual prey to the *actual* predator

density. This ‘response variance’ reflects constraints on phenotypic plasticity and determines the effect of inducibility at the population level. We analyze how the dynamic behavior of the predator–prey system is influenced by the response variance, the mean induction threshold, and the benefits and costs of the defense. We find that inducibility of the defense facilitates persistence of the predator–prey system. Furthermore, it has the potential to stabilize predator–prey cycles, but only if the defense is not too strong and the response variance not too small.

2. The model

2.1. The inducible defense

We assume that both species have discrete, non-overlapping generations. At the beginning of each generation, each prey individual uses a cue (e.g., a kairomone) to estimate current predator density. The defense is expressed if the estimated predator density \hat{P} exceeds an induction threshold $\hat{\mu}$. Both \hat{P} and $\hat{\mu}$ are subject to stochastic variation. Variation in \hat{P} is brought about by variation in the level of the predator cue (e.g., kairomone concentration) experienced by individual prey. We assume that \hat{P} is normally distributed with mean P (the true predator density) and variance σ_c^2 . Therefore, σ_c^2 is a measure of cue unreliability or imperfect information. Variation in $\hat{\mu}$ is caused by developmental noise, which may occur at any step of the signal transduction chain leading from the perception of the predator cue to the eventual expression of the phenotype. We assume that $\hat{\mu}$ is normally distributed with mean μ and variance σ_d^2 . Here, μ is the (individual) mean induction threshold, which may be viewed as determining the prey’s strategy. In principle, μ might vary between individuals due to heritable genetic variation and be subject to selection (see Discussion). In the present paper, however, we will assume that μ is identical in all individuals (although some non-heritable variation in μ might be viewed as a component of developmental noise).

Together, cue unreliability and developmental noise determine the prey’s ‘response variance’, that is the variance in the response of individual prey to the actual predator density P

$$\sigma^2 = \sigma_c^2 + \sigma_d^2. \quad (1)$$

Thus, σ^2 measures the inaccuracy of the prey’s response to P , which may be viewed as a constraint on phenotypic plasticity (in the sense that a plastic response to the environment cannot be more accurate than the available information and the precision of the developmental system). The probability of a prey individual to express the defense at a given predator density P is

$$d(P) = \int_{-\infty}^P \frac{1}{\sqrt{2\pi\sigma^2}} e^{-(1/2)((x-\mu)/\sigma)^2} dx, \quad (2)$$

which is a cumulative normal distribution with mean μ and variance σ^2 (Fig. 1). P is, of course, always positive, but as a

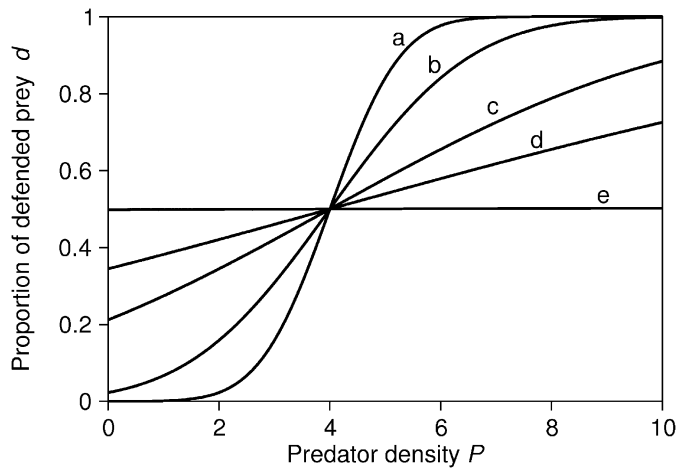


Fig. 1. Prey induction frequency d as a function of predator density P at the beginning of a generation. For all graphs, the mean induction threshold $\mu = 4$. The slope of the graphs is determined by the response variance σ^2 . Its values are 1 (a), 4 (b), 25 (c), 100 (d), and 10^6 (e). The smaller σ^2 , the stronger phenotypic plasticity is ‘seen’ at the population level. Note that for $\sigma^2 = 10^6$, d is practically independent of P , and hence, plasticity has no population-level effects at all.

consequence of using the normal distribution, integration starts at $-\infty$, leading to a positive value of d for $P = 0$. Furthermore, and somewhat counter-intuitively, the mean induction threshold μ may be negative. In this case, the probability of defense induction in the absence of predators is greater than $\frac{1}{2}$. At the population level, $d(P)$ is the proportion of defended prey, which we will refer to as the prey’s ‘induction frequency’. Importantly, σ^2 determines the slope of this function, and therefore, it determines the degree of phenotypic plasticity ‘seen’ at the population level.

If σ^2 becomes very large, $d(P)$ approaches a horizontal line, that is the induction frequency becomes independent of predator density (Fig. 1(e)). (Note that any value of d can be achieved for arbitrarily large σ^2 ; however, for $d \neq 0.5$ and $\sigma^2 \rightarrow \infty$, also $\mu \rightarrow \pm\infty$.) This may happen if the prey either has no useful information about predator density (large σ_c^2 : the cue is totally unreliable) or is unable to react to this information in a predictable manner (large σ_q^2 : developmental noise is maximal). Thus, in the limit of $\sigma^2 \rightarrow \infty$, our model reduces to a constant-proportion defense model (see below). In this case, even though the prey still is phenotypically plastic (in the sense of being able to produce two alternative phenotypes), it no longer makes sense to speak of an inducible defense, since there is no correlation between phenotype and environment. Therefore, we will treat the case $\sigma^2 \rightarrow \infty$ (i.e., the constant-proportion defense model) as a null model with a constitutive (i.e., non-plastic) defense.

2.2. Population dynamics

Within each generation, undefended prey are killed by predators at rate aP . If they survive, they produce an average of λ offspring. Thus, a measures the vulnerability and λ the fecundity of undefended prey. Note that

vulnerability is determined by several components of the predation cycle, such as encounter rate, detection probability, attack rate, attack efficiency, handling time, and digestion time (Jeschke et al., 2002). The mean fitness of undefended prey is

$$\bar{w}_{n,u} = \lambda e^{-aP}. \quad (3)$$

(The index n stands for prey and the index u for undefended.) The exponential term gives the proportion of surviving prey at the end of the generation, that is the prey’s survival probability. Note that there is no direct density-dependence in the prey (see Discussion).

The defense has the effect of reducing prey vulnerability by a proportion b and reducing prey fecundity by a proportion c ($0 \leq b \leq 1, 0 \leq c \leq 1$). Thus, b and c represent the benefits and costs of the defense. The mean fitness of defended prey is

$$\bar{w}_{n,d} = (1 - c)\lambda e^{-(1-b)aP}. \quad (4)$$

(The index d stands for defended.) We assume that $\lambda(1 - c) > 1$, which assures that $\bar{w} > 1$ for both prey types in the absence of predation. The overall mean fitness of the prey is

$$\bar{w}_n = (1 - d)\bar{w}_{n,u} + d\bar{w}_{n,d}. \quad (5)$$

Prey that are killed by predators are converted into new predators in the next generation. The number of new predators produced per killed prey is denoted by β . If the density of prey is N the mean fitness of predators equals

$$\bar{w}_p = \beta \frac{N}{P} [(1 - d)(1 - e^{-aP}) + d(1 - e^{-(1-b)aP})]. \quad (6)$$

(The index p stands for predator.) This equation can be understood by noting that, of the undefended prey, a proportion $1 - e^{-aP}$ is killed by predators, as is a proportion $1 - e^{-(1-b)aP}$ of the defended prey. Finally, the dynamics of the predator–prey system are given by

$$N_{t+1} = N_t \bar{w}_n, \quad (7a)$$

$$P_{t+1} = P_t \bar{w}_p, \quad (7b)$$

where the index t measures time in units of one generation.

If d is independent of predator density (for $\sigma^2 \rightarrow \infty$, see above), the model reduces to an extended version of the constant-proportion defense model by Hassell and May (1973), who assumed that the defense provides complete protection ($b = 1$) coming at no costs ($c = 0$). (In their original paper, Hassell and May (1973) termed this model the ‘constant-proportion refuge model’, because they envisaged the prey using a physical refuge.) And for $d = 0$ or $d = 1$ (prey are either never or always defended), the model reduces to the classical Nicholson–Bailey model, which (for $d = 0$) is given by

$$N_{t+1} = N_t e^{-aP_t}, \quad (8a)$$

$$P_{t+1} = \beta N_t (1 - e^{-aP_t}). \quad (8b)$$

3. Results

3.1. Equilibria

We start by analyzing the equilibrium structure of system (7). Equilibrium requires $\bar{w}_n = \bar{w}_p = 1$. For any given prey density, there is exactly one predator density that satisfies $\bar{w}_p = 1$. Therefore, a necessary and sufficient condition for equilibrium is $\bar{w}_n = 1$. Unfortunately, this equation has no closed analytical solution, and so we are restricted to solving it numerically.

The behavior of the mean prey fitness \bar{w}_n as a function of predator density P is illustrated in Fig. 2. \bar{w}_n is close to $w_{n,u}$ for small P and switches towards $w_{n,d}$ around $P = \mu$. If σ^2 is small, the switch is fast, and if it happens in an area where $w_{n,d} > w_{n,u}$, then \bar{w}_n may increase with predator density over a certain range of P . In this range, the increased encounter rate with the predator is outweighed by the increased proportion of defended prey. If the increasing part of \bar{w}_n crosses the line $w_n = 1$ then system (7) has three non-trivial equilibria (in addition to the trivial equilibrium $N = P = 0$): a *low equilibrium* characterized by low predator density and low induction frequency, a *high equilibrium* with high predator density and high induction frequency, and an intermediate (saddle) equilibrium, which

separates the other two equilibria and is always unstable (see below). If the increasing part of \bar{w}_n does not cross the line $w_n = 1$ then there is only one non-trivial equilibrium.

Conditions for multiple equilibria. As can be seen from Fig. 2, a necessary condition for multiple equilibria is $\bar{w}_n(\tilde{P}) > 1$, where

$$\tilde{P} = \frac{-\ln(1-c)}{ab} \tag{9}$$

marks the intersection point of $w_{n,u}$ and $w_{n,d}$. $\bar{w}_n(\tilde{P}) > 1$ requires $b > \tilde{b}$ with

$$\tilde{b} = \frac{-\ln(1-c)}{\ln \lambda}. \tag{10}$$

Multiple equilibria also require an intermediate value of the mean induction threshold μ , as illustrated in Figs. 2(b)–(d). Fig. 3(a) shows the ‘domain of multiple equilibria’ (i.e., the set of parameter combinations where multiple equilibria occur) in the μ versus b plane. Its lower boundary is always close to \tilde{P} (see dashed line in Fig. 3(a)), because only for $P > \tilde{P}$ does defense induction lead to an increase in prey fitness. The upper boundary of the domain of multiple equilibria increases with b . In particular, multiple equilibria do not exist for any value of μ if b is

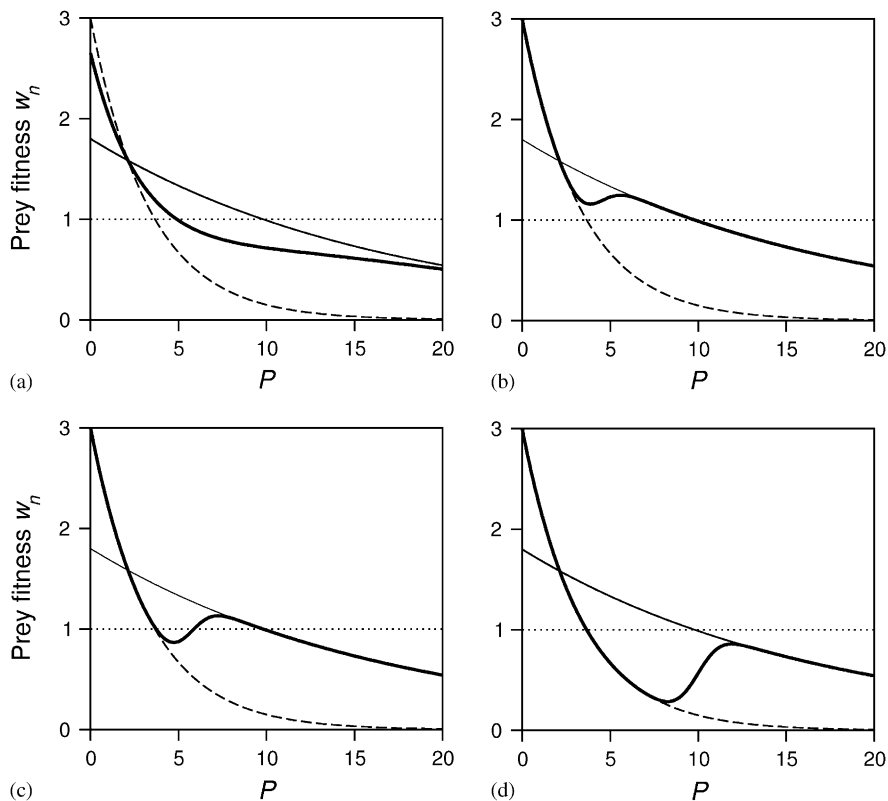


Fig. 2. Prey fitness as a function of predator density. The figures show the mean fitness \bar{w}_n of the prey (thick line), together with the fitness $w_{n,u}$ of undefended prey (dashed line) and $w_{n,d}$ of defended prey (thin solid line), as a function of predator density P . Equilibrium predator densities are given by the intersections of \bar{w}_n with the line $w_n = 1$ (dotted line). In (a), the response variance of the prey is high ($\sigma^2 = 100$), and there is only one non-trivial equilibrium. (b)–(c) show cases with low response variance ($\sigma^2 = 1$), which leads to an increase of mean prey fitness over a range of predator densities. The mean induction threshold μ is 4.0 in (b), 5.5 in (c), and 10.0 in (d). Only in (b), the system has three non-trivial equilibria. Parameters: $a = 0.3$, $b = 0.8$, $c = 0.4$, $\lambda = 3$, $\mu = 5.5$.

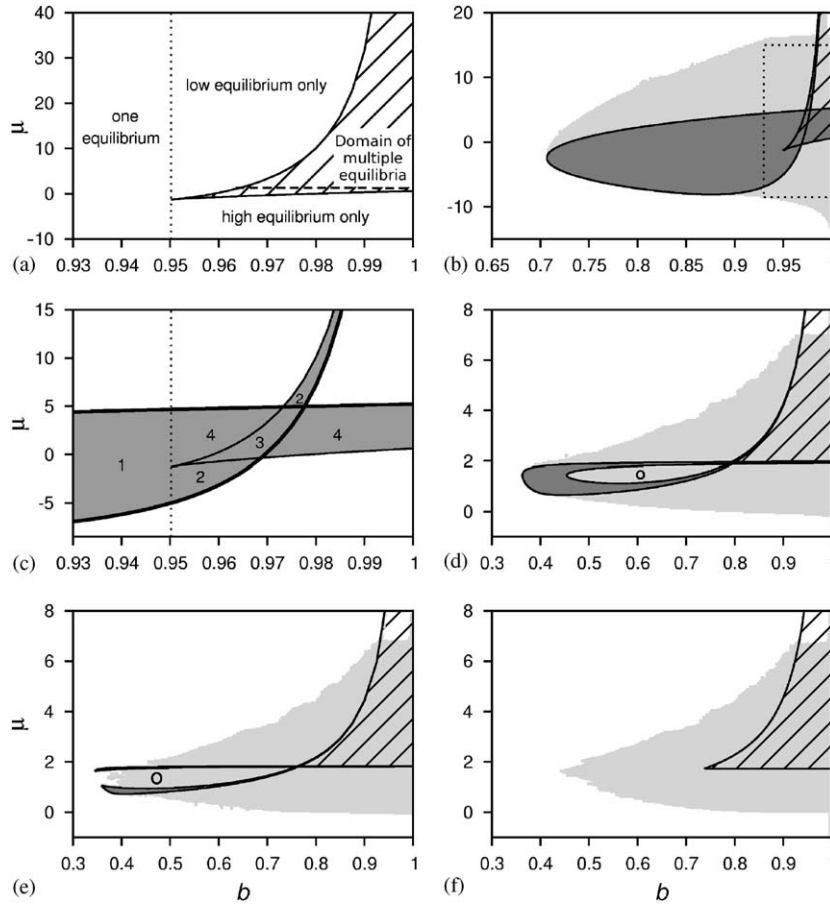


Fig. 3. The domains of multiple equilibria, persistence, and stability in a μ versus b plane for different values of the prey’s response variance σ^2 . Values of σ^2 displayed here were chosen to show typical and qualitatively different results. μ is the mean induction frequency of the prey (i.e. the predator density where each prey individual expresses the defended phenotype with probability 0.5), and b is the benefit of the defense. The hatched area is the domain of multiple equilibria and the shaded area is the domain of persistence. Dark grey marks the domain of stability, and light grey marks parameter combinations where the predator–prey system persists via non-equilibrium dynamics. No persistence is possible in unshaded areas. (a) The domain of multiple equilibria. Above the dashed line, $\mu > \bar{P}$ (see Eq. (9)). The dotted line marks b_{\min} , the minimal value of b for which multiple equilibria are possible (see text). (b) The domains of persistence and stability for high response variance ($\sigma^2 = 25$). Corresponds to Fig. 5(b). The area demarcated by dotted lines is magnified in (c). (c) A detailed view of (b). To keep the graph simple, only the domain of stability is shaded. The thin line marks the boundary of the domain of multiple equilibria. (1) Single equilibrium stable, (2) high equilibrium stable, (3) low and high equilibria stable, (4) low equilibrium stable. (d) The domains of persistence and stability for low response variance ($\sigma^2 = 0.01$). The ‘outer’ stability boundary is defined by condition (13b) and is equivalent to the stability boundary in (b) and (c). The ‘inner’ stability boundary arises due to violation of condition (13c). In the area marked “O”, predator–prey cycles are induced by overcompensation (see text). Corresponds to Fig. 5(d). (e) Fragmentation of the domain of stability for extremely low response variance ($\sigma^2 = 0.001225$). Corresponds to Fig. 5(e). (f) The domain of persistence for $\sigma^2 \rightarrow 0$. Note that the domain of stability does not exist. Parameters: $a = 0.4$, $\beta = 0.4$, $c = 0.4$, $\lambda = 2$.

less than a minimal value b_{\min} . b_{\min} , in turn, depends on σ^2 (see other panels in Figs. 3 and 5). In the limit of $\sigma^2 \rightarrow 0$ (where \bar{w}_n equals $w_{n,u}$ for $P \leq \mu$ and $w_{n,d}$ for $P > \mu$) b_{\min} equals \bar{b} . Numerical approximations show that it increases with increasing σ^2 and approaches 1 for $\sigma^2 \rightarrow \infty$. In summary, multiple equilibria exist if the defense is strong, the mean induction threshold μ is high but not too high, and the response variance σ^2 is low.

Fig. 4 illustrates the effects of μ and b on the equilibrium structure by plotting the equilibrium induction frequency d^* versus μ for $b > b_{\min}$ and $b < b_{\min}$, respectively. The figure also indicates the stability of the equilibria, which will be discussed below. For $b > b_{\min}$, the graph forms a hysteresis loop within the range of multiple equilibria (Fig. 4(a)). For

$b < b_{\min}$, the hysteresis loop and the multiple equilibria disappear (Fig. 4(b)).

3.2. Stability and persistence

3.2.1. General stability conditions

The simplifying assumption that the prey’s mean fitness \bar{w}_n is not density-dependent makes it possible to analytically derive some general conditions for stability. In the following, we regard a general formulation of model (7):

$$N_{t+1} = N_t \bar{w}_n(P_t), \tag{11a}$$

$$P_{t+1} = P_t \bar{w}_p(N_t, P_t). \tag{11b}$$

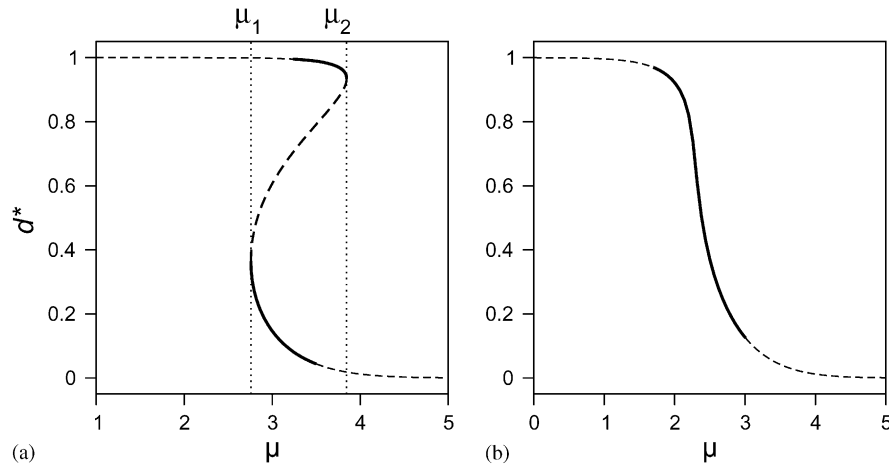


Fig. 4. Alternative equilibrium induction frequencies d^* plotted against the mean induction threshold of the prey, μ . μ is the predator density where each prey individual expresses the defended phenotype with probability 0.5. (a) If the defense is strong ($b = 0.75$) the high and low equilibria are linked by an unstable saddle equilibrium (dotted line), resulting in a hysteresis loop between μ_1 and μ_2 . This means that a system at or near the high equilibrium will switch to a neighborhood of the low equilibrium if μ is gradually increased beyond the value μ_2 , but will return to the high equilibrium only if μ is decreased beyond μ_1 . The high and low equilibria are stable at intermediate d^* (solid lines) and unstable at extreme d^* (dashed lines). (b) If the defense is weak ($b = 0.6$) the hysteresis loop and the intermediate equilibrium disappear. Parameters: $a = 0.4$, $\beta = 0.4$, $c = 0.1$, $\lambda = 2$, $\sigma^2 = 1$.

The Jacobian of system (11), evaluated at equilibrium, is

$$J^* = \begin{pmatrix} 1 & N^* \bar{w}'_n \\ \frac{P^*}{N^*} & 1 + P^* \bar{w}'_p \end{pmatrix}, \tag{12}$$

where

$$\bar{w}'_n = \frac{\partial}{\partial P} \bar{w}_n(P^*),$$

$$\bar{w}'_p = \frac{\partial}{\partial P} \bar{w}_p(N^*, P^*),$$

and the asterisks indicate equilibrium densities.

Applying the Jury test (e.g., Kot, 2001, p. 184) yields three conditions for local stability

$$\bar{w}'_n < 0, \tag{13a}$$

$$\bar{w}'_p < \bar{w}'_n, \tag{13b}$$

$$\bar{w}'_p > \frac{1}{2} \bar{w}'_n - \frac{2}{P^*}. \tag{13c}$$

Condition (13a) states that stability is only possible if, at equilibrium, the mean fitness of the prey decreases with increasing predator density (see Fig. 2). This proves that, in the case of multiple equilibria, the intermediate equilibrium can never be stable. According to condition (13b) stability requires that the within-species effect of an increase in predator density (increased competition for prey) must be stronger than the between-species effect (increased predation pressure exerted on prey; note that, if (13a) is satisfied, (13b) implies $|\bar{w}'_p| > |\bar{w}'_n|$). An inducible defense facilitates fulfillment of this condition, because it weakens the intraspecific effect of predator density and enhances the interspecific effect. It is condition (13b) that is never

satisfied in the basic Nicholson–Bailey model (8). Condition (13c) gives a lower boundary for \bar{w}'_p relative to \bar{w}'_n , thereby showing that stability can be lost again if the intraspecific effect of the defense becomes too strong.

3.2.2. Numerical results for stability and persistence

In the following, we use numerical methods to investigate the stability and persistence of model (7) in greater detail. In addition to the domain of multiple equilibria we describe two more domains in parameter space: the ‘domain of stability’ (i.e., the set of parameter combinations where at least one stable equilibrium exists) and the ‘domain of persistence’ (the set of parameter combinations where predator and prey coexist for at least some initial conditions). Note that the domain of stability is a subset of the domain of persistence. The domain of stability was determined by numerically investigating conditions (13). The domain of persistence was determined by simulating Eqs. (7). Simulations were started with initial population densities close to equilibrium, and the interaction was judged persistent if neither population dropped below a density of 10^{-4} for 10^5 generations. In the vast majority of simulations that did not meet this criterion, the predator population went extinct in less than 10^4 generations.

Fig. 3 shows the domains of stability and persistence in a μ versus b plane for various values of the prey’s response variance σ^2 . Additionally, in Fig. 5, the domains of stability and persistence are plotted in a d^* versus b plane, where d^* is the induction frequency at equilibrium. These two ways of presenting the results are complementary. Their relationship is illustrated by Fig. 4, which contains information about the domain of stability with respect to both d^* and μ for a specific value of b . The μ versus b plots have the

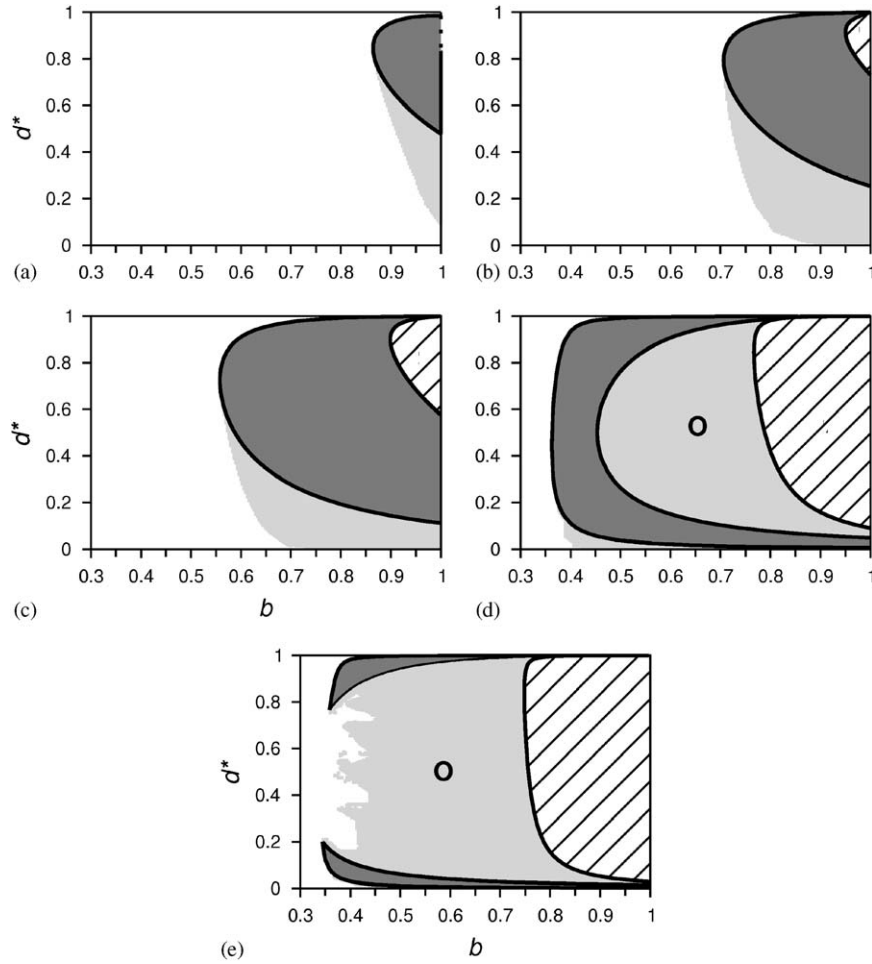


Fig. 5. The domains of stability and persistence for various values of the prey’s response variance σ^2 in a d^* versus b plane. d^* is the induction frequency at equilibrium (even if the equilibrium is not stable). b is the benefit of the defense. The shaded area is the domain of persistence. Dark grey marks the domain of stability, and light grey indicates persistence via non-equilibrium dynamics. The hatched area is the domain of the unstable intermediate equilibrium. Its boundaries correspond to those of the domain of multiple equilibria in Fig. 3 (see text and Fig. 4). (a) The constant-proportion defense model ($\sigma^2 \rightarrow \infty$, i.e. no plasticity ‘seen’ at the population level). $d^* = d$ is a constant, and there is only a single equilibrium. For $b = 1$, no equilibrium exists for $d > 1/[(1 - c)\lambda]$ (dotted line). (b) High response variance ($\sigma^2 = 25$). For high b , there are three alternative equilibria. The domain of stability is reduced by the domain of the unstable intermediate equilibrium. Corresponds to Figs. 3(b) and (c). (c) Intermediate response variance ($\sigma^2 = 2.25$). (d) Low response variance ($\sigma^2 = 0.01$), creating a second, ‘inner’ stability boundary, and an area of overcompensation (marked “O”, see text and legend to Fig. 3). Corresponds to Fig. 3(d). (e) Fragmentation of the domain of stability for extremely low response variance ($\sigma^2 = 0.001225$). Corresponds to Fig. 3(e). Parameters: $a = 0.4$, $\beta = 0.4$, $c = 0.4$, $\lambda = 2$.

advantage that both μ and b are model parameters, whereas d^* is a state variable. On the other hand, the d^* versus b plots have a much simpler structure, and they allow to include results for the constant-proportion defense model (Fig. 5(a)). Furthermore, d^* has a direct interpretation in terms of stability, because the slope of the function $d(P)$ at this point determines the strength of the negative feedback between predator density and prey vulnerability at equilibrium. Note that, for each d^* , there is a unique equilibrium. Therefore, the d^* versus b plots do not have a domain of multiple equilibria. Instead, for $b > b_{\min}$ there are separate domains for the low, intermediate and high equilibrium, respectively, and the boundaries of the domain of the intermediate equilibrium are analogous to the boundaries of the domain of multiple equilibria in the μ versus b plane. The effects of inducibility on the behavior

of the model can be assessed by comparing the structure of the various domains for different values of σ^2 .

Infinite response variance—constant-proportion defense model. We start with our null model, the non-plastic constant-proportion defense model ($\sigma^2 \rightarrow \infty, d(P) = d = \text{const.}$). Fig. 5(a) shows the domain of stability in the d versus b plane. (There is no corresponding plot in Fig. 3, because the results of this model cannot be properly displayed in the μ versus b plane.) The constant-proportion defense model has a unique non-trivial equilibrium, which is stable if the defense is sufficiently strong (large b) and both prey phenotypes are present at a sufficiently high frequency (intermediate d). The requirement of intermediate d can be understood by noting that, for $d \rightarrow 0$ or $d \rightarrow 1$, the model approaches the basic Nicholson–Bailey model (8), which is always unstable. The boundary of the

domain of stability is defined by stability condition (13b). For $b = 1$ (complete defense), no equilibrium exists if $d > 1/((1 - c)\lambda)$. In this case, predation is too weak to regulate prey population growth, and both populations increase without bounds (Hassell and May, 1973). Persistence via non-equilibrium dynamics mainly occurs for values of d below those allowing for stability. Interestingly, if b is less than a minimal value, neither stability nor persistence are possible.

Large response variance. Figs. 3(b) and (c) and 5(b) illustrate the behavior of the model for a large but finite response variance σ^2 . A comparison of Fig. 5(b) with Fig. 5(a) shows that the domains of stability and persistence have expanded towards the left, that is towards parameter combinations with a weaker defense than in the constant-proportion defense model. This is a stabilizing effect of inducibility: a weak plastic defense may be able to stabilize the system, where a weak non-plastic (i.e., constant-proportion) defense cannot. Note that for some regions, the boundary of the domain of stability seems to coincide with the boundary of the domain of persistence. However, we have no proof that the two boundaries actually are identical. Note also that the part of the domain of persistence located below the domain of stability in Fig. 3(b) cannot be seen in Fig. 5(b), because it is ‘condensed’ into an extremely narrow stripe close to $d^* = 1$.

Furthermore, the structure of the domain of stability is complicated by the presence of multiple equilibria, as can be seen on the right-hand sides of both plots. The right-hand side of Fig. 5(b) shows the domains of stability for both the low and the high equilibrium, separated by the domain of the unstable intermediate equilibrium. For $b = 1$, the high equilibrium does not exist and the upper limits of the domain of stability converge towards $d^* = 1$. (The non-existence of the high equilibrium can be easily seen from Fig. 2 by realizing that, for $b = 1$, the fitness of defended prey does not decrease with P .) Note that Fig. 5(b) does not show whether the high and the low equilibrium can be stable simultaneously for a given set of parameters. This information can be gained from Figs. 3(b) and (c) however, which show that simultaneously stable equilibria are only possible for relatively low values of μ . Looking back at Fig. 5(b), it seems that the domain of stability is reduced in size by the domain of the intermediate equilibrium. Figs. 3(b) and (c) offer a more mechanistic explanation for this finding: a shift in the mean induction threshold μ may destabilize the predator–prey system not because a stable equilibrium loses its stability but because a stable equilibrium ceases to exist and the alternative equilibrium is unstable (see Fig. 4(a)). Thus, the creation of multiple equilibria represents a destabilizing effect of defense inducibility. This effect only operates for highly effective defenses, and it contrasts the stabilizing effect of inducibility for weaker defenses. Note also that multiple equilibria reduce only stability, but not persistence of the model (Fig. 3(b)).

Intermediate response variance. If the response variance σ^2 is decreased, the domains of stability and persistence as well as the domain of multiple equilibria further expand to the left, that is towards weaker defense (Fig. 5(c); note that there is no corresponding plot in Fig. 3). We also varied the values of the other model parameters and found that the same pattern holds true for decreasing defense costs c , increasing prey fecundity λ , and decreasing prey vulnerability a (results not shown).

Low response variance. If σ^2 drops below a critical value, the structure of the domain of stability changes qualitatively: at high b , stability is lost for parameter combinations that lead to *intermediate* induction frequencies d^* (Figs. 5(d) and 3(d)). The critical value of σ^2 leading to this behavior decreases with increasing defense costs c (results not shown). Whereas the ‘outer’ stability boundary discussed previously is defined by stability condition (13b), the new, ‘inner’ stability boundary arises due to violation of stability condition (13c): at intermediate d^* and low σ^2 , the slope of the function $d(P)$ is so steep that the negative feedback between predator density and predation efficiency leads to overcompensation (e.g., Gurney and Nisbet, 1998, p. 60). This is another destabilizing effect of inducibility. As was the case with multiple equilibria, however, overcompensation does not affect persistence of the predator–prey system via non-equilibrium dynamics.

Finally, as σ^2 becomes smaller and smaller, the domain of stability becomes fragmented (Figs. 3(e) and 5(e)), and for $\sigma^2 \rightarrow 0$ it is easy to see that stability is impossible (because only one prey type will be present at any given predator density, as in the unstable original Nicholson–Bailey model). However, even in this extreme case, persistence of the predator–prey pair is still possible in a domain not much smaller than that for larger σ^2 (Fig. 3(f)).

3.2.3. Non-equilibrium dynamics

The non-equilibrium dynamics of model (7) are extremely complex, and a full analysis is beyond the scope of this study. Elementary theory of discrete dynamical systems (e.g., Gurney and Nisbet, 1998, p. 60) predicts that violation of stability condition (13b) (i.e., crossing of the ‘outer’ stability boundary in Figs. 3 and 5) induces stable limit cycles of non-integer period, whereas violation of stability condition (13c) (i.e. crossing of the ‘inner’ stability boundary) induces cycles of period 2. Further away from the stability boundaries, the system undergoes additional bifurcations which can lead to very complex and chaotic behavior. Fig. 6 shows a bifurcation diagram with respect to μ for a parameter combination where both types of stability boundary exist, and Fig. 7 presents some selected attractors that arise beyond the ‘outer’ stability boundary.

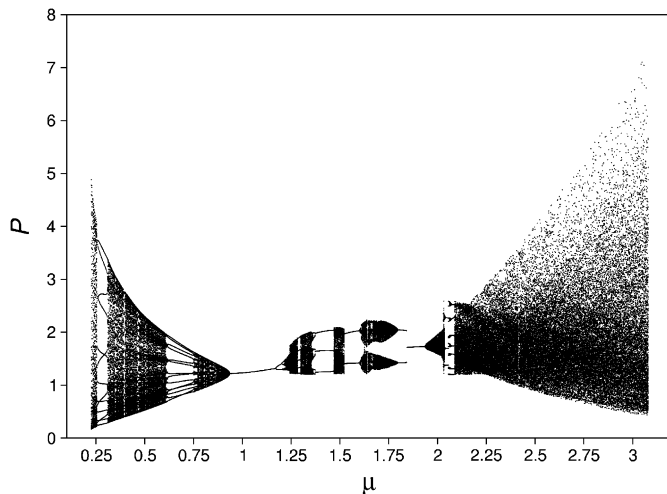


Fig. 6. Bifurcation diagram for predator density P as a function of the prey's mean induction threshold μ , for a parameter combination with only one non-trivial equilibrium and potential destabilization due to overcompensation. μ is the predator density where each prey individual expresses the defended phenotype with probability 0.5. The gradient of μ shown in the figure corresponds to a vertical cross-section at $b = 0.625$ in Fig. 3(d). The graph shows the predator densities that occur in the system after transient dynamics have been damped away. Single lines represent stable equilibria, multiple lines represent cycles with integer period, filled regions with clear-cut boundaries reflect cycles with non-integer period, and filled regions without clear boundaries point to chaotic dynamics. The complex structure in the middle of the graph arises due to overcompensation. Parameters: $a = 0.4$, $b = 0.625$, $\beta = 0.4$, $c = 0.4$, $\lambda = 2$, $\sigma^2 = 0.01$.

4. Discussion

4.1. Dynamic effects of defense inducibility

We have shown that an inducible prey defense has important effects on the dynamics of the Nicholson–Bailey predator–prey model. The parameters characterizing the inducible defense determine the persistence and stability properties of the model, as well as the number of equilibria and the nature of non-equilibrium dynamics. Of particular importance is the prey's response variance, which determines how strongly phenotypic plasticity is 'seen' at the population level (i.e., how fast induction frequency increases with predation risk). If the response variance is very large, the behavior of the model approaches that of the constant-proportion defense model (Hassell and May, 1973), which serves as a null model in our analysis. In the constant-proportion defense model, induction frequency is independent of predator density. The equilibrium structure is simple, and persistence and stability are possible only if the defense is very strong. Gradually decreasing the response variance reveals three major effects of inducibility.

First, a decrease in the response variance (i.e., an increase in the amount of plasticity seen at the population level) decreases the minimal strength of the defense that is required for persistence or stability. That is, a weak

inducible defense can stabilize the system, or at least promote its persistence, whereas a weak constitutive defense cannot. In this sense, inducibility is stabilizing. Mechanistically, the inducible defense creates a negative feedback between predator-density and prey vulnerability, which reduces the impact of predator density on prey fitness and increases the impact of predator density on predator fitness. Stability requires that the former is weaker than the latter. This condition resembles similar conditions for coexistence in standard models of interspecific competition (e.g., Begon et al., 1990). In the context of Nicholson–Bailey models, inducible defenses join a long list of potentially stabilizing mechanisms, such as density-dependent prey growth (Beddington et al., 1975), predator aggregation (Hassell and May, 1973), dispersal (Adler, 1993), phenological asynchrony between predator and prey (Godfray et al., 1994), or genetic variation in prey characteristics (Doebeli, 1997).

Second, if the defense is strong (large b), a finite response variance can lead to the existence of multiple non-trivial equilibria, which bring about interesting non-linear effects such as hysteresis and bistability. The potentially stable equilibria are characterized by low and high predator densities and prey induction frequencies, respectively. Multiple equilibria have no effect on persistence, but they reduce stability, because for certain parameter values, stable equilibria are replaced by unstable alternative equilibria. A necessary condition for the existence of multiple equilibria is an increase of prey fitness over a certain range of predator densities. This somewhat counter-intuitive behavior is a consequence of our assumption that prey do not behave optimally (see also Ramos-Jiliberto, 2003). Many models of inducible defenses do, indeed, make such an optimality assumption (e.g. Ives and Dobson, 1987; Gyllenberg et al., 1996; Křivan, 1998; Kokko and Ruxton, 2000). However, in our model, optimal prey behavior is not possible due to the constraint set by the response variance (see below).

Third, a very low response variance can lead to destabilization due to overcompensation. In this case, the negative feedback between predator density and prey vulnerability (i.e., the increase of prey induction frequency with predator density) becomes so strong that it induces oscillations, which are of high dynamic complexity but limited amplitude (see Fig. 6). Therefore, even strong overcompensation has no marked effect on persistence. Destabilization due to overcompensation is typical for discrete dynamical systems (e.g., May, 1974; Beddington et al., 1975) and, therefore, is a consequence of our assumption of non-overlapping generations. The effect of non-overlapping generations can also be seen by comparing our model to that of Křivan (1998). Křivan's model is similar to ours, but it is based on the continuous-time Lotka–Volterra model and it assumes that prey behave optimally. Therefore, at any given predator density, prey are either all defended or all undefended. The optimality assumption can be satisfied in our model in the limiting

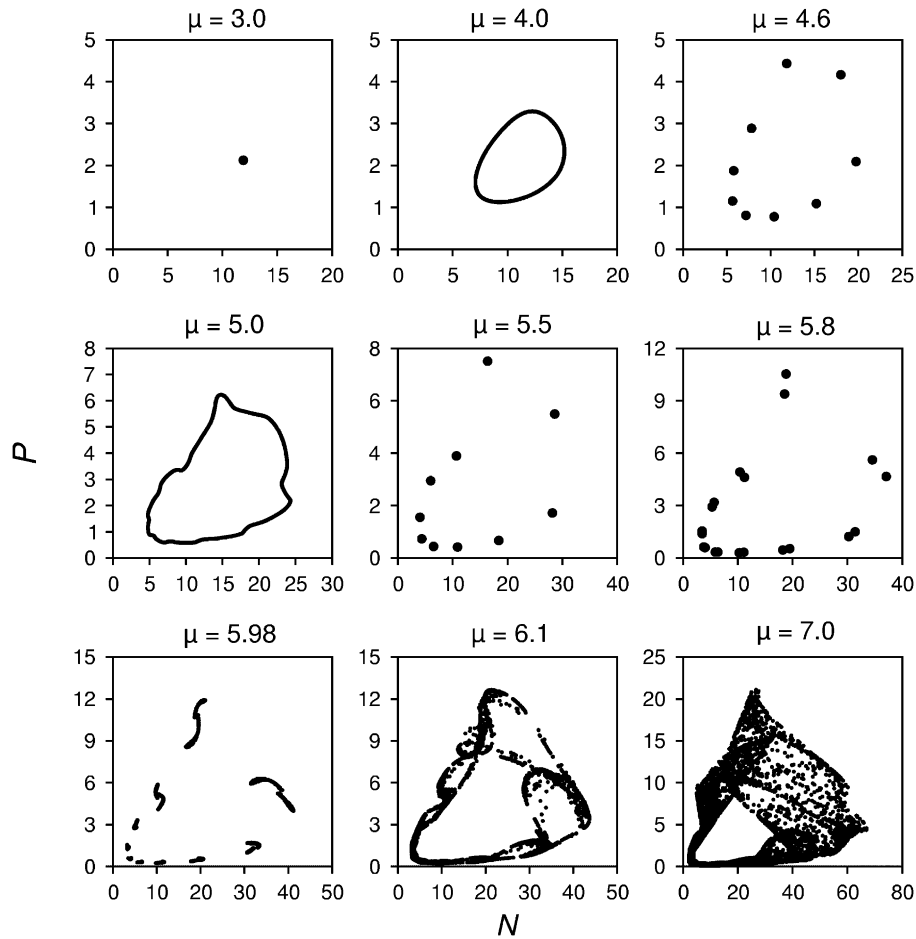


Fig. 7. Some possible attractors of model (7) in the P versus N phase plane, for a gradient of the mean induction threshold μ (i.e., the predator density where each prey individual expresses the defended phenotype with probability 0.5). The gradient corresponds to the right-hand side of a graph similar to that in Fig. 6 (but without overcompensation), that is it crosses the ‘outer’ stability boundary. As μ increases (and moves away from the domain of stability) the dynamics change from a stable equilibrium to stable limit cycles with either integer (distinct points) or non-integer (closed curve) period and, finally, to chaos (complex, ‘ragged’ plots). Parameters: $a = 0.4$, $b = 0.75$, $\beta = 0.4$, $c = 0.2$, $\lambda = 2$, $\sigma^2 = 4$.

case where $\sigma^2 \rightarrow 0$ and $\mu = \tilde{P}$ (see Eq. (9)). In Křivan’s model, population dynamics follow Lotka–Volterra cycles with only one prey phenotype. These cycles are neutrally stable, but their amplitude is limited because they cannot cross the threshold predator density \tilde{P} . In our model, in contrast, the system performs chaotic oscillations with predator density constantly crossing the value $P = \tilde{P}$ due to overcompensation.

In general, our results are in line with those of previous models. For a large parameter range, they support the traditional view that prey behavior stabilizes predator–prey dynamics. This view has been laid down in early papers on refuges by Hassell and May (1973), Maynard Smith (1974) and Murdoch and Oaten (1975) and has since been confirmed in a large number of studies (e.g., Ives and Dobson, 1987; Sih, 1987; Ruxton, 1995; Gyllenberg et al., 1996; Ruxton and Lima, 1997; Křivan, 1998; Ramos-Jiliberto, 2003; Rinaldi et al., 2004; Vos et al., 2004). However, recent work has shown that inducible defenses or related mechanisms are not stabilizing in all cases. Several mechanisms can lead to destabilization. For example,

McNair (1986) found that prey refuges can both increase or decrease stability, depending in part on how prey in the refuge influence the predators functional response. Kokko and Ruxton (2000) pointed out that the dynamic effects of predator-induced breeding suppression in small mammals depend on details of the prey’s density-dependence at equilibrium. Similarly, Ramos-Jiliberto et al. (2002) showed that the effects of antipredator behavior depend critically on details of the assumed costs. Finally, Luttbeg and Schmitz (2000) reported a destabilizing effect of flexible prey behavior and attributed this finding to time delays created by their assumptions of non-overlapping generations and imperfect information for the prey (see also Underwood, 1999). To these destabilizing mechanisms, we here add the effects of multiple equilibria and overcompensation.

4.2. Influence of defense costs on stability

Increasing the costs of the defense increases the minimal strength of the defense required for stability. High defense

costs tend to counteract the effect of low σ^2 by increasing the effect of predator density on prey fitness (i.e., they impede fulfillment of stability condition (13b)). Furthermore, high costs increase the likelihood of overcompensation. In these regards, defense costs are destabilizing. However, high costs also decrease the likelihood of multiple equilibria and are, in this regard, stabilizing. These results are in line with previous studies that have shown equivocal effects of defense costs on stability. In the model by Ramos-Jiliberto and González-Olivares (2000), high costs of refuge use increase stability. Similarly, Sih (1987) and Ruxton (1995) noted that costs for the use of refuges reduce the probability that the prey escapes from predator control. However, Sih (1987) also found that costs counteract the direct stabilizing effect of refuge use. In the models by Ramos-Jiliberto et al. (2002) and Ramos-Jiliberto (2003), costs are either stabilizing or destabilizing, depending on the details of the model assumptions and parameters.

4.3. Discussion of the model assumptions

We will now discuss some of the key simplifying assumptions of our model. First, we assume that the prey can express only two discrete phenotypes. Such discrete polyphenisms do indeed exist both in morphological (Lively, 1986) and life-history (Washburn et al., 1988; Ślusarczyk, 1995) characters. A model with two discrete phenotypes is also appropriate if the prey choose between two alternative habitats (e.g., de Meester et al., 1999). Nevertheless, many inducible defenses can be graded according to the magnitude of predation risk (e.g., Kuhlmann and Heckmann, 1985; Tollrian, 1993). We expect, however, that our results might also apply to systems with a graded defense. This is because inducibility of the defense influences population dynamics entirely through the prey's induction frequency, which indeed is a graded and continuous function of predator density (Fig. 1). Furthermore, our threshold model captures an important property of inducible defenses in the wild, namely that defended and undefended prey are usually present simultaneously (see, Vos et al., 2004).

Second, in our model, the defense is the only stabilizing mechanism. In particular, there is no direct density-dependence. Neglecting density-dependence in order to analyze other potentially stabilizing factors has a long history in the study of Nicholson–Bailey type models (e.g., Hassell and May, 1973; Adler, 1993; Doebeli, 1997; see also, Sih, 1987; Křivan, 1998). However, future studies might investigate the interplay between inducible defenses and other stabilizing mechanisms.

Finally, some discussion is warranted by our treatment of constraints on phenotypic plasticity. The way our model is formulated, constraints such as imperfect information and developmental noise are summarized by the response variance σ^2 , which directly determines the effect of plasticity at the population level. Therefore, the effects of

constraints on population dynamics are just the inverse of the effects of inducibility. In particular, strong constraints (large σ^2) lead to destabilization. A destabilization of the dynamics due to imperfect information in the prey has also been found by Luttbeg and Schmitz (2000), but the underlying mechanisms (time-delays due to information gathering of the prey) was different. Furthermore, constraints on plasticity prevent optimal prey behavior. This is not only true in the direct sense that prey make mistakes in estimating or reacting to predator density. It also means that there is no a priori optimal value for the mean induction threshold μ . Instead, μ should equal $-\infty$ for $P > \tilde{P}$ and $+\infty$ otherwise (with \tilde{P} being the predator density at which both prey phenotypes have equal fitness, see Eq. (9)), because this minimizes the probability of developing the ‘wrong’ phenotype. If μ were allowed to evolve in a temporarily heterogeneous environment (with predator densities fluctuating above and below \tilde{P}), its current value would reflect the history of predator densities encountered by the prey's ancestors (see, Hazel et al., 1990, for a similar argument applying to spatially heterogeneous environments). This is why we treated μ as a model parameter that can be chosen arbitrarily and we investigated the population dynamics for *all* possible values of μ .

Furthermore, in treating μ as a constant, we implicitly assumed that evolution of μ takes place on a time-scale much larger than that of population dynamics. However, a separation of ecological and evolutionary time-scales need not always hold true (Thompson, 1998). Therefore, an interesting extension of the present model would incorporate evolution of μ into the dynamic equations of system (7). For example, μ could be modeled as a quantitative genetic trait (Hazel et al., 1990, 2004). Preliminary results suggest that, in this case, evolution of μ leads to an eventual destabilization of population dynamics and induces cycles in both the population densities and the value of μ itself (Kopp, 2003).

4.4. Conclusions

In summary, the effects of inducible defenses on the stability of predator–prey systems can be complex. In our model, inducibility reduces the strength of the defense required to stabilize the system, but it also increases the likelihood of destabilization due to multiple equilibria or overcompensation. Therefore, stability is greatest if the defense is strong but not too strong, defense costs are low, and the prey's response variance is low but not too low. In contrast to the effects on stability, inducibility almost universally facilitates persistence. Most generally, our results show that inducible defenses can have important consequences not only for the fitness of individual predators and prey, but also for the dynamics of populations and communities.

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