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The joint evolution of defence and inducibility against natural enemies

Karine Poitrineau*, Sam P. Brown, Michael E. Hochberg

Institut des Sciences de l'Evolution de Montpellier-UMR 5554, Université Montpellier II, CC065, Place Eugène Bataillon, 34090 Montpellier Cedex 5, France

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Abstract

We develop and analyse a model of inducible defence where two traits—defence and its inducibility—jointly evolve. Inducibility reduces costs of defence in the absence of enemies thereby permitting higher defence levels when attacked. If the cost of inducibility is low, then inducibility and defence may reinforce one another, resulting in a runaway leading to a highly inducible and highly effective defence. When inducibility is more costly, a new joint-equilibrium in defence/inducibility emerges displaying intermediate levels of both traits, and the prior 'run-away' scenario (high defence, high inducibility) may disappear. In contrast to the cost of inducibility, the cost of defence has mixed effects. An increase in costs of defence generally diminishes the level of both defence and inducibility at the intermediate locally stable equilibrium, but can favour the existence of the 'run-away' scenario of high defence-high inducibility. The enemy encounter-rate also has mixed effects. At high encounter rates an increase in encounters can lead to a higher/maximal defence and a lower level of inducibility (defence being almost always useful), but at low rates, an increase in encounters can lead to both higher defence and higher inducibility. We finally consider potential enemy responses to defensive change, and illustrate that herd immunity (reduction of encounter rates due to population-level defence) can affect both individual defence and induction that can be, depending on conditions, increased or decreased.

Keywords: Defence; Predator; Parasite; Herd immunity; Plasticity

1. Introduction

Inducible defences are distinguished from constitutive ones by their facultative mode of expression, defence being initiated only when enemies actually attack or when there are cues of enemy presence or threat (Harvell, 1990). Inducible defences are very diverse, ranging from plant chemicals (Maleck and Dietrich, 1999), to vertebrate and invertebrate immune systems (Strand and Pech, 1995), morphological changes (Sell, 2000; Young and Okello, 1998), behavioural responses (Van Burskirk, 2001), and indirect defences mediated by hosts (Agrawal and Fordyce, 2000).

One of the most common explanations for the success of induction is cost reduction, whereby in defending against enemies only when present, victims benefit from reduced or absent costs when not threatened by attack (Agrawal and Karban, 1999). A second possibility is that inducible defences permit a more efficient defence against different enemies, via resource reallocation to different modes of defence, depending upon which enemy attacks (Tollrian and Harvell, 1999). Third, a variable defence may limit damage to organisms (more specifically plants) since enemies have to frequently adapt to changes in quality of their victims (Karban and Baldwin, 1997; Adler and Karban, 1994). Further, it could disperse the damaged sites on one individual (enemies moving from induced parts to non-induced) and increase enemy movement, which can have a

*Corresponding author. Tel.: +33-467-149357.
E-mail address: karine@isem.univ-montp2.fr (K. Poitrineau).

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positive effect on victim fitness too (Tollrian and Harvell, 1999). And finally, induced defences could slow the evolutionary adaptation of herbivores by submitting them to variable selection pressures instead of constant, high selection pressures as would be the case for constitutive defences (Tollrian and Harvell, 1999).

Numerous theoretical models have addressed the diverse factors that could affect the evolution of inducible defences (Clark and Harvell, 1992; Adler and Karban, 1994; Shudo and Iwasa, 2001). For example, Shudo and Iwasa (2001) investigated the optimal investment in inducible versus constant defence, under the assumption that inducible defences incur a time delay cost, which potentially enables an enemy to exploit its host. Constant defence was found to be favoured by high enemy virulence and growth rate, and high encounter rates. Other work has considered inducible defence acting alone. Frank (1993) developed a model of the evolution of inducible defence in a population dynamic context. Here, induction was linked to the threat of attack and not to attack itself. Frank's model predicts that selection can favour inducible defence, with a sharp transition between induced and non-defensive states, and that high parasite density favours defence induction.

In models and experimental studies, constitutive and inducible defence are often considered as alternative modes of defence, neglecting possible coexistence and transitions between the two. However, on closer inspection many defences have both a basal constitutive level and an induced component (Harvell, 1990; Karban and Baldwin, 1997). Moreover, inducibility can vary between populations and can be genetically determined (Karban and Baldwin, 1997).

We develop theory of the joint evolution between investment in defence and in defensive inducibility. The induction trait in our model controls the reduction in defence costs when the victim is not attacked by enemies. We studied the effects of encounter rates with enemies and costs of defence and inducibility on the evolution of both traits. Further, we consider how a feedback between population defensive level and encounter rates (reflecting the victim-dependent dynamics of a specialist enemy) is likely to mould the evolutionary optima of host or prey defence.

2. The model

We begin by considering some simple trade-off models of defensive investment, illustrating how optimal defence levels are determined by balances of costs and benefits. Sufficient defences may make a victim invulnerable, but may be too expensive if always expressed. We reason that flexibility can make very strong defences affordable, by targeting the costs. This cost saving can be thereby redirected to other functions or to increase efficiency of defence itself. This suggests that investment in defence may jointly evolve with the degree of flexibility (or inducibility) in the coordination of defence. Note that this mechanism contrasts with tolerance strategies insofar as the former controls when defences are expressed, whereas the latter is itself a constitutive strategy for resource reallocation.

We assume that defence and inducibility are expressed as quantitative genetic traits. The host is more likely to survive if defence is pronounced relative to enemy 'virulence'. Thus these traits are 'graded', as opposed to 'matched' in certain types of gene-for-gene interaction (e.g. Sasaki and Godfray, 1999). We assume that both defence and inducibility come at costs to fitness. In particular, we assume that inducibility comes at an accelerating cost. Although the empirical form of such costs remains elusive, accelerating costs are a reasonable assumption if error rates in induction increase with defensive levels, or if increasing inducibility requires increasingly complex physiological machinery.

We are interested in the possible co-evolutionary outcomes between two traits linked to the expression of victim defence. Let d denote an individual's investment in defence (once induced) and i denote investment in induction capacity. By considering victim fitness as a function of both d and i, we derive the following victim fitness function:

$$w(d,i) = w_0 - ve(1-d) - cd^x(1 - i(1-e)) - ki^y. \qquad (1)$$

Here, baseline fitness, w_0, is the reproductive value of an undefended victim in the absence of enemies. The remaining negative terms reflect expected fitness losses endured by the victim as a result of successful enemy attack (second term), defensive investment (third term) and induction investment (fourth term).

Specifically, e represents enemy encounter rate (scaled 0–1) and v represents enemy virulence. Thus the magnitude of typical enemy-induced fitness loss (second term in Eq. (1)) will rise with encounter rate and virulence, and decline with victim defence. The costs associated with a specific level of defence d are determined by the parameters c and x alone, in the absence of induction ($i = 0$). In contrast, when $i > 0$, the costs of defensive investment are increasingly paid in proportion to enemy encounter rate, e. When $i = 1$ (perfect inducibility), the costs of defence d are paid *only* when the defence is induced by the enemy. Finally, the costs associated with a specific level of induction i are determined by the parameters k and y alone.

The victim's strategy-set is thus defined by the two continuous variables, d and i (limited to the range 0–1). In the results below, we analyse the co-evolutionary outcomes of competing investment in these two traits, as a function of the costs of investment (magnitude of costs: c, k; form of costs: x, y) and the enemy encounter

rate (e). Furthermore, we adapt the above model (Eq. (1)) to include the effects of herd immunity, whereby heavily defended populations reduce the local enemy encounter rate.

In order to define analytically an evolutionary optima d^* (for a given value of i), we solve $\mathrm{d}w/\mathrm{d}d|_{d=d^*} = 0$ for d^*:

$$d^* = \left(\frac{c(1-i+ie)x}{ev}\right)^{1/(1-x)}. \quad (2)$$

Likewise, in order to define analytically an evolutionary optima i^* (for a given value of d), we solve $\mathrm{d}w/\mathrm{d}i|_{i=i^*} = 0$ for i^*:

$$i^* = \left(-\frac{d^{-x}ky}{c(e-1)}\right)^{1/(1-y)}. \quad (3)$$

Eqs. (2) and (3) illustrate that the optimal strategies of defence and of inducibility are interlinked, with d^* being a function of i, and vice versa. To investigate this interdependence, we plot the two optima in a series of phase–plane diagrams of $d-i$ space.

3. Results

3.1. Generalist enemies, accelerating costs

Fig. 1 illustrates some of the possible coevolutionary outcomes when the costs of investment in both traits are an accelerating function of the level of investment (x and $y>1$). In other words, small levels of investment in d and/or i are relatively cheap. The diagrams in Fig. 1 can be interpreted in terms of the 'best response' strategies $i^*(d)$ and $d^*(i)$. A 'best response' approach can be used to derive the point(s) of evolutionary stability, as follows.

In the absence of inducibility ($i = 0$), an intermediate level of defence is favoured ($d^*\sim0.25$ when $c = 2$, ~0.5 when $c = 1$). However, in the cases illustrated in Fig. 1,

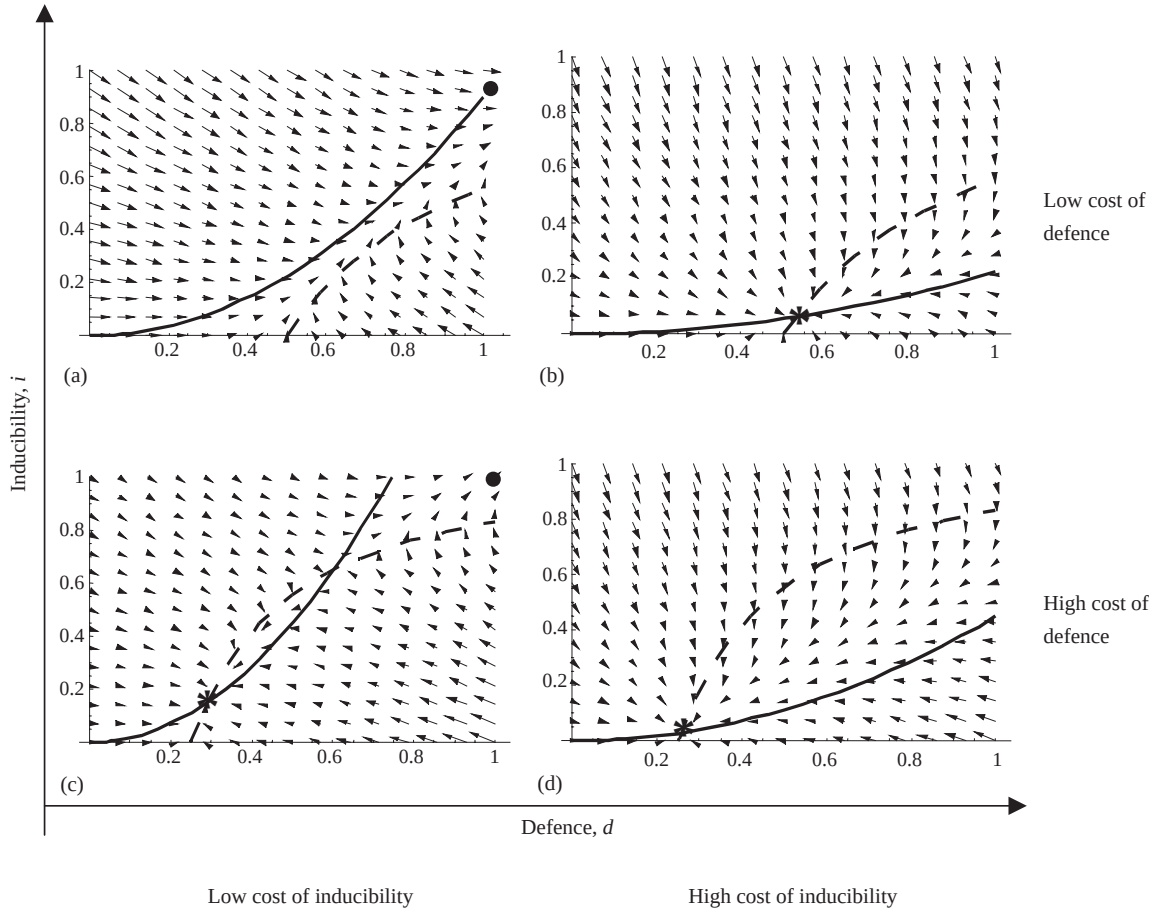

Fig. 1. Effects of costs defence and inducibility. Phase diagram illustrating the simultaneous dynamics of defense and inducibility for different costs of defence and inducibility. The dashed line is the isocline for $\mathrm{d}w/\mathrm{d}d = 0$ separating regions in which d increases or decreases. Similarly the solid line is the isocline for $\mathrm{d}w/\mathrm{d}i = 0$. The two possible stable equilibria are highlighted by the star and the spot. Parameter values are: $v = 10$, $x = 2$, $y = 2$, $p = 0.10$, top: $c = 1$, bottom $c = 2$, left $k = 0.5$, right, $k = 2$.

this equilibrium in d is vulnerable to invasion by positive values of i. When both costs are cheap (Fig. 1a), the introduction of inducibility favours higher levels of investment in defence, which in turn favours higher levels of inducibility, creating a mutually reinforcing runaway process, halting at the limit values of $i = d = 1$.

Starting from the same parameters as in Fig. 1a, and increasing the cost of inducibility to $k = 1$, we introduce a stable intersection point, where $d^* = 0.51$, $i^* = 0.2$ (Fig. 1b). Here an innovation in i leads to increases in d and i, but no longer to a runaway extreme. Instead, the final stable equilibrium is limited by the increased costs of manipulation.

3.1.1. Effect of cost of defence

Increasing cost of defence to $c = 2$, introduces a second intersection point (Fig. 1c). This increase also leads to a reduced value of d^* in the absence of inducibility ($d^* = 0.25$, versus $d^* = 0.5$ in Fig. 1a), and a reduced value of d^* at the first intersection stable point ($d^* = 0.26$, versus $d^* = 0.6$ in Fig. 1b). However, increasing the cost of defence acts to raise the benefit of inducibility. This shift upwards in the inducibility curve $i^*(d)$ leads to a second unstable intersection, which introduces a new runaway region of parameter space, allowing a second stable endpoint where $d^* = i^* = 1$. Note however that this final stable point can only be reached from a small region of parameter space (from already high values of d and i), due to the saddle separating this point from the alternate attractor where $d^* = 0.26$ and $i^* = 0.08$.

3.1.2. Effect of encounter rate

The general effect of increasing the encounter rate is, not surprisingly, to increase investment in the defensive trait. At low encounter rates, an increase raises the level of inducibility (equilibrium with intermediate defense and inducibility, highlighted by a star in the figures, see Figs. 2a and b) and favours the existence of another stable equilibrium (maximal defence, high inducibility);

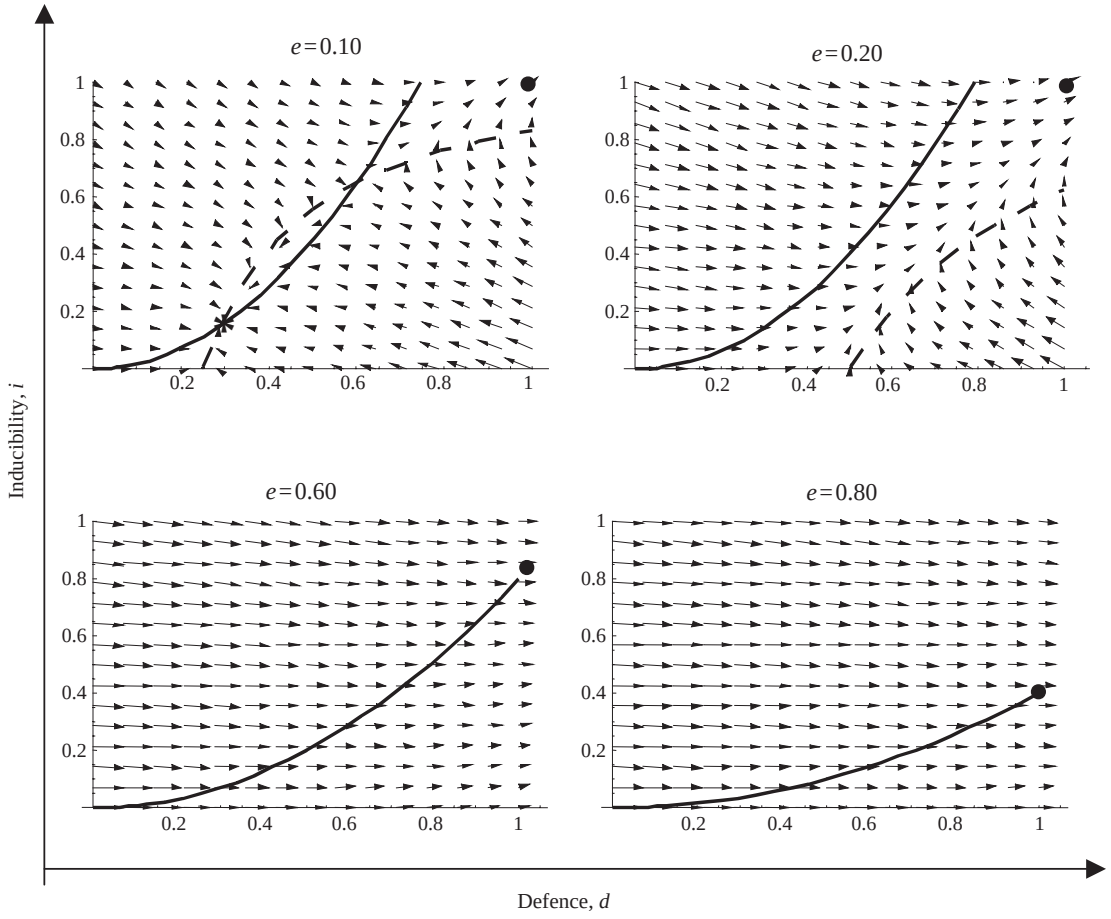

Fig. 2. Effects of costs defence and inducibility. Phase diagram illustrating the simultaneous dynamics of defense and inducibility for different encounter rates. The dashed line is the isocline for $dw/dd = 0$ separating regions in which d increases or decreases. Similarly the solid line is the isocline for $dw/di = 0$. The two possible stable equilibria are highlighted by the star and the spot. Parameter values are: $v = 10$, $x = 2$, $y = 2$, top: $c = 1$, $k = 0.5$.

however at high encounter rates the effect is the opposite: increases diminish the investment in inducibility (see Figs. 2c and d) when investment in defence is maximal.

A decrease of inductibility with increasing encounter rate (Figs. 2c and d) is consistent with results of other models (if victims are almost always attacked, there is no point in using an inducible defence (van Baalen, 1998; Tollrian and Harvell, 1999). The increase of inducibility with increasing encounter rate (at low encounter rate: Figs. 2a and b) is a more original result.

3.2. Effect of herd immunity: defence against a "specialist" enemy

We expect that both evolutionary and population-dynamical changes in enemies can lead to transitions between any number of the attractors outlined above (cf. 3.1.2). This suggests that feedbacks between population defence and encounter rate are important in specialist enemy–host systems. Here we link the population biology of a parasite to the defensive strategy of its host, to explore the possibility that increasing host defence leads to a reduction in enemy population density.

Let d = individual defensive effort (0–1), D = mean population defensive effort (0–1) and "effective encounter rate" = e–D (e–$e\,mig$). Where e = 'initial encounter rate' (the encounter rate in the absence of host defence) and mig (0,1) = migration, or the extent to which effective encounter rate is increased, by parasite migration from other patches or other hosts. Note if $mig = 0$, the host can potentially locally exterminate the parasite if population defence is 1.

Substituting into the previous fitness function we have

$$w = w_0 - v(e - D(e - p.mig))(1 - d) - cd^x(1 - i(1 - (e - D(e - e.mig)))) - ki^y. \quad (4)$$

Note that when $mig = 1$, we revert to the prior fitness function (Eq. (1)).

At a defence equilibrium, $d = D = d^*$, hence i^* can be found by optimality

$$i^* = \left(\frac{d^{-x}ky}{c - ce + cde - cd\,mig\,e}\right)^{1/(1-y)}. \quad (5)$$

Next we turn to d^*, necessitating a game-theoretical treatment, because fitness w depends on both the strategy of the individual (d) and of the group (D). Following the assumption that the host population is large and well-mixed, variation in individual defence d is assumed to be only negligibly correlated with population defence D.

Solving $dw/dd|_{d=D=d^*} = 0$ for d^* (Maynard Smith, 1982) we find d^* implicitly defined by

$$d^* = \left(\frac{d^{-x}\left(dev + d^2(mig - 1)ev - cd^x(1 + i(e - 1))\right)x}{ci - (1 + mig)ex}\right)^{1/(1-y)}. \quad (6)$$

Note that the 'population dynamical feedback' described by the new encounter rate function amounts to a representation of herd immunity (Anderson and May, 1991).

When mig is 1 (no herd immunity), we revert to a 'generalist pest' scenario, i.e. no local suppression of parasite density following defensive innovation. In other words, we revert to the previous scenario.

In Fig. 3 we present a simple comparison of dynamics with and without herd immunity, for both low and high 'undefended' encounter rate. Note how introducing herd immunity reduces the value of d^* for a given i. This is because herd immunity reduces selection for defence, since increasing defence reduces the effective encounter rate—and hence benefit—of defence (Frank, 1998)

Consider the case where encounter rate is high (Figs. 3a and b). When mig is 0 (high herd immunity) we introduce a local 'wipe out' scenario where runaways (mutually reinforcing selection on i and d, Figs. 3a and b) lead to the local extinction of the parasite population, via a 'generalist exterminator' mechanism of extinction, permitted by the targeted (i.e. induced) use of a powerful defence. Here, herd immunity favours higher inducibility: without herd immunity, defence against enemies is favoured as encounter rate increases ($d^* = 1$ for all i), and inducibility becomes practically useless (when encounter rate is high, there is little discrimination to be done). Consequently, there is a single stable attractor, $d = 1$; $i = 0.2$ (Fig. 3a). When herd immunity is introduced, d^* is reduced for low i as above. However, the threshold to investment in inducibility is also considerably reduced, allowing the establishment of a single attractor at $d = i = 1$. This enhancement of selection for inducibility can be understood as a consequence of the reduced encounter rate following increases in defence.

For lower encounter rate (Figs. 3b and c), herd immunity favours the existence of another equilibrium with intermediate defence and inducibility; the maximal defence–maximal inducibility equilibrium being reached only for a small area of the defence–inducibility space. For even lower encounter rates, the equilibrium with intermediate defence and inducibility moves toward even lower defence and inducibility, while the equilibrium with maximal defence/maximal inducibility is even more difficultly reached (Figs. 3d and e). The explanation is that the reduction of encounter rate due

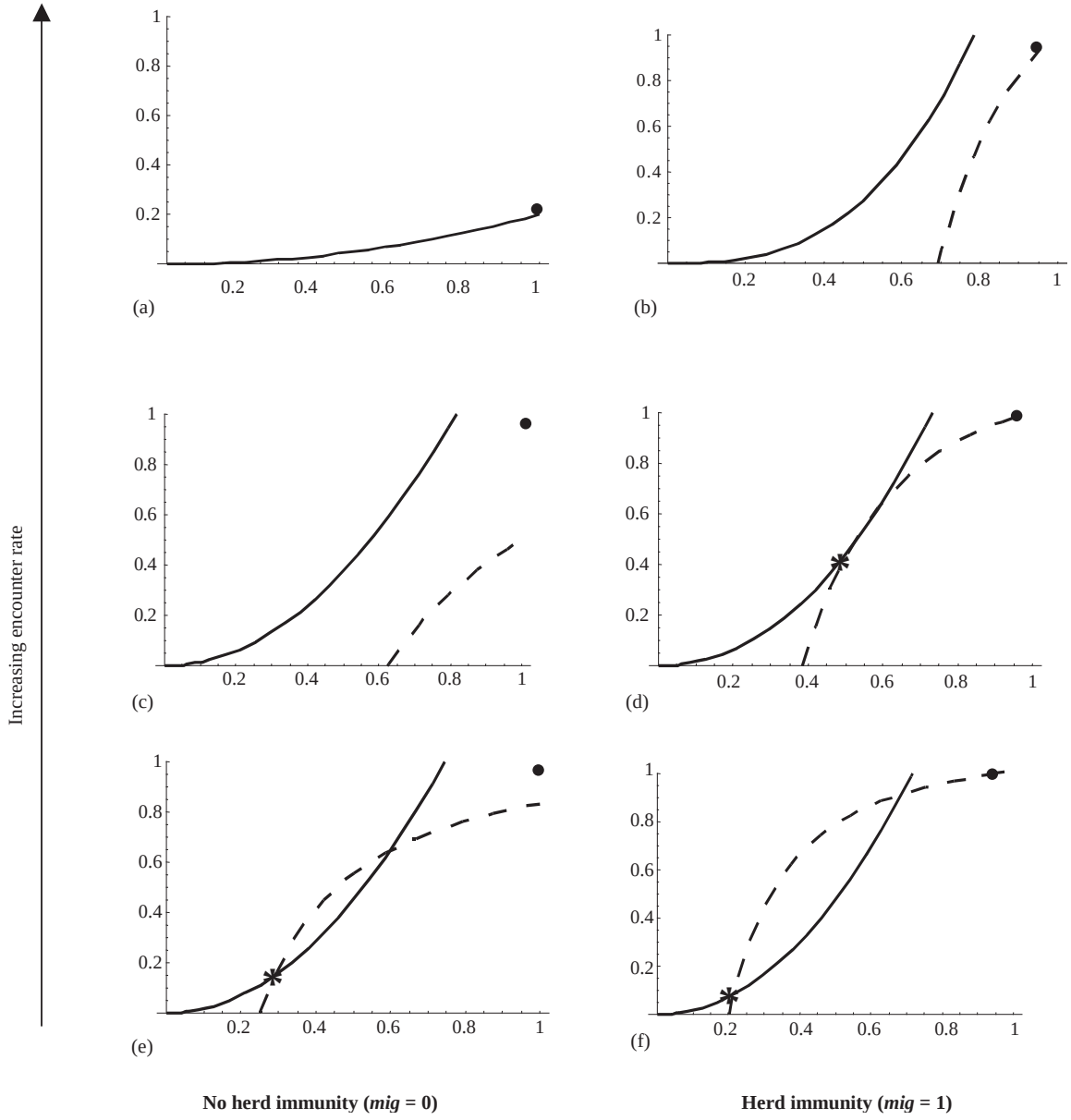

Fig. 3. Effects of herd immunity. Phase diagram illustrating the differences in stable equilibria for cases without and with herd immunity. The dashed line is the isocline for $dw/dd = 0$ separating regions in which d increases or decreases. Similarly the solid line is the isocline for $dw/di = 0$. Parameter values are: $v = 10$, $c = 2$, $x = 2$, $y = 2$, $k = 0.5$. Top panels: $e = 0.9$; middle panels: $e = 0.25$; bottom panels: $e = 0.1$.

to herd immunity permits less investment in defence. Because defence is less costly, it becomes less profitable to invest in inducibility to reduce this cost (because inducibility is costly too).

4. Discussion

One of the original features of our study is to have modelled defence inducibility as an independent evolutionary trait that modulates the costs and benefits of actual defences. The inclusion of inducibility leads to some conclusions that differ importantly from previous studies (Adler and Karban, 1994; Agrawal and Karban, 1999; Shudo and Iwasa, 2001), which consider the evolution of totally inducible defences versus constant defences. In general, we found that the evolutionarily flexibility of inducibility permits both more effective and less costly defences against natural enemies. The biological interest of our results aside, we suggest that models neglecting the complex inner-workings of defence systems may sometimes miss salient features of their action and evolution.

The joint dynamics of defence and inducibility can lead to sharp transitions between low defence–low inducibility and high defence–high inducibility optima.

However, whether one observes low defence–low inducibility or high defence–high inducibility depends on both the costs of defence and inducibility, together with the initial state of both traits. The enemy encounter rate is an additional, important determinant of the position of defence/inducibility equilibria. When encounter rates are low yet increasing, both defence and inducibiliy increase (as it becomes worthwhile to invest in both when the threat increases). But as encounter rate becomes very high, investment in inducibility diminishes (enemies become so abundant that the cost of inducibility is not compensated by cost savings when not attacked, as reported in other studies (e.g. Shudo and Iwasa, 2001)), while the defence remains high. We thus predict low defense/low inductibility for low encounter rates, high defence/high inductibility for moderate encounter rates, and high defence, moderate or low inductibility for high encounter rates.

There is evidence that selection pressures can influence the inducibility of defence. Many defences are not totally inducible or totally constitutive, and selection can therefore act both on inducibility and on basal level defence. For example, some of the best understood systems of defence induction involve invertebrate immune responses to their pathogens and parasites (Strand and Pech, 1995), and in particular immunity in *Drosophila* (Godfray and Hassell, 1991). Hemocyte load is one of the most important factors determining success of the Drosophila immune reaction (Eslin and Prevost, 1998; Eslin and Prevost, 1996) . During the immune response, circulating hemocytes stick to a foreign body, potentially encapsulating it. Importantly, new hemocytes differentiate as an induced response to infection, and effective hemocyte load can be amplified by as much as four- or five-fold as compared to the background constitutive load (Eslin and Prevost, 2000). Although constitutive hemocyte load does correlate with immune success in *Drosophila*, induced changes may play an important role too. Fellowes and colleagues (Fellowes et al., 1998, 1999) selected for increased resistance against two parasitoids in two separate selection experiments, and found higher constitutive hemocyte rates within the resulting resistant strains. However, despite the uniform increase in constitutive haemocyte levels, selection for greater resistance against one parasitoid did not necessarily result in a high resistance against a second parasitoid species (Fellowes et al., 1999). It seems that other factors beyond basal haemocyte level are involved in the success of the immune reaction, such as the activation system for the immune system (Carton and Nappi, 2001). For example, one *Drosophila* strain known to be resistant against the parasitoid *Leptopilina boulardi* showed the same hemocyte load before parasitism as a susceptible strain, but a lower hemocyte load and a different hemocyte morphology 15 h after infestation by the parasitoid (Russo et al., 2001). Selection for resistance can therefore act on different levels of a defence: basal levels of defence—e.g., basal haemocyte count, or induction of this defence when attacked—as suggested by differing levels or specificities of inducibility of haemocytes, perhaps due to differing mechanisms of foreign body recognition (Fellowes et al., 1999).

Another well-studied system is chemical defences in plants. Plants often possess a basal level of a variety of chemical compounds, and studies show how these levels may be amplified by herbivore attack (Maleck and Dietrich, 1999). Interestingly, different parts of a plant can have different levels of induced defence, and this may be related to the actual rates of attack by enemies on these different parts (Karban and Baldwin, 1997; Zangerl and Rutledge, 1996).

Interest in relationships between defence and group size or sociality is increasing, and one major prediction is that species living in groups should invest more in defences because they have higher encounter rates with enemies/higher transmission of pathogens (Hochberg, 1991). Depending on the type of defence investigated, this prediction may sometimes be upheld (Hochberg, 1991) and sometimes rejected (Wilson et al., 2003). A possible confounding factor is the action of 'herd immunity' (more generally, reduction in encounter rate with specialist enemies in better defended populations) together with inducible defence. Our model predicts that lower group defence and inducibility should prevail against rare enemies, and higher inducibility and group defence should be selected against common enemies. More experiments are needed to verify if these predictions.

The cost of inducibility has been extensively discussed in the literature. Some studies have failed to show such costs, whereas other empirical studies suggest that these costs may be widespread (Tollrian and Harvell, 1999). We believe it is important to point out a possible confusion in the literature, whence many references to "inducible defence costs" actually include both (1) the implementation of an induced defence (which corresponds to cost of defence in our model) and (2) the cost of inducibility itself (i.e. the cost of receptors, of amplification systems, and/or costs due to time delays in the activation of defence; Karban and Baldwin, 1997). Our model plainly shows that the absolute and relative costs of each must be identified if we are to better understand their evolutionary origins and dynamics.

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