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Induction and herbivore mobility affect the evolutionary escalation of plant defence

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ABSTRACT

Questions: Does the evolution of anti-herbivore defences depend on whether the defences are induced or constitutively expressed?

Mathematical methods: Cost–benefit model examining invasion and fixation conditions.

Key assumptions: Annual plant, haploid. Defence affects herbivore preference and performance. Induced defences are expressed only after herbivore attack and there is a lag between attack and induction. Herbivore and plant population sizes are fixed or vary as a function of the size and genetic composition of the other population. Two types of herbivores are examined: those that are immobile once they land on a plant and those that may move if plant quality changes. No costs to herbivore movement.

Predictions: Alleles at induced loci invade a population under a wider range of costs and benefits than alleles at constitutive loci. Alleles that invade at constitutively expressed loci will spread to fixation, whereas alleles at induced loci may be maintained in stable polymorphisms. Alleles at both constitutive and induced loci invade and fix under less restrictive conditions when defences are active against immobile compared to mobile herbivores.

Keywords: co-evolution, constitutive, host preference, induced resistance, parasite, pathogens.

INTRODUCTION

Herbivores and pathogens can cause reductions in plant fecundity and population sizes, and be important selective forces acting on plant populations. In response to these selective pressures, plants have evolved a wide variety of traits that serve as defences against herbivores. These traits differ in their physiological efficacy against different herbivores, as well as their expression patterns, with some defences expressed constitutively and others expressed only following damage. Several theoretical models have investigated the evolution of more effective defence traits (e.g. Rhoades, 1979; Simms and Rausher, 1987). Recently, much attention has also been devoted to understanding the relative benefits of induced versus constitutive

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expression of defences (e.g. Agrawal and Karban, 1999). Mathematical models predict two conditions under which induction offers a selective advantage over constitutive expression. One set of conditions involves costs of defence; if defences are costly and/or the probability of attack is low, then induced defences are likely to confer higher fitness than constitutive defences (e.g. Clark and Harvell, 1992; Adler and Karban, 1994; Jaremo *et al.*, 1999; Lively, 1999; Shudo and Iwasa, 2001). Alternatively, induced defences may be selectively favoured when changes in diet quality or composition negatively affect herbivore performance or preference (Adler and Karban, 1994).

Previous models have not, however, explored how the evolution of alleles that alter the physiological efficacy of defence (i.e. alleles produced through mutations in coding regions) might be affected by the pattern of expression (induced versus constitutive). In this paper, we use models to investigate whether defence alleles produced through mutations in coding regions exhibit different evolutionary dynamics depending upon whether those mutations occur at constitutive or induced loci. Clarifying the effect expression has on the evolution of defences may provide insight into the relative evolutionary stability of constitutive and induced defences, which may be important for long-term ecological and evolutionary consequences of plant–herbivore interactions.

There are several reasons to think that expression pattern may affect the evolution of defence genes, even if those genes produce identical proteins. For example, constitutively expressed defences are expected to incur costs even when herbivores are not present and thus may only confer an advantage when the probability of damage is high (Clark and Harvell, 1992; Shudo and Iwasa, 2001). In addition, theoretical (Underwood, 1999; Gardner and Agrawal, 2002) and empirical (Underwood and Rausher, 2002) work has shown that induced and constitutive defences can have different effects on herbivore population dynamics, which in turn may affect the evolution of defence (May and Anderson, 1983; Frank, 1991; Koella and Restif, 2001; Gandon *et al.*, 2002).

To investigate how expression pattern affects the evolution of defence genes, we present models describing the conditions for the invasion and fixation of alleles that increase the efficacy of defence but do not alter the pattern of expression. We do not directly compare the evolution of alleles that differ in both expression patterns and physiological characteristics. Such alleles are likely to be rare given that they would be produced only if a single mutation affected both expression and amino-acid sequence, or if two mutations, one affecting regulation and one affecting the coding region, occurred within a very short period of time (i.e. before an allele produced by the first mutation is lost from the population). We begin by analysing cases with fixed plant and herbivore population sizes, and then examine whether feedbacks between defence allele frequency and population sizes affect defence gene evolution. Moreover, because the pattern of defence expression can affect herbivore behaviour (Morris and Dwyer, 1997), which may in turn have strong effects on the efficacy of defence and the rate at which counter-defences evolve (Gould, 1984; Adler and Grunbaum, 1999), we examine two types of enemies – those that are mobile throughout the season (e.g. grasshoppers, beetles and some caterpillars) and those that are immobile once they land on a plant (e.g. leaf miners, scale insects and pathogens).

THE MODELS

Our approach is to examine whether expression pattern affects whether a new allele that increases the efficacy of defence is expected to confer a selective advantage to individuals that express that allele. Following the general form of previous cost–benefit models

(e.g. Rhoades, 1979; Simms and Rausher, 1987; Clark and Harvell, 1992; Jaremo *et al.*, 1999; Tiffin, 2000), we assume that the plant is an annual with discrete, non-overlapping generations and a haploid genome. We first examine dynamics in the plant population assuming the herbivore population is fixed for an allele that cannot detoxify or circumvent the defence – that is, the herbivore population is uniformly susceptible. Although the vast majority of plants and herbivores are diploids, a haploid model can capture the dynamics of alleles with additive gene action and simplifies the analyses (Bürger, 2000). Moreover, with additive gene action, conditions that produce stable polymorphisms in a haploid population are also expected to produce stable polymorphisms in diploid populations.

We consider three plant genotypes with alternative defensive traits: null (absence of defence), constitutive and induced, indicated by subscripts N, C and I respectively. We start by defining the mean fitness of an individual plant of genotype X ($\bar{W}_X$) as

$$\bar{W}_X = W_0 - C_X - \alpha r_{X_t} \frac{H_t}{P_t}(1 - B_X) \tag{1a}$$

where W_0 is the fitness of a null genotype (i.e. one that confers no resistance relative to the allele being evaluated) in the absence of herbivores, C_X is the cost of defence, α is the proportion of the plant's life that is subject to herbivore attack, r_{X_t} is the relative preference of herbivores for genotype X at generation t, H_t/P_t is the number of herbivores per plant at generation t, and B_X is the efficacy of the defence at reducing feeding rates (Table 1). To simplify analyses, we assume $0 = B_N < B_I = B_C \leq 1$, $0 = C_N \leq C_C = C_I \leq 1$. Because $B_N = 0$ and $B_I = B_C$, and $C_N = 0$ and $C_I = C_C$, throughout the rest of the manuscript, B_C, B_I, C_C and C_I are written without subscripts and B_N and C_N are omitted. By assuming $B > 0$, we examine only alleles that increase the efficacy of defence. The cost term (C) can be interpreted as any constraint that is independent of gene frequency (e.g. self-toxicity or allocation costs). We further assume that herbivore attack is the cue that induces expression of the inducible defence, and once defence is induced it is expressed throughout the time herbivores feed.

To examine the importance of induction in more detail we include a lag time, L, equal to the length of time (measured as a proportion of the plant's life cycle) between herbivore

Table 1. Model parameters

$\bar{W}_X$	mean fitness of genotype X (where $X = I$, N or C)
W_0	fitness of initial (undefended) allele in the absence of enemies ($W_0 = 1 + \alpha$)
p_X	frequency of plant allele X, between 0 and 1
C_X	physiological and/or ecological costs of expressing allele X
α	proportion of the time that a constitutive defence is expressed during which herbivores attack, which ranges from 0 to 1
L	time between herbivore attack and induction, which ranges from 0 to α
r_X	the relative preference of herbivores for genotype X (proportion of herbivores on genotype X relative to p_X)
B_X	efficacy of defence allele X, from 0 to 1
H_t	size of herbivore population at time t
P_t	size of plant population at time t
K_{P_t}	carrying capacity of the plant population at time t
K_{H_t}	carrying capacity of the herbivore population at time t

attack and defence expression, and assume $0 < L < \alpha$. Incorporating these assumptions into equation (1a) leads to the following expressions for individual fitness of the three defence genotypes:

$$\bar{W}_N = W_0 - \alpha r_{N_t} \frac{H_t}{P_t}$$

$$\bar{W}_C = W_0 - C - \alpha r_{C_t} \frac{H_t}{P_t}(1 - B) \tag{1b}$$

$$\bar{W}_I = W_0 - (\alpha - L)C - r_{I_t} \frac{H_t}{P_t}(L + (\alpha - L)(1 - B))$$

If herbivores are unable to discriminate between plants of different quality, then $r_{X_t} = 1$. To examine defences that affect herbivore behaviour as well as performance, we assume that herbivore preference is directly negatively correlated with the efficacy of defence (herbivores prefer high-quality plants) so that herbivores are distributed over genotypes out of proportion to the frequency of those genotypes:

$$r_{X_t} = \frac{Q_X}{\sum_X p_{X_t} Q_X} \tag{2}$$

where $Q_N = 1$, $Q_C = (1 - B)$ and $Q_I = 1$ for immobile herbivores and $Q_I = (L/\alpha) + [1 - (L/\alpha)](1 - B)$ for mobile herbivores. Thus $r_{X_t} > 1$ for plants of higher quality (preferred plants) and $r_{X_t} < 1$ for plants of lower quality. We define Q_I separately for immobile and mobile herbivores because immobile herbivores choose plants before the defence is expressed, whereas mobile herbivores are able to switch plants if they detect a change in plant quality (i.e. after induction). For simplicity, we also assume there is no cost associated with searching or switching. Describing preference in this way results in large differences in the number of herbivores that attack undefended compared with defended plants when the efficacy of defence (B) approaches 1. Values of $B \to 1$ imply that herbivores are unable to feed upon defended plants and thus strong differences in herbivore preference seem reasonable. We also note that preference is not defined when $B = 1$ and $p_C = 1$, although this does not affect our analyses for invasion or fixation.

Population size

We examined the evolution of defence under two cases. In the first case, the carrying capacities of the plant and herbivore populations are fixed. Here we assume that the plant and herbivore populations are regulated by factors outside the plant–herbivore interaction such as predators (for the herbivore) or competition (for the plant). In the second case (variable carrying capacities), the carrying capacity of the plant population is affected by the size of the herbivore population [and the carrying capacity of the herbivore population is reciprocally affected by plant quality (e.g. Underwood and Rausher, 2002)].

The size of the plant population (P_t) is described using a modified Ricker equation:

$$P_{t+1} = P_t e^{\bar{W}\left(1 - \frac{P_t}{K_{P_t}}\right)} = P_t e^{(\Sigma p_X W_X)\left(1 - \frac{P_t}{K_{P_t}}\right)} \tag{3}$$

where P_t is the size in the previous generation, $\bar{W}$ represents mean plant fitness, and K_{P_t} is the plant carrying capacity. When the plant carrying capacity is fixed, $K_{P_t} = K_{P_0}$. When the plant carrying capacity is affected by the size and composition of the herbivore population, it is defined as a function of damage caused by herbivores, $K_{P_{t+1}} = K_{P_0} - \beta(H_t - H_0)$ (equation 4), in which K_{P_0} is the carrying capacity under initial conditions and β is a coefficient for the decrease in plant carrying capacity as the herbivore population size increases, which for simplicity we fix at 0.05.

Herbivore population dynamics are governed by a similar recursion,

$$H_{t+1} = H_t e^{\lambda\left(1 - \frac{H_t}{K_{H_t}}\right)} \tag{5}$$

where λ is the herbivore population growth rate and K_{H_t} is the herbivore carrying capacity at time t. When the herbivore carrying capacity is fixed, $K_{H_t} = P_{H0}$. When the herbivore carrying capacity is variable, $K_{H_{t+1}} = P_t \sum P_{X_t} r_{X_t} Q_{X_t}$ (equation 6). Thus herbivore and plant carrying capacities are initially equal, but the herbivore carrying capacity will decrease as the average value of Q_X decreases (cf. equation 2). Unlike the preference function (r_{X_t}), for which the quality of induced plant genotypes differs between mobile and immobile herbivores, the quality of plant material herbivores consume is independent of herbivore mobility, so for equation (6), $Q_I = (L/\alpha) + [1 - (L/\alpha)]\,(1 - B)$.

The equations governing the population dynamics of both species have the potential to generate cyclic and chaotic population dynamics at high population growth rates (May, 1974). We have chosen to keep population growth rates constant and at a level such that populations closely track any changes in carrying capacities, without the potential for persistent population cycles (i.e. growth rates are given a value of 1 in equations 3 and 5). This means that when carrying capacities are fixed, population sizes for both species are constant and at their respective carrying capacities.

Analyses

To compare evolution at constitutive and induced loci, we first examine the evolution of alleles at a constitutive locus in an initially undefended population (i.e. $p_N = 1$) and then the evolution of alleles at an induced locus in an initially undefended population. For each pair of alleles, we present the conditions under which a new allele is expected to invade, under which that allele is expected to spread to fixation ($p_X \to 1$), and under which allelic dimorphisms are stable. We identify the invasion and fixation conditions by evaluating $W_X > W_N$ as $p_X \to 0$ and $p_X \to 1$, respectively. A stable polymorphism is possible when there is a set of conditions that allow an allele to invade but not go to fixation.

We used analytical solutions to examine the spread of alleles when plant and herbivore carrying capacities are fixed. Numerical simulations of recursion equations were used to examine the spread of alleles when carrying capacities vary over time or when both the plant and herbivore populations are genetically variable. Recursions were conducted in an Excel spreadsheet and run until changes in allele frequency were less than 1×10^{-5} per 1000 generations (i.e. at equilibrium). The initial conditions for the recursions were: frequency of the undefended plant = 0.999, frequency of the new defended plant = 0.001, and the initial sizes and carrying capacities of the herbivore and plant populations were set to 1000.

Preliminary analyses revealed that results were robust to differences in initial population sizes and carrying capacities.

RESULTS

Invasion and fixation at constitutive loci

At a constitutive locus, an allele with higher efficacy will confer higher fitness than a null allele, $W_C > W_N$, when

$$C < \frac{H_t}{P_t}\alpha(r_N - r_C(1 - B)) \tag{6a}$$

Regardless of herbivore mobility, this equation is equivalent to

$$C < \frac{H_t}{P_t}\alpha\left(\frac{B(2 - B)}{p_N - p_C(1 - B)}\right) \tag{6b}$$

Evaluating (6b) when $p_C \to 0$ reveals that a new constitutive allele will invade when $C < \frac{H_t}{P_t}_{p_C = 0}\alpha B(2 - B)$, and evaluating when $p_C \to 1$ reveals that a constitutive allele will fix when $C < \frac{H_t}{P_t}_{p_C = 1}\alpha\left(\frac{B(2 - B)}{(1 - B)}\right)$. Because invasion conditions are evaluated when the frequency of the new allele is vanishingly small, invasion conditions are independent of the effect the new allele has on population sizes and the invasion conditions for a new C allele are equivalent for fixed and variable herbivore carrying capacities. Moreover, when herbivore population size is invariant, equations (6a) and (6b) can be rearranged to show that the invasion condition is always more restrictive than the fixation condition ($0 < B$). Similarly, when herbivore population sizes vary with plant quality, numerical recursions show that the invasion condition is always equal to the fixation condition (Figs. 1 and 2). Therefore, regardless of effects on herbivore population size, if a constitutive allele invades it will go to fixation and stable dimorphisms are not possible. In other words, costs of constitutive alleles may prevent alleles from invading; however, if an allele invades, then costs will never prevent that allele from going to fixation. This result applies to both mobile and immobile herbivores. We reiterate that we are examining situations where herbivores are able to discriminate between defended and undefended plants. If herbivores are unable to discriminate, then defences that invade will go to fixation when population sizes are fixed, but can be maintained in a stable equilibrium if the defence reduces the size of the herbivore population [results not shown (Tiffin, 2000)].

Invasion and fixation at induced loci, immobile herbivores

At an induced locus, an allele with higher efficacy will confer higher fitness than a null allele, $W_I > W_N$, when

$$C < \frac{H_t}{P_t}\frac{1}{\alpha - L}(r_N\alpha - r_I(L + (\alpha - L)(1 - B))) \tag{7a}$$

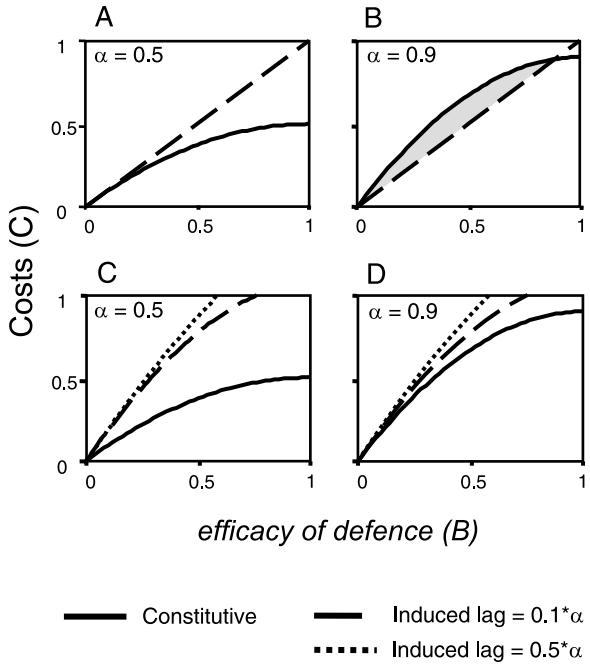

Fig. 1. Invasion conditions for alleles at induced (dashed and dotted lines) and constitutive (solid lines) loci when plants are attacked by immobile (A and B) or mobile (C and D) herbivores with fixed carrying capacities. In (A) and (C), herbivores attack for 50% of the plant's life ($\alpha = 0.5$); in (B) and (D), $\alpha = 0.9$. For all alleles fixation conditions are less or equally restrictive to invasion conditions. In (B), the shaded area indicates the range of values that allow alleles at induced but not constitutive loci to invade. In (A), (C) and (D), alleles at induced loci always invade under less restrictive conditions than alleles at constitutive loci.

This condition is dependent on herbivore mobility and therefore invasion and fixation conditions must be evaluated separately for immobile and mobile herbivores. With immobile herbivores, equation (7) simplifies to $C < (H_t/P_t)B$, which with fixed plant and herbivore carrying capacities is independent of allele frequency. Thus an allele that invades will always fix (Figs. 1 A,B). When plant and immobile herbivore carrying capacities vary, the condition for the invasion of an allele at an induced locus (the right side of 7a evaluated when $p_I \to 0$) will be less restrictive than the conditions for fixation (the right side of 7a evaluated when $p_I \to 1$) when $1 > \alpha - (\alpha - L)B$, which is always true. Therefore, stable polymorphisms are possible. Numerical solutions for the recursion equations show polymorphisms will be stable over a wide range of parameter values and that this range is larger with longer lags between attack and induction (Figs. 2 A,B).

Invasion and fixation at induced loci, mobile herbivores

With mobile herbivores, equation (7a) becomes

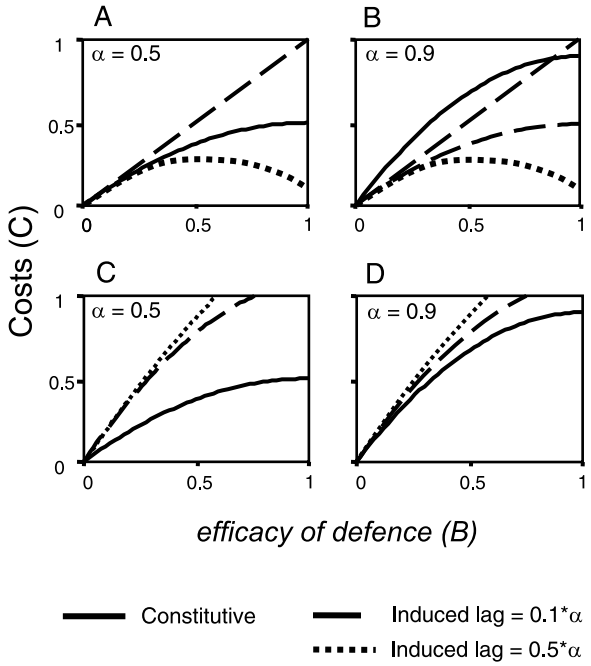

Fig. 2. Invasion and fixation conditions for alleles at induced (dashed and dotted lines) and constitutive (solid lines) loci when plants are attacked by immobile (A and B) or mobile (C and D) herbivores with varying carrying capacities. In (A) and (C), herbivores attack for 50% of the plant's life ($\alpha = 0.5$); in (B) and (D), $\alpha = 0.9$. For immobile herbivores the invasion conditions are independent of α. In (A), the fixation conditions for induced alleles with 0.1α are equivalent to the invasion and fixation conditions for a constitutive allele. In (B), the upper dashed line is the invasion conditions, the lower dashed line is the fixation conditions. Fixation conditions for alleles active against mobile herbivores are less restrictive or equal to the invasion conditions (not shown). For induced defences active against immobile herbivores, the regions below the solid but above the dashed lines are combinations of costs and efficacies that result in stable dimorphisms.

$$C < \frac{H_t}{P_t}\frac{1}{\alpha - L}\left(\frac{\alpha - \left(\frac{L}{\alpha} + \left(1 - \frac{L}{\alpha}\right)(1 - B)\right)(L + (\alpha - L)(1 - B))}{p_N + p_I\left(\frac{L}{\alpha} + \left(1 - \frac{L}{\alpha}\right)(1 - B)\right)}\right) \tag{7b}$$

Comparing this inequality when $p_I \to 0$ and $p_I \to 1$ reveals that the conditions under which an allele invades are less restrictive than the conditions for fixation when $\underset{p_I \to 0}{\frac{H_t}{P_t}}\left(1 - B + \frac{L}{\alpha}B\right) > \underset{p_I \to 1}{\frac{H_t}{P_t}}$. If H_t and P_t are fixed, this inequality simplifies to $L - \alpha > 0$, which is never true; therefore, induced alleles that invade will spread to fixation (Figs. 1 C,D). Similarly, because the quality of plants with inducible defences is lower than the quality

of plants with no defences, $\frac{H_t}{P_t}_{p_I \to 0} > \frac{H_t}{P_t}_{p_I \to 1}$, and thus polymorphisms are also not stable with varying populations sizes.

Comparing evolution at constitutive loci to evolution at induced loci

Having calculated invasion conditions, we can compare directly whether new alleles will invade under less restrictive conditions at constitutive or induced loci – in other words, are costs more likely to impede the invasion of new alleles at induced or constitutive loci? When plants are attacked by immobile herbivores, alleles at constitutive loci invade under less restrictive conditions than alleles at induced loci when

$$\frac{H_t}{P_t}_{p_C \to 0} \alpha(2 - B) > \frac{H_t}{P_t}_{p_I \to 0} \tag{9}$$

or $\alpha(2 - B) > 1$, which is true when α is large or B is small. Therefore, constitutive alleles can invade under less restrictive conditions when herbivores attack during most of the time constitutive defences are expressed and the effect of defence is not large (Fig. 3). Because invasion conditions are evaluated when the frequency of the new allele is negligible, invasion conditions are unaffected by the influence defence has on population sizes.

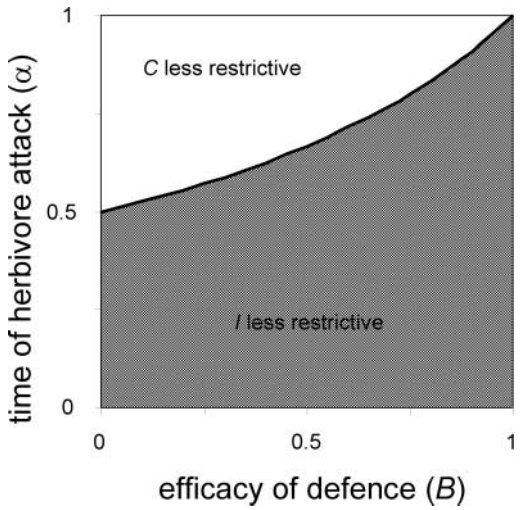

Fig. 3. The effects of expression on the conditions under which alleles active against immobile herbivores are able to invade. Above the solid line, alleles at constitutive loci invade under less restrictive conditions (can incur greater costs) than alleles at induced loci. In the grey region, below the solid line, alleles at induced loci invade under less restrictive conditions (can incur greater costs). With mobile herbivores, conditions for invasion at induced loci are always less restrictive than conditions of invasion at constitutive loci.

When plants are attacked by mobile herbivores, alleles at constitutive loci would invade under less restrictive conditions than alleles at induced loci when

$$\frac{H_t}{\underset{p_C \to 0}{P_t}} \alpha(2 - B) > \frac{H_t}{\underset{p_I \to 0}{P_t}} \left(2 - B\frac{(\alpha - L)}{\alpha} + \frac{\alpha}{(\alpha - L)}\right) \tag{10}$$

a condition that is never satisfied. Thus when plants are attacked by mobile herbivores, alleles at induced loci always invade under less restrictive conditions than alleles at constitutive loci. The same result is found with numerical recursions. Once again, because invasion conditions are independent of effects on population size, this is true for both fixed and variable carrying capacities.

DISCUSSION

Most models examining the evolution of constitutive and induced defences have focused on clarifying the conditions under which induced expression is selectively favoured over constitutive expression. In other words, these models have analysed the conditions under which a mutation that affects gene regulation (induced or constitutive) but not physiological efficacy is selectively advantageous. In this paper, we compare the conditions under which mutations that increase the efficacy of defence will be selectively advantageous when those mutations occur at constitutive compared to induced loci. Our results indicate that expression pattern affects the conditions under which more effective defence alleles are able to invade and the probability for stable polymorphisms. Moreover, we find that herbivore mobility and the regulation of plant and herbivore population sizes affect the evolution of induced but not constitutively expressed defences. Specifically, we find that: (i) constitutive alleles that invade will go to fixation; (ii) induced alleles that invade either fix, if plant and herbivore carrying capacities are fixed, or may be maintained in a stable polymorphism, if carrying capacities vary as a function of population size and genetic composition; and (iii) alleles at induced loci invade under less restrictive conditions than alleles at constitutive loci, except when herbivores are immobile and absent for most of the time constitutive defences are expressed.

Previous models of host–enemy interactions have shown that defence gene polymorphisms can be stable if the strength of selection acting on defences changes as allele frequency alters enemy genotype frequency (Mode, 1958; Gillespie, 1975; Clarke, 1976; May and Anderson, 1983; Frank, 1992) or population density (Clarke, 1976; Antonovics and Thrall, 1994). We find that polymorphisms at constitutive loci are not stable even when plant defence affects herbivore population sizes, at least as long as herbivore population growth rates are low enough not to cause population cycles (when herbivore populations cycle dramatically, unstable polymorphisms are sometimes maintained without fixation for long periods; results not shown). The reason for the different predictions regarding stable polymorphisms appears to be our assumption that defence affects both herbivore performance and herbivore preference – a relationship that results in herbivores preferentially attacking undefended plants. When herbivore preference and performance are completely uncoupled, stable polymorphisms are possible for defences that affect herbivore performance (antibiosis) but not those that only affect herbivore preference [results not shown (Tiffin, 2000)]. The reason for this is that an increase in the frequency of antibiosis defence suppresses herbivore population sizes, thereby reducing the benefits of defence. Reduced herbivore population sizes do not, however, reduce costs. The net effect therefore is a reduction in the relative fitness benefits

associated with defence alleles. In contrast, alleles that affect only preference have no effect on herbivore population sizes and simply shift herbivore loads towards undefended (more preferable) individuals. In the absence of frequency-dependent shifts in preference, increased frequency of defence alleles that affect only preference do not decrease the relative benefits associated with defence and those alleles that invade will go to fixation.

Our assumption that defence affects both herbivore preference and performance also explains why the evolution of alleles at induced loci is dependent upon herbivore mobility. When herbivores are mobile, they respond to defence induction by moving to undefended plants. With immobile herbivores, in contrast, herbivores land on plants before expression and are unable to move after induction occurs. Therefore, the evolution of induced defences active against immobile herbivores depends upon the effects of defence on performance only. Not surprisingly, induced alleles active against immobile herbivores evolve in a qualitatively similar manner to constitutive alleles that affect only performance (not shown), and stable polymorphisms are possible. Interestingly, the range of cost values over which polymorphisms are stable actually increases with increasing efficacy of defence (Fig. 2B). The reason for this is that defences with higher efficacy suppress herbivore population sizes more than less efficacious defences, and as population sizes fall the benefits of defence are reduced, resulting in reduced fitness benefits associated with defence.

In addition to being affected differently by herbivore mobility, evolution at constitutive and induced loci is affected differently by costs of defence. Alleles at induced loci are selectively advantageous over a greater range of costs than alleles at constitutive loci. The reason for this is simple: induced defences incur no costs before herbivores attack or during the lag period between attack and expression. This is only true, however, when the effect of defence on herbivore feeding is not large and herbivores are absent for most of the time constitutive defences are expressed (Fig. 3). When herbivores are immobile and present during most of the time defence is expressed, then the costs incurred prior to herbivore attack are more than offset by the effect of defences on herbivore choice (constitutive but not induced defence causes herbivores to preferentially attack undefended plants), resulting in higher herbivore loads on those plants.

The likelihood of this model's predictions being true depends upon the relationships among defence traits, herbivore preference, herbivore performance and herbivore movement following defence induction. We have assumed that preference and performance are tightly correlated and that mobile herbivores move away from plants following induction of defence. These assumptions will not apply to all plant–herbivore interactions. However, empirical studies of oviposition preference and offspring performance reveal evidence for correlations in some systems (reviewed in Karban and Agrawal, 2002). Moreover, there is empirical evidence that preference and performance of a single generation are correlated for some plant–herbivore systems (e.g. Haddad and Hicks, 2000; Steppuhn *et al.*, 2004). Although studies of the effect of induced resistance on herbivore movement are relatively rare, induced resistance can affect herbivore movement within plants in the laboratory (Barker *et al.*, 1995) and preference for plants (taxis towards undefended plants) in field choice tests (Thaler *et al.*, 2001). These empirical results indicate that our assumptions of a coupling in the relationships between preference and performance, and movement of herbivores away from plants following induction, are reasonable in at least some cases.

Differences in invasion and fixation conditions may translate into considerable differences in the long-term stability of constitutive and induced defences. The proportion of mutations that invade a population will be directly related to the range of cost–benefit

values over which new alleles are selectively favoured. Therefore, our model predicts that a greater proportion of mutations at induced than constitutive loci will be selectively favoured when defences are active against mobile herbivores. In contrast, for defences active against immobile herbivores a greater proportion of induced alleles will be selectively advantageous only if herbivores attack for a small portion of the plants' life and the efficacy of defence is large. Finally, for induced loci a greater proportion of mutations will be selectively advantageous when defences are active against herbivores whose carrying capacities are not strongly affected by plant quality, regardless of herbivore mobility. These predictions for long-term evolutionary rates cannot be tested through empirical manipulations of contemporary populations. However, DNA sequence data, which are providing new insight into the long-term evolution of defence genes (e.g. Stahl *et al.*, 1999; Bishop *et al.*, 2000; Tiffin *et al.*, 2004), may lead to the identification of general trends in the relative evolutionary rates of constitutive and induced defences, thereby providing a test of this model.

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