Insularity and adaptation in coupled victim–enemy associations

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Abstract

Employing a mathematical model we show how insularity, genotypic interactions and victim life-history/demography can influence adaptation in a simple enemy–victim interaction where genotypes migrate between a large source and a smaller, initially unoccupied, isolated habitat. We find that when there are explicit costs to heightened enemy virulence and victim resistance, large/close islands resemble their immigration sources, whereas small and/or distant islands tend to be occupied only by the least defended victims and least virulent enemies. In a model with no explicit cost to genotypic identity, frequencies do not differ on average between source and island. Despite these trends in genotype frequencies, for a range of realistic conditions, both cost and cost-free genotypic interactions yield an increase in the frequency of resistant encounters as a function of isolation. Moreover, in models with explicit costs, maximal island to island variation in genotypic frequencies is found on islands of intermediate distance from the source. In contrast, the model without explicit costs produces more variable communities, attaining maximum variability in genotypic frequencies at the most isolated islands. We hypothesize that adaptive patterns in mainland–island comparisons may differ substantially from those generated by centre-periphery comparisons in continental systems.

Introduction

Insularity, or the extent to which populations are isolated from immigration, can have important consequences for population densities, genetics, and demographic and life-history characteristics. Indeed, island species often exhibit similar biological particularities to one another, called the 'island syndrome' (discussions in Adler & Levins, 1994; Grant, 1998; Whittaker, 1998). The island syndrome has several facets. First, island populations tend to be less genetically diverse than their mainland counterparts (Frankham, 1997). At least two factors may contribute: island populations (1) originate from a small number of immigration events and (2) are generally small compared with mainland populations, giving rise to small effective population sizes. Secondly, island populations are generally sedentary. Once individuals of a newly arriving species have immigrated to an island, there is often intense selection for reduced dispersal (Darlington, 1943; Carlquist, 1974; Roff, 1990, 1994; McNab, 1994; Steadman, 1995; Cody & Overton, 1996; Peck, 1996; Shaw, 1996). Thirdly, islands generally have impoverished floras and faunas, a possible reason being reduced immigration (MacArthur & Wilson, 1967; Williamson, 1981). Fourthly, island populations tend to show higher densities than their mainland counterparts. This is thought to be in part because of the lower limiting effects on island populations due to impoverished natural enemy (Kramer, 1946; Case, 1983) or competitor (Williams, 1972; Case, 1978; Feinsinger & Swarm, 1982; Roughgarden, 1995) communities: the so-called 'density-compensation' effect (Grant, 1966; MacArthur *et al.*, 1972; Yeaton & Cody, 1974; Emlen, 1979; Wright, 1980). Fifthly, island species commonly differ from mainland counterparts by having evolved different body sizes (Williamson, 1981; Brown *et al.*, 1993; Grant, 1998; Whittaker, 1998) and life-history traits such as high survival rate, low fecundity and deferred age of maturity (e.g. Kramer, 1946; Blondel, 1985; Wiggins *et al.*, 1998).

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An additional, less studied component of the island syndrome is that island populations tend to show less defences against their natural enemies, ostensibly because islands tend to have fewer predators and parasites than similar mainland sites (Lack, 1976; Adler & Levins, 1994; Paulay, 1994; Whittaker, 1998; see also Holt *et al.*, 1999). For example, Schoener (1987) studied plant–herbivore interactions using natural variation in island size and isolation for the buttonwood *Conocarpus erectus* and its insect enemies on small islands in the Bahamas. The highly pubescent leaves of the 'silver' phenotype of buttonwood are less susceptible to herbivory than the less pubescent leaves of 'green' buttonwood (reviewed in Schoener, 1987). Schoener (1987) found that larger islands had a higher percentage of pubescent plants than smaller islands, probably because larger islands have more herbivores. Furthermore, islands nearer the mainland had a higher frequency of pubescence, apparently because of immigration from the mainland. Finally, islands with abundant lizards (predators of foliage herbivores) had a smaller percentage of pubescence than islands without lizards. Schoener (1987) concluded that insularity affected the degree of herbivory and the degree of anti-herbivore defence in this system.

A second example is a study of variation in the major histocompatibility complex (MHC) in the endemic Australian rat *Rattus fuscipes* (Seddon & Baverstock, 1999). The MHC complex is the most variable genetic region of the vertebrate genome, and different haplotypes have been implicated in molecular mechanisms of successful anti-parasite defence (Klein, 1990). Seddon & Baverstock (1999) analysed the variability in MHC haplotypes for 14 island and two mainland populations. They found extensive genetic variability, although this was considerably reduced on small islands where often only one or two haplotypes persisted. The link between genetic variability and parasitism, if there is any, remains to be determined for this particular system.

A final example is a study of parasite virulence and host defence, following the introduction of avian malaria into native bird populations of the Hawaiian archipelago (van Riper *et al.*, 1986). Although these parasites have now exterminated endemic species in disturbed habitats of the lowland, many endemics still remain in prime natural habitat in the mountains. Thus, as long as the vectors transmitting the parasites cannot penetrate the native habitats, the endemic species are safe from such introduced diseases. Some native bird populations have apparently developed resistance to introduced malarial parasites, as appears to be the case for the omao, *Myadestes obscurus*, which is now able to coexist with both the mosquito vectors and the malarial parasite (Ralph & Fancy, 1994). This observation suggests that island extinction is not a ubiquitous outcome of rare immigration events by virulent parasites.

The island syndrome is an extreme example of the more general phenomenon of evolution over spatially heterogeneous landscapes. Reciprocal selection may vary predictably over geographical ranges, depending on the relative forces of gene flow, genetic drift, and the dynamics of extinction and colonization (Thompson, 1999). Several theoretical studies have examined evolutionary patterns in antagonistic associations over explicitly spatial settings (Gandon *et al.*, 1996; Hochberg & van Baalen, 1998; Lively, 1999; Gomulkiewicz *et al.*, 2000; Nuismer *et al.*, 2000). For example, Gandon *et al.* (1996) found that when the migration rate of one antagonist or the other (but not both) was sufficiently low, then the faster-migrating species tended to be better adapted locally as compared with any other arbitrary site in their metapopulation setting. Hochberg & van Baalen (1998) employing an optimization approach showed that as long as migration rates were low, predators and their prey equilibrated at higher frequencies of virulent and resistant genotypes in areas where the prey's growth rate was predictably high as compared with more marginal sites for prey persistence. These authors also found that prey tend to exhibit higher levels of resistance to their predators in marginal compared with productive sites for prey population growth.

The aim in the present study is to develop a quantitative basis and testable predictions for antagonistic coevolution on islands and on mainland habitat fragments. This is a rapidly growing area of research (see Thompson, 1999) and as a point of departure, we investigate a biological scenario where migrants from a persistent source colonize an isolated patch on which victims and enemies are initially absent. Although this is a simplification of real systems, credence is lent by Harrison's (1991) suggestion that the regional persistence of species is likely to depend on the presence of a small number of large patches, rather than networks of many, extinction-prone patches. Small habitat patches do nonetheless exist, and it would be interesting to know to what extent communities these should resemble their sources, and what variables may be responsible for any differences.

We begin by developing a micro-evolutionary model of an enemy–victim association on two patches coupled by gene flow, with one patch representing a large island or mainland habitat (referred to as the 'source') and the other a smaller, isolated patch (the 'island'). We then consider how distance, size, migration and mutation rates, and the victim's intrinsic rate of increase affect adaptation for two contrasting models of genotypic interactions. We finally discuss our findings in the light of the island syndrome and more generally enemy–victim coevolution over heterogeneous landscapes.

Methods

Population model

The mathematical formalism integrates a series of frequently used characterizations of antagonistic interactions between victims and their monophagous enemies (May, 1976; Hassell, 1978). Although the underlying assumptions are simple, the full model is complex, and we proceed in its analysis by establishing a baseline model, followed by the variation of proxy variables for insularity. The model is not intended to mimic any single type of system and would have to be modified to simulate specific biological systems.

The two species interact on each of the two patches, connected by gene flow between the 'source' (which is sufficiently large so that genotype extinctions never occur) to a smaller 'island' of radius v and distance d from the source (where genotype extinctions can occur). Insularity in our model therefore directly intervenes in the colonization of the island (given its distance from the source and its size) and the extinction of local genotypes (given the size of the island).

The densities of the haploid populations of the enemy and victim on the island are, respectively, P_t and N_t at time t. The population algorithm is comprised of three sequential processes for each arbitrarily small time increment t to $t + 1$: (i) density dependent and density independent population changes, (ii) extinction, and (iii) immigration. The same algorithm is used to model dynamics on the source, with the exception that the size of the source is effectively infinite, and so local extinctions do not occur (see below).

Population changes

During the period Δ, the victim is subject to two sequential forms of density dependence, followed by reproduction. First, the victim's population may be taxed by the enemy (controlled by the encounter parameter a), where it is assumed that the damage done by the latter is randomly distributed over the victim population (Hassell, 1978). Once these victims are removed from the population a second form of density dependent limitation acts, namely self-limitation (e.g. intraspecific competition). We assume a Ricker function for this process, with intensity parameter θ_N. Once these density dependent factors have acted, the victim population reproduces at rate r. Although not the main focus of the present study, the population dynamics of the ecological model were observed to be more erratic with large r (beyond c 0.3 in the studies presented below) and this behaviour is consistent with other models of this type (e.g. Gandon *et al.*, 1996).

The demographic model takes the form

$$N_{t+\Delta} = \xi_N \exp\{r - \theta_N \xi_N\}, \quad (1)$$

$$P_{t+\Delta} = \xi_P \exp\{-\theta_P \xi_P\}, \quad (2)$$

where

$$\xi_N = N_t \exp\{-aP_t\} \quad (3)$$

is the density of victims surviving exploitation and

$$\xi_P = P_t + cN_t(1 - \exp\{-aP_t\}) \quad (4)$$

is the summed density of enemies and their progeny. The parameter c is the number of enemies produced per victim attacked, and as the impact of the enemy on its victim varies in similar (although not identical) ways with increases in c and in a (but see Heimpel, 2000), for simplicity we take $c = 1$. Note that the enemy population grows as a direct consequence of exploiting the victim population and that after growth has occurred enemies are self-limited (with parameter θ_P).

Extinction

We assume that at the end of this demographic period, a population on the island goes extinct if its numbers (the area πv^2 multiplied by density) fall below unity. We restrict our study to this highly simplified characterization of the effects of local extinction, and future studies should encompass more realistic models which incorporate individual probabilities of reproduction and survival (Halley & Iwasa, 1998).

Immigration

Once local extinction is assessed in both species, gene-flow potentially occurs from the source to the island. A single individual of a genotype currently absent from the island, arrives only if a random number drawn from a uniform distribution is less than C_N and C_P for the victim and enemy, respectively. C_N and C_P are determined by the current densities on the source habitat and are functions of the ratio of island diameter ($2v$) and distance (d) from the source, whence

$$C_N = (1 - \exp\{-I_N N_{\text{source}}\})(1 - \exp\{-2v/\pi d\}), \quad (5)$$

$$C_P = (1 - \exp\{-I_P P_{\text{source}}\})(1 - \exp\{-2v/\pi d\}), \quad (6)$$

where N_{source} and P_{source} are the current densities of victim and enemy on the source. The first term in parentheses is an exponentially decreasing function of source population density and represents the effect of density on the pool of potential colonists. The constants I_N and I_P relate population densities to actual numbers of immigrants, and to their propensity to leave the source environment. The second term relates the effect of target distance and size on the probability that an immigrant from the source reaches the target. This process assumes that islands are not obstructed by others, and that the probability of contacting an island is an exponential function of the part of the 180° perimeter at distance d occupied by the island.

The resultant populations after the immigration episode are N_{t+1} and P_{t+1}. The algorithm then starts again with population changes (eqns 1–4).

Evolutionary models

The population model developed above is now extended to interactions between different clones of each species, where eqns 1 and 2 become

$$N^0_{t+\Delta} = \xi^0_N \exp\{r - \theta_N \xi^0_N\}, \quad (7a)$$

$$N^1_{t+\Delta} = \xi^1_N \exp\{r - X_N - \theta_N \xi^1_N\}, \quad (7b)$$

$$P^0_{t+\Delta} = \xi^0_P \exp\{-\theta_P \xi^0_P\}, \quad (8a)$$

$$P^1_{t+\Delta} = \xi^1_P \exp\{-X_P - \theta_P \xi^1_P\}. \quad (8b)$$

Most of our analysis employs the gene-for-gene (GFG) model for genotypic interactions; however, as a check on the patterns produced we also analyse a matching-allele (MA) model. For discussion of these two models, see Thompson & Burdon (1992), Frank (1994) and Parker (1996).

Gene-for-gene model

The simple form of the GFG model employed here partitions the enemy and the victim each into two subpopulations: one which interacts with both subpopulations of the antagonist and one which interacts with only one type of antagonist. In the case of the enemy, the 'virulent' type can exploit both types of victim (susceptibles and resistants). Virulence comes at an explicit cost X_P to the growth rate of the virulent enemy. For the victim, the susceptible type can be attacked by both virulent and avirulent enemies, whereas the resistant type always completely defends itself (incurring a cost X_N) against avirulent enemies. We assume that virulent enemies successfully attack all resistant and susceptible victims encountered, whereas avirulent enemies can only exploit susceptible victims (Table 1). Furthermore, we assume random and independent attack distributions by virulent and avirulent enemies over susceptible victims.

The interactions are represented by

$$\xi^0_N = N^0_t \exp\{-aP^1_t\} \exp\{-aP^0_t\}, \quad (9a)$$

$$\xi^1_N = N^1_t \exp\{-aP^1_t\}, \quad (9b)$$

$$\xi^0_P = P^0_t + (N^0_t - \xi^0_N)\eta_0, \quad (10a)$$

$$\xi^1_P = P^1_t + (N^0_t - \xi^0_N)(1 - \eta_0) + (N^1_t - \xi^1_N). \quad (10b)$$

Table 1 Fraction of victim clones encountered by enemy clones that are successfully attacked for the (a) gene-for-gene (GFG) and (b) matching-allele (MA) models.

		Avirulent enemies	Virulent enemies
a	Susceptible victims	1	1
	Resistant victims	0	1
		Enemy allele 0	Enemy allele 1
b	Victim allele 0	1	0
	Victim allele 1	0	1

where $\eta_0 = P^0_t / (P^0_t + P^1_t)$ is the frequency of avirulent enemies, superscripts '0' indicate susceptible victims or avirulent enemies, whereas '1' indicates resistant victims or virulent enemies. Note that eqns 10a and 10b assume that when a single susceptible victim is attacked by both enemy types, only one enemy prevails, the winner occurring in proportion to its frequency.

Matching allele model

The MA model employed here is structurally simpler that the GFG model (Table 1). We assume that (1) each of the two subpopulations of the enemy can attack only one, different, subpopulation of the victim (i.e. a given enemy clone attacks a given victim clone with a matching number, whereas a victim clone resists an enemy clone with a different number), and (2) there are no explicit differential costs between subpopulations of either species ($X_n = X_p = 0$). Thus, eqns 9 and 10 become

$$\xi^0_N = N^0_t \exp\{-aP^0_t\}, \quad (11a)$$

$$\xi^1_N = N^1_t \exp\{-aP^1_t\}, \quad (11b)$$

$$\xi^0_P = P^0_t + (N^0_t - \xi^0_N), \quad (12a)$$

$$\xi^1_P = P^1_t + (N^1_t - \xi^1_N). \quad (12b)$$

Immigration, mutation and extinction

Immigration of genotypes from source to island follows the same rules as outlined above in the population model, that is, a single genotype can only immigrate and potentially colonize if it is currently absent from the island. Furthermore, for both GFG and MA models we assume that at reproduction, there are constant probabilities M_N and M_P that any given new-born victim and enemy, respectively, will mutate from one genotype to the other. Mutation takes place if a random number from a uniform distribution is less than M. We assume that mutation is sufficiently infrequent such that at most one individual mutates per time step t and that mutation is equally likely between genotypes. Finally, after survival, reproduction and mutation, each of the four subpopulations can go locally extinct and/or can colonize the island if not already present. Other rules for each subpopulation are the same as outlined in the population model.

Numerical simulation methods

Because of the probabilistic nature of immigration and mutation, the densities and frequencies of enemy and victim genotypes varied from simulation to simulation. We therefore conducted 100 replicates of each set of conditions. The mean and standard errors of these 100 runs were readily interpretable using a baseline set of model parameters that resulted in all four genotypes persisting at positive densities on the source (Table 2) and then varying parameters to observe their influences. Extensive simulations of other parameter sets resulting in

Table 2 Parameters and their baseline values employed in this study.

Process	Symbol	Value
Net rate of population increase	r	0.1
Encounter rate	a	0.025
Cost to victim resistance	X_N	0.025
Cost to enemy virulence	X_P	0.025
Host density dependence	θ_N	0.01
Enemy density dependence	θ_P	0.01
Victim migration parameter	l_N	0.05
Enemy migration parameter	l_P	0.05
Victim mutation rate	M_N	0.000001
Enemy mutation rate	M_P	0.000001
Distance to island	d	50000
Radius of island	v	50

all four genotypes persisting on the source after 100 000 time steps, yielded the same basic patterns presented below. In this baseline parameter set, we employ immigration rates which exceed mutation rates by approximately one to two orders of magnitude depending on island distance and size.

Each numerical simulation was conducted as follows. First, populations on the source were established by iterating the algorithm for 100 000 time steps, and in each time step maintaining the density to at least one individual. (Although never observed, we cannot exclude the possible existence of two or more alternative equilibria with all four genotypes present.) Secondly, the populations of all four genotypes on the source were recorded at generation 100 000 and used as initial conditions for the remainder of the simulation. The initial densities of the populations on the island were set to zero and genotypes were thereafter permitted to migrate from source to island. Unless otherwise specified, each simulation was conducted over 30 000 time steps, which was sufficient time for repeatable patterns to be produced. Densities were free to vary on both source and island. Population indices were recorded at time step 30 000.

Results

Rejoining points made by Paulay (1994), the time elapsed since the extinction of both species on the island (e.g. following a major environmental disturbance) has important influences on population patterns (Fig. 1). Total victim population density is maximal at intermediate times, whereas enemy density rises and eventually asymptotes with time (Fig. 1a). These patterns reflect the necessity that victims colonize islands before their (monophagous) enemies. Figure 1(b) also illustrates this effect where, on average, the number of victim genotypes always exceeds enemy genotypes. This difference is maximal at intermediate times after immigration commences and by time-step 80 000, both genotypes of both

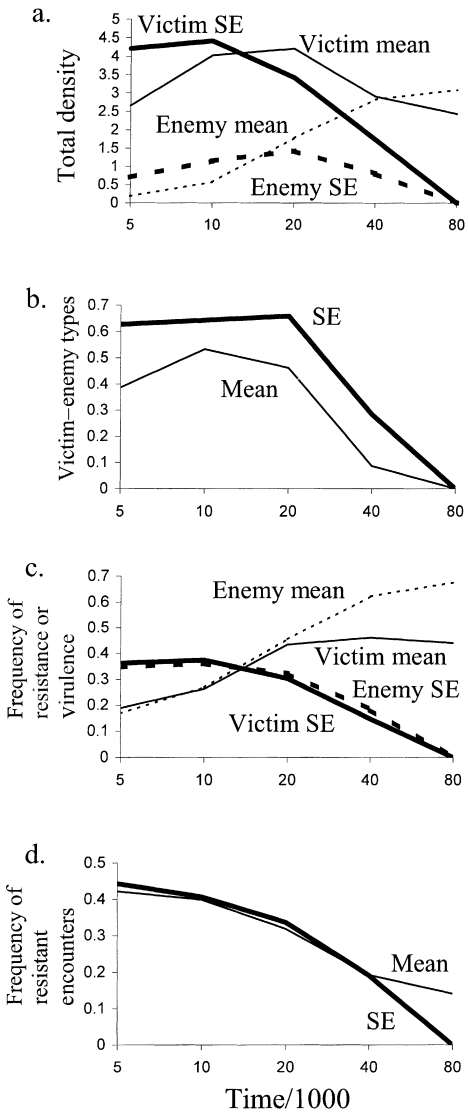

Fig. 1 Effect of sampling time postdisturbance on (a) total population densities on the island, (b) number of victim–enemy genotypes, (c) frequencies of resistant victims and virulent enemies, and (d) frequency of resistant encounters between enemy and victim. All population parameters are recorded from the island. Each of the five sampling points represents a separate set of at least 100 numerical simulations, based on gene-for-gene model (Table 1a). Frequency of resistant encounters calculated as frequency of resistant victims × frequency of avirulent enemies (Table 1a), SE = standard error. Parameter values are as in Table 2.

species are observed at a point equilibrium in all simulations (see standard errors of the island sample in Fig. 1a). This point equilibrium is the same as is observed on the immigration source. Moreover, mean frequencies of virulent enemies and resistant victims increase with time, indicating that these (costly) strategies tend to be the last genotypes to establish (Fig. 1c). Because enemy genotypes tend to invade after victims have established

and been selected for decreased resistance, the frequency of resistant encounters tends to decrease with time (Fig. 1d).

Whereas there is general agreement that island distance is an important parameter influencing immigration rates, the effect of island size is more open to discussion (e.g. Connor & McCoy, 1979; Brown & Lomilino, 1998). We therefore concentrate for the remainder of the study on island distance as the main biogeographical parameter associated with insularity. We nevertheless employ eqns 5 and 6 when investigating the effects of island size, below.

Patterns produced as a function of decreasing distance are essentially the same as those found with increasing time after immigration commences (cf. Figs 1 & 2). This highlights the similar 'currencies' represented by isolation and time (e.g. island age) in the population biology of victim–enemy interactions modelled here.

Specifically, the density of victims is, on average, maximal for islands of intermediate distance, whereas that of enemies decreases with distance (Fig. 2a). This is because of the aforementioned colonization bias that results in enemy genotypes tending to become more limiting than victim genotypes at intermediate distances (Fig. 2b). Variability in the population densities of enemies and their victims is maximal at intermediate distances (Fig. 2a), because many close islands have attained the same equilibrium genotypic community configuration, whereas far islands tend to contain victim genotypes only. Islands at intermediate distance can contain any of several possible sets of one, two and three genotype communities (not shown), and this is reflected by the mean and errors of the number of victim genotypes minus the number of enemy genotypes (Fig. 2b).

Moreover, the frequencies of resistant victims and virulent enemies tend to decrease with distance and sampling errors are maximal at intermediate distances (Fig. 2c). These results are explained by the fact that at close distances islands are effectively samples from the source and they rapidly equilibrate. At far distances, the victim occupies the island first, tends to be selected for susceptibility, and a subset of these islands are subsequently colonized by either an avirulent or a virulent enemy (note that the frequency of resistant genotypes on enemy-occupied islands actually increases; not shown; see below). Because of limited opportunities for immigration, these far islands tend to be more equilibrated ecologically than are islands at intermediate distances.

Finally, despite the fact that the frequency of resistant genotypes decreases with distance, interestingly, the frequency of resistant encounters increases with distance (Fig. 2d). In effect, as colonization begins, there is selection for victim susceptibility on islands harbouring both victim types (through migration or mutation). As the enemy begins to colonize, it initially tends to be

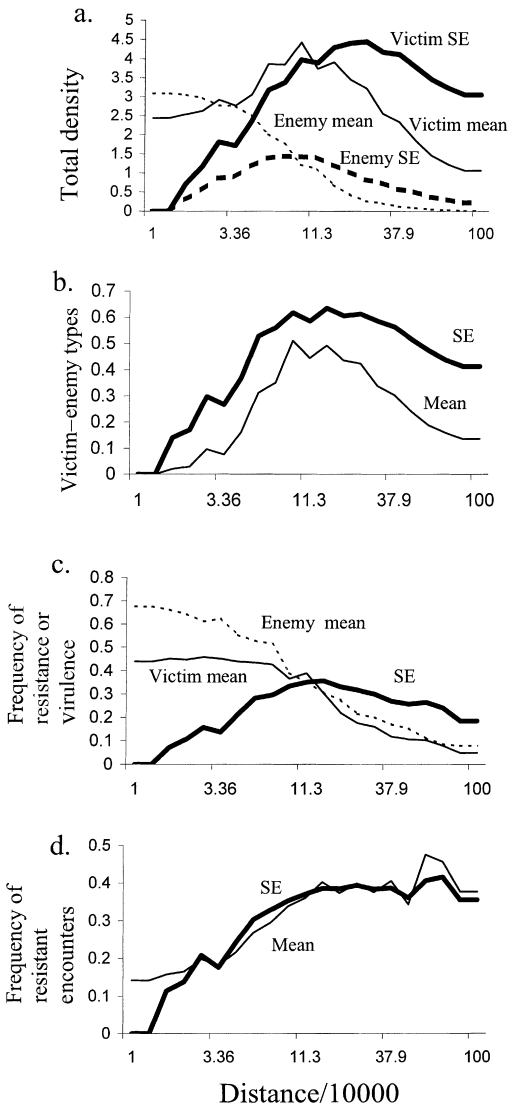

Fig. 2 Effect of island distance from source on population parameters. Captions (a)–(d) and parameters as in Fig. 1. Population parameters recorded at 30 000 time steps. Standard error (SE) curves of genotypic frequencies were very similar for both species and so, for simplicity, only one SE curve is shown in caption (c).

selected for avirulence. These avirulent enemies, in turn, select for victim resistance, which eventually then selects for enemy virulence. At any given distance, through time there is, therefore, a wave of states as the genotypic community is constructed, going from relative resistance to susceptibility (see Fig. 1c,d). The robustness of this pattern is suggested by its persistence even when the cost to enemy virulence (X_P) was lowered to 1% of its canonical value (Table 2) (not shown).

Evidently larger island size is associated with increased immigration and mutation and decreased local extinction, and therefore it shifts incomplete genotypic

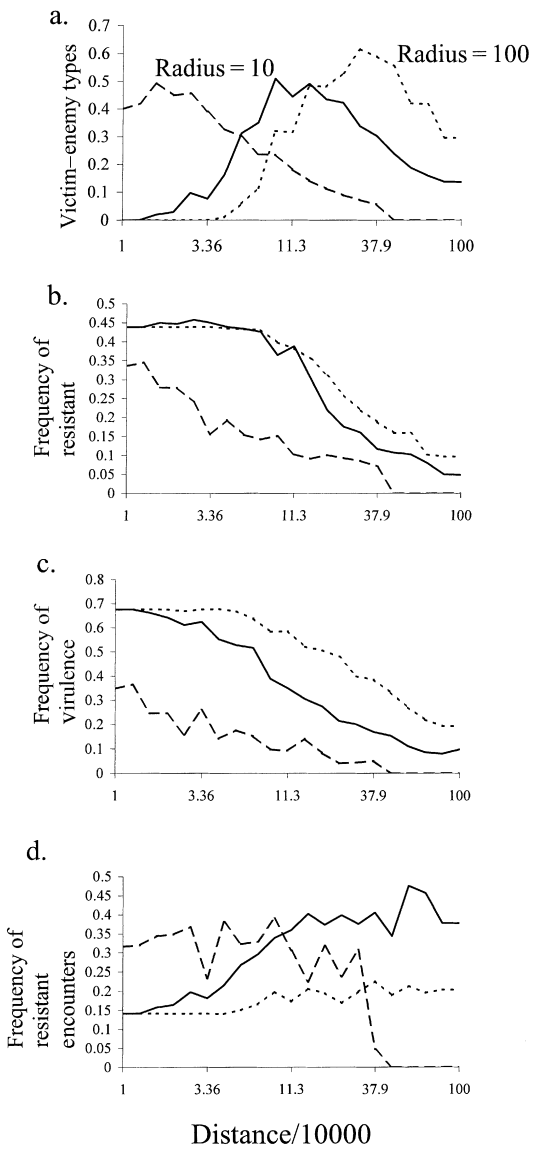

Fig. 3 Effects of island distance for three different island sizes on (a) number of victim–enemy genotypes, (b) frequencies of resistant victims, (c) frequencies of virulent enemies, and (d) frequency of resistant encounters between enemy and victim. Solid lines refer to canonical model (Table 2). Standard errors are not shown. Other conditions as in Fig. 2.

communities to further distances (Fig. 3a). More interestingly, frequencies of resistant victims (Fig. 3b) and virulent enemies (Fig. 3c) increase with island size. A distance-resistance effect is most likely to be observed for islands of intermediate size (Fig. 3d). The reason is that as island size increases (cf. radius 50–100 in the examples of Fig. 3), genotypes establish more rapidly at any given island distance and such islands are more likely to resemble the genotypic communities on the source. As island size decreases (cf. radius 50–10), maximum resistance is shifted to closer islands (Fig. 3d). Resistant interactions on the furthest islands can drop to zero (beyond 400 000 distance units in the example of 10 unit radius in Fig. 3d), but only if the island is sufficiently small such that mutation does not become an important factor in adaptation. In effect, the only two-species interactions which are found on far, small islands almost invariably involve susceptible victims.

Victim mutation has important impacts on population patterns, whereas enemy mutation has minimal effects (Fig. 4). Victim mutation tends to increase the net excess of victim genotypes as compared with enemy genotypes, and this is most apparent at intermediate distances (Fig. 4a). More distant islands show less difference because they are more limited by victim immigration and contain few enemies. Lessening victim mutation results in the attenuation of the relationships highlighted in Fig. 2(c) between distance and the frequency of resistant victims (Fig. 4b) and in Fig. 2(d) between distance and the frequency of resistant encounters (Fig. 4d). In contrast, the relationship involving enemy virulence in Fig. 2(c) is accentuated (Fig. 4c). These first two effects are explained by victim mutation permitting adaptation to the susceptible state (especially on far islands) (Fig. 4b), and the lack of victim mutation resulting in a simple sampling effect of equally migrating enemies and victims from the source (Fig. 4d). The latter effect arises because, at intermediate and far distances, islands tend to contain either susceptible or resistant victims, but not both, meaning that lower frequencies of virulent enemies are selected. Virulent enemies tend to perform better on islands when both victim genotypes are present than do avirulent enemies (see Table 1).

Migration too can have far reaching effects on population patterns (Fig. 5). Looking first at victim migration, we see that increases from its canonical value not only enable victims to colonize islands, but also permit enemies to follow (Fig. 5a). Increasing victim migration also tends to make victims more susceptible and enemies more virulent at intermediate distances (*c* 10^5 distance units in Fig. 5b,c), but has little effect on resistant encounters (Fig. 5d). These results can be explained by increased opportunities for victim adaptation: susceptible victims prevail on islands without enemies and resistant victims tend to be found on islands with (more virulent) enemies. At longer distances (*c* 10^6 distance units in Fig. 5) victims actually become more resistant (Fig. 5b) and have slightly fewer resistant encounters (Fig. 5d), and enemies continue to be more virulent than for the canonical model (Fig. 5c). This is because most far islands have victims only and, because of incessant migration, essentially sample genotypes from the immigration source.

Focusing now on enemy migration, we see that associated net diversity patterns are more sensitive at intermediate distances, whereas victim migration had a

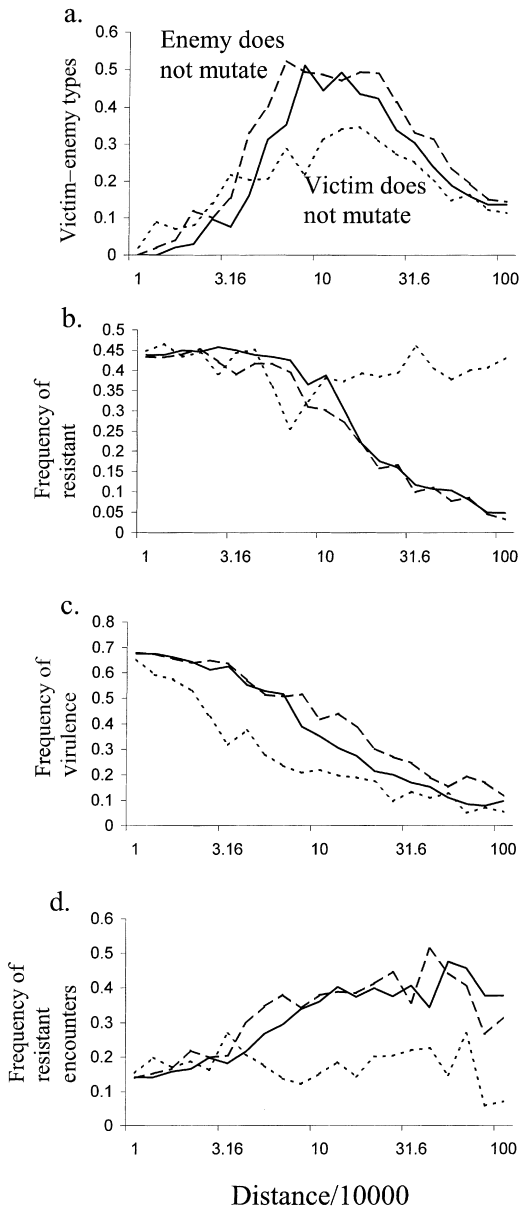

Fig. 4 Effects of island distance and mutation on (a) number of victim–enemy genotypes, (b) frequencies of resistant victims, (c) frequencies of virulent enemies, and (d) frequency of resistant encounters between enemy and victim. Solid lines refer to canonical model (Table 2). Population parameter and other conditions as in Fig. 3.

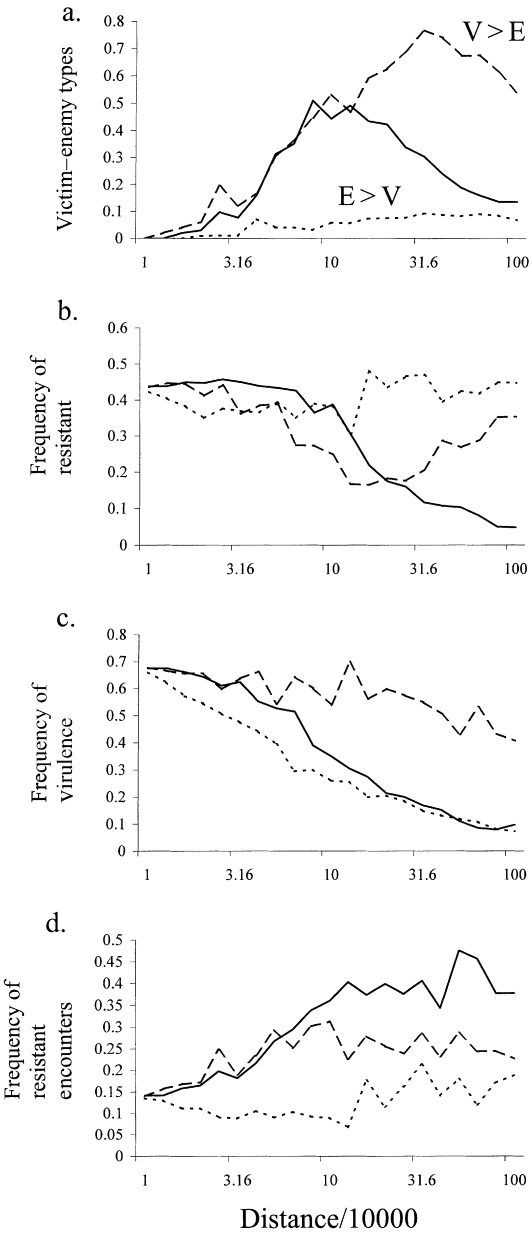

Fig. 5 Effects of island distance and migration rate on (a) number of victim–enemy genotypes, (b) frequencies of resistant victims, (c) frequencies of virulent enemies, and (d) frequency of resistant encounters between enemy and victim. '$V > E$': $I_N = 0.5$, $I_P = 0.05$; '$E > V$': $I_N = 0.05$, $I_P = 0.5$. Solid lines refer to canonical model (Table 2). Population parameters and other conditions are as in Fig. 3.

more considerable effect at far distances (Fig. 5a). At short to intermediate distances, increasing enemy migration decreases the frequencies of victim resistance (Fig. 5b), enemy virulence (Fig. 5c) and resistant encounters (Fig. 5d). The first of these three results may seem counterintuitive, but can be explained by the decreased prevalence of virulent enemies at these sites, which, in turn, tends to select for susceptible victims. At long distances the frequency of resistant victims exceeds the canonical case (Fig. 5b), whereas there is no difference in enemy virulence (Fig. 5c). The former result is a consequence of enemy arrival to these distant islands negating the otherwise tendency towards selection for susceptible victims. Finally, we observe that the distance effect on the

frequency of resistant encounters can be fundamentally altered when enemies migrate faster than victims (Fig. 5d). Resistance initially decreases with insularity and then increases for sufficiently far distances. The decrease is simply an effect of victim genotypes becoming limiting with distance faster than enemy genotypes, whereas the subsequent increase reflects the fact that, at the longest distances, the priority effect of having victims before enemies is stronger than the adaptive advantage to enemies of rapid migration.

The above results concern parameters extrinsic to population growth and decline. To see how demographic parameters affect pattern, we investigated differences in the victim's intrinsic rate of increase r between source and island. In contrast to island distance, size, and mutation and migration rates, r influences equilibrium populations. (This is reflected by the intercepts with the y-axis in Fig. 6, which approximate source conditions.) We found that the effect of differences in r on resistance levels and actual resistant encounters between source and island depend to some extent on whether r does or does not exceed a threshold level (ca. 0.03 for the canonical parameter set in Table 2) on the island. For example, the frequency of resistant victims decreases with insularity as long as r on the island is above a threshold value, whereas it actually increases when islands are of sufficiently lower productivity than are sources (Fig. 6b). In effect, because low r is associated with enemies having more difficulty establishing in more distant (i.e. less diverse and less dense) victim communities, and reduced opportunities for victim evolution (because of the less frequent appearance of mutants), far islands essentially sample victims from the source. This indicates that if one samples close islands, then the relationship between r and the frequency of resistant genotypes will be convex, whereas if one samples distant islands, it will decrease with r. Islands at intermediate distances should show little pattern. In contrast to victim resistance, enemy virulence increases with victim growth regardless of r (Fig. 6c). Finally, although the frequency of resistant encounters was always observed to increase with insularity, the effect of r is nonlinear (Fig. 6d): the highest frequencies of resistant encounters are observed for intermediate levels of victim growth.

Patterns produced by the MA model (Table 1b) as a function of distance are very similar to those produced by the GFG model (Table 1a), with the notable exception of genotypic frequencies which remain more or less constant at 50% regardless of distance for the MA model (not shown). This is because there is no explicit cost or benefit to genotypic identity in the MA model as a function of the number of genotypes present. Note too that in contrast to maximal sampling errors occurring at intermediate distance for the GFG model (Fig. 2c), sampling errors increase asymptotically with distance for the MA model (not shown).

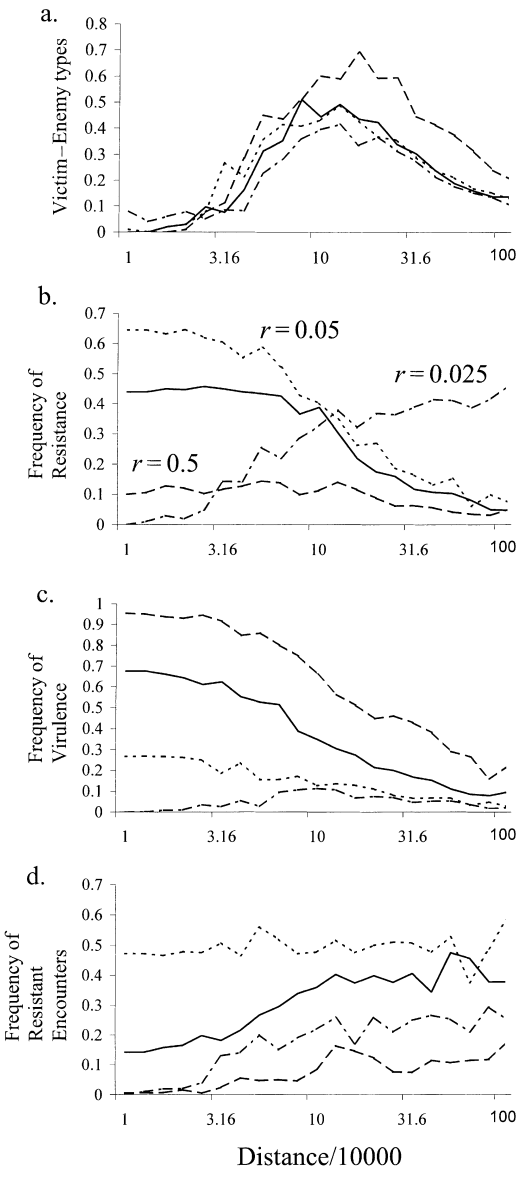

Fig. 6 Effects of island distance and victim intrinsic rate of increase on the island on (a) number of victim–enemy genotypes, (b) frequencies of resistant victims, (c) frequencies of virulent enemies, and (d) frequency of resistant encounters between enemy and victim. Solid lines refer to canonical model (Table 2). Population parameters and other conditions are as in Fig. 3.

Discussion

We have shown how biogeographical variables such as time elapsed since a major disturbance, island size, and distance between an island and its mainland source, as well as species-specific variables such as mutation and migration, can have important impacts on island population biology. Although prompted from empirical data on oceanic islands, our study is more generally relevant

to the immigration of enemies and victims from large self-sustaining habitats to smaller, isolated habitats (Brown, 1984; Lawton, 1993; Harrison & Taylor, 1997). This is but one of several basic types of spatially structured population (Hanski & Gilpin, 1997), and further study is needed to see to what extent our results are applicable to other spatial systems. Below we highlight important findings and present some directions for future research.

The main factor generating pattern in our study is the evolutionary edge the victims have over their specialized enemies via priority effects in colonization and subsequent adaptation. This bias generates several clear predictions. First, assuming a cost to generality in genotypic interactions (as in the GFG model employed here), frequencies of enemy virulence and victim resistance should tend to decrease with increases in island distance from the source and with decreases in island size. In contrast, genotypic frequencies are expected to remain unchanged with insularity in cost-free, matching allele models. Secondly, sample variation in the frequencies of costly genotypes should peak at intermediate levels of insularity, whereas sample variation should increase and asymptote with insularity in cost-free genetic models (i.e. the matching allele model). And thirdly, for an intermediate range of island sizes, the frequency of resistant encounters should increase with distance. Lower victim mutation rates and/or increased enemy migration rates lessen this temporal effect on sympatric adaptation in the two species.

Depending on island size and distance from the source, different biological processes influence the coevolutionary process. Dynamics on small, close islands are dominated by metapopulation processes (extinction–colonization). The high extinction rates on these island types tend to render adaptive patterns highly variable. Despite this variability, we predict a tendency towards the prevalence of susceptible victim and avirulent enemy on these small islands. Consistent with this prediction, Schoener (1987) found that frequencies of pubescent buttonwood (hypothesized to be differentially more resistant to certain types of herbivory than nonpubescent morphs) tend to decrease with decreases in island size. In contrast, coevolution on large, distant islands resembles more of an optimization process in which selection based on species' life-history and demography plays an important role (Figs 3 & 4; Hochberg & van Baalen, 1998). That mutation can be important to adaptation in a macroevolutionary context has been recently demonstrated by Losos & Schluter (2000), who found that within-island speciation of *Anolis* lizards is significantly higher on islands exceeding a threshold size.

Our results can explain the empirical observation of attenuation in resistant victim frequency with increasing insularity (Lack, 1976; Whittaker, 1998), but only when genotypic interactions followed the GFG model with costs to resistance and virulence genotypes, and system productivity on the island was not too low compared with that on the source. Regarding the former requirement, genetic architectures underlying coevolutionary dynamics is a topic of some debate (Frank, 1994; Parker, 1996). Matching allele interactions are often associated with the ability of an individual to recognize self from nonself, whereas more graded interactions with explicit costs or trade-offs (e.g. the GFG model) have been used to represent the subsequent phases of interactions (Godfray 2000). Although our results do not preclude the existence of matching allele interactions, they do indicate that the MA model is neither sufficient to explain attenuation in the frequency of resistant victims, nor necessary to explain trends in the frequency of resistant encounters. We nevertheless urge caution in interpreting the accord between model and observation, as the data concerns macroevolutionary pattern (i.e. ecological dynamics are virtually unknown) and negative results are less likely to be reported.

The latter requirement referred to above – that victim population growth on the island be sufficient compared with that at the source – is a consequence of the convex structure in the relationship between r and the impact of the enemy (controlled in part by parameter a) predicted by our model (Hochberg & van Baalen, 1998; Loreau & de Mazancourt, 1999, see also Abrams, 2000). General trends in population density (a proxy measure for a species' intrinsic rate of increase) as functions of patch size are somewhat equivocal. Studies comparing species on oceanic islands with their mainland sources often find that densities on the former exceed those on the latter, and this is thought to be a consequence of isolation by distance leading to less predation and competition (Grant, 1966; MacArthur *et al*., 1972; Yeaton & Cody, 1974; Emlen, 1979; Wright, 1980). In contrast, population density in continental systems tends to decrease as one goes from large, central patches towards smaller, more peripheral sites. The mechanism here is the higher habitat suitability of large, central patches compared with smaller, more peripheral patches (Lawton, 1993; Connor *et al*., 2000). Taken together, these observations and our results suggest that patterns in adaptation may be very different for mainland–island comparisons and purely continental ones.

In agreement with previous work (Gandon *et al*., 1996; Nuismer *et al*., 2000), we found that migration rates are important for creating adaptive patterns. Gandon *et al*. (1996) examined a metapopulation framework with matching allele interactions between hosts and parasites. Although they did not look for distance effects on sympatric adaptation, they did examine how migration rates affected this quantity, finding that (as long as migration rates were not too pronounced) the faster migrating species was more likely to be adapted in any arbitrary patch (Fig. 1 in Gandon *et al*., 1996). Our results confirm this finding (see our Fig. 5d). Moreover, Gandon and colleagues compared how genotypes of host

or parasite taken from any arbitrary patch were adapted to other patches of known distance apart. They found that the faster migrating species tended to be better adapted sympatrically than allopatrically to its antagonist. Although the main thrust of our study did not concern local adaptation, the frequency of resistant and virulent genotypes on the island is a reflection of this same phenomenon, as (for our GFG model) genotypic frequencies on the source attained at the same, unique four-genotype equilibrium in all simulations. Thus, we predict enemies and their victims sampled from mainland sources should appear to be locally adapted to island populations in phases of recolonization as long as (1) victim mutation rate is sufficient (Fig 4b,c), (2) the migration rate of either species is not too pronounced (Fig. 5b,c), and (3) the growth rate of the victim on the island is sufficient (Fig. 6b,c). With the exception of the latter condition, relaxing these conditions generally only attenuates the pattern, but does not reverse it. Unfortunately, differences in model structures and assumptions preclude direct comparison with the results of Gandon *et al*. (1996).

We only know of a single empirical study that has examined a sufficient variety of population parameters and can be usefully compared with our predictions. Schoener (1987) investigated how the frequency of pubescent buttonwood varied with island size, distance and presence or absence of lizards (potential predators of herbivores). Pubescence in this plant is effective at deterring certain types of herbivore (Schoener, 1987). Consistent with predictions, resistance phenotypes increased with island area, decreased with distance, and increased with the presence of lizards. Our study makes two additional predictions which are both verified by his study (Schoener's Fig. 2): (1) phenotypic variability in buttonwood is reduced for large and for distant islands, and (2) although pubescent plants tend to increase in frequency with island area, they exhibit intermediate frequencies on the largest islands. Although the concordance between theory and data is encouraging, additional study is needed with regard to crucial assumptions of the model (e.g. demonstration that resistant plants have lower fitnesses than susceptible plants when in competition and in the absence of herbivores).

In closing, although our analysis extends previous studies on coevolution in explicitly spatial settings (Gandon *et al*., 1996; Hochberg & van Baalen, 1998; Gomulkiewicz *et al*., 2000; Nuismer *et al*., 2000), several lines of research require future attention.

First, our two-patch approach is surely an oversimplification of many spatial population structures. Models enabling flows within island networks and between these networks and the mainland would be particularly invaluable, insofar as they could reveal scale effects of coevolution. Although an open problem, several studies have recently made headway in comparing how scaling affects metapopulation dynamics in evolutionary systems (Hess, 1996; Hochberg & van Baalen, 1998; Nuismer *et al*., 2000). For example, Nuismer *et al*. (2000) have shown how the relative impacts of gene flow, selection and habitat size combine to determine the scale at which structured coevolution occurs. On the empirical front, Paulay (1994) discusses the importance of the structure of island groups, such as archipelagos, in understanding radiations.

Secondly, we assumed that all island genotypes were extinct at the beginning of the simulation period. This would be a reasonable assumption for lower trophic level interactions (e.g. between plants and their herbivores), where community infrastructure is not necessary for two-species colonization dynamics to occur. In the future, a more community-orientated view of the evolutionary process in space is required. Species on the three lower trophic levels can be quite ecologically diverse (Strong *et al*., 1984; Hawkins *et al*., 1997) and it remains an open question as to how food web assembly on islands influences local adaptations (Holt *et al*., 1999). Moreover, our single extinction assumption is obviously of limited applicability to more complex disturbance regimes in the frequency and amplitude of mortalities (Schoener & Schoener, 1983; Wardle *et al*., 1997; Spiller *et al*., 1998). This too merits further investigation.

Thirdly, our results suggest that large-scale spatial heterogeneity in demography or life-history can have important impacts on adaptation patterns. We only examined the victim's intrinsic growth parameter, r, but numerical studies not presented here suggest that other biological parameters can have important impacts on adaptive patterns (M. E. Hochberg, unpublished data). For some species, demographic and life-history parameters could be a function of island size and distance from the source (T. W. Schoener, personal communication; Paulay, 1994). Given the sometimes nonlinear relationships produced by the canonical model (Fig. 2), and the influence of one of the most basic population parameters on these relationships (Fig. 6), it is our view that it will be a substantial challenge to dissect the finer influences of other components of the island syndrome on antagonistic coevolution.

Fourthly, although modification would be necessary, our modelling approach and the questions posed in our study could be usefully applied to the emerging problem of the biological control or eradication of exotic invaders, be they intentionally or accidentally introduced (for special case of island invasives, see Simberloff, 2001). With respect to our evolutionary approach, especially interesting in this regard is the choice of enemy genotypes from source sites to be introduced into target populations, as our results indicate that this choice can be important to the spatial and temporal trajectories of victim populations.

Finally, we need more investigation of macro-evolutionary pattern. The recent comparative study by Losos & Schluter (2000) makes interesting predictions of how

adaptive radiations may be dependent on island size. Their study supports the view that speciation is more likely to occur when migrants from source populations are isolated by distance, and form large enough populations so that favourable mutations appear and local adaptation within the island occurs (see also Paulay, 1994). MacArthur & Wilson (1967) and Diamond (1977) have emphasized how low diversities of colonists to peripheral oceanic islands facilitate extraordinary local adaptations. Conducting theoretical studies of antagonistic macro-coevolution would require the development of more complex genetic architectures in interacting species (e.g. Frank, 1993; Hochberg, 1997).

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