Chaotic Red Queen coevolution in three-species food chains

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Coevolution between two antagonistic species follows the so-called 'Red Queen dynamics' when reciprocal selection results in an endless series of adaptation by one species and counteradaptation by the other. Red Queen dynamics are 'genetically driven' when selective sweeps involving new beneficial mutations result in perpetual oscillations of the coevolving traits on the slow evolutionary time scale. Mathematical models have shown that a prey and a predator can coevolve along a genetically driven Red Queen cycle. We found that embedding the prey–predator interaction into a three-species food chain that includes a coevolving superpredator often turns the genetically driven Red Queen cycle into chaos. A key condition is that the prey evolves fast enough. Red Queen chaos implies that the direction and strength of selection are intrinsically unpredictable beyond a short evolutionary time, with greatest evolutionary unpredictability in the superpredator. We hypothesize that genetically driven Red Queen chaos could explain why many natural populations are poised at the edge of ecological chaos. Over space, genetically driven chaos is expected to cause the evolutionary divergence of local populations, even under homogenizing environmental fluctuations, and thus to promote genetic diversity among ecological communities over long evolutionary time.

Keywords: adaptive dynamics; arms race; chaos; coevolution; genetic divergence; predator–prey

1. INTRODUCTION

Antagonistic coevolution describes the reciprocal evolutionary interactions between populations belonging to an 'exploiter' (such as a predator or a parasite) and a 'victim' (such as a prey or a host). It is a change in the genetic make-up of a population in response to a genetic change in the antagonistic population (Thompson 1994). Antagonistic interactions have the potential to drive coevolutionary dynamics of adaptive traits: an evolutionary advantage gained by one antagonist is often associated with a disadvantage for the other antagonist, and may therefore prompt a counteradaptation. This may drive stabilizing selection and evolutionary specialization with extreme refinement of the coevolving traits (convergence to an evolutionary equilibrium); or runaway selection and evolutionary escalation with the exaggeration of traits (with the possible extinction of some or all coevolving populations; Matsuda & Abrams 1994; Ferrière 2000); or fluctuating selection and the so-called 'Red Queen dynamics' of perpetual reciprocal changes in the coevolving traits (convergence to a non-equilibrium evolutionary attractor; Van Valen 1973; Stenseth & Maynard Smith 1984; Vermeij 1994). It has been suggested that Red Queen dynamics underlie a large number of important biological processes, some of which are still poorly understood, such as genetic recombination and sexual reproduction (Hamilton *et al.* 1990), the extraordinary diversity of genes related to immune function, resistance and virulence (Salathe *et al.* 2008), and the spatial diversity and local adaptation of exploiter–victim systems (Gandon 2002).

An important dichotomy exists between two main types of Red Queen dynamics (Khibnik & Kondrashov 1997; Ebert 2008; Gaba & Ebert 2009): ecologically driven by negative frequency-dependent selection and genetically driven by beneficial mutations. This distinction is significant because the two types strongly differ in their mechanisms, their underlying genetic architecture, their ecological and evolutionary consequences, and the time scales on which they develop (Ebert 2008). With ecologically driven Red Queen dynamics, extant variants of the exploiter genotype that benefit the most from the numerically dominant victim genotypes are favoured, and, similarly, victim genotypes that best resist the numerically dominant exploiter genotypes are favoured. This pattern results in selection against common exploiter and victim genotypes in a time-lagged negative frequency-dependent fashion (ecological instability). A consequence of this form of fluctuating selection on extant genetic variation is that genetic polymorphism is maintained in the population for long periods (balanced selection) and that allele frequencies can oscillate considerably over time periods of a few generations.

In contrast, genetically driven Red Queen dynamics involve the repeated incidence, spread and fixation of new beneficial mutants in populations that stabilize at

ecological equilibria. Mutants are driven to fixation by directional selection (selective sweeps). Thus, genetic polymorphism is transient only, and the evolutionary dynamics are slow—for two reasons. First, new mutations causing variation in the adaptive traits involved are rare events. Second, a new mutant starts with a very low frequency ($1/N$, where N is the number of wild-type alleles in the population); thus, empirically it can take hundreds of generations until the mutant becomes recognizable (e.g. 1%) at the population level (Elena *et al.* 1996). Therefore, genetically driven Red Queen dynamics develop on an evolutionary time scale that is several orders of magnitude slower than the time scale of ecological processes.

The slow time scale involved hampers the empirical investigation of genetically driven Red Queen dynamics, and mathematical models have been useful to seek conditions that could favour the Red Queen over specialization or escalation. So far, the majority of these models have focused on the two coevolving species and ignored the community context in which coevolution takes place. In this setting, genetically driven Red Queen dynamics develop as regular, predictable cycles in the adaptive trait space. However, pairs of coevolving species are inevitably embedded in community-level interactions of varying degrees of complexity. It is because most species interact with suites of other species that vary dynamically across geographical landscapes that coevolutionary processes can be important in shaping the structure and maintaining variability within specific pairwise interactions, such as predator–prey or host–parasite systems (Abrams 1991, 1996; Strauss *et al.* 2005; Thompson 2005; Thrall *et al.* 2007). For example, some trematode parasites have strong effects on the evolutionary dynamics of their snail hosts, but themselves are dependent upon waterflow for completion of their life cycle (Lively 1999). How the community context of coevolution affects the occurrence and manifestation of genetically driven Red Queen dynamics remains poorly known.

Seminal steps in the theoretical study of coevolutionary dynamics in the community context have been taken recently (Caldarelli *et al.* 1998; Loeuille *et al.* 2002; Gandon 2004; Nuismer & Doebeli 2004; Loeuille & Loreau 2005; Kisdi & Liu 2006; Bell 2007; Ferrière *et al.* 2007; Shoresh *et al.* 2008; Jones *et al.* 2009; Stegen *et al.* 2009), but models of genetically driven coevolutionary dynamics in which more than two species coevolve in a multi-dimensional trait space are still lacking. Here, we extend a simple two-species predator–prey coevolutionary system (Dieckmann *et al.* 1995; where genetically driven Red Queen cycles were first documented) to model coevolution in a three-dimensional trait space among three species forming a food chain. The function of each species in the food chain is determined by a continuous character subject to rare and small genetic mutations. One may expect that the addition of a coevolving species to a coevolving pair could stabilize the evolutionary process at an evolutionary equilibrium, thereby suppressing the Red Queen dynamics (Vermeij 1982; Futuyma 1983), or that the addition could destabilize the periodic evolutionary oscillation and drive the genetically driven Red Queen into chaos (Gavrilets 1997). Here we show that conditions leading to genetically driven periodic cycles in

the two traits of coevolving predator and prey favour chaotic dynamics in the three coevolving traits of the three-species food chain.

2. MODEL CONSTRUCTION

We focus on a single adaptive trait per species that characterizes the function of the species in the food chain. The trait determines competitive ability in the prey, and foraging success in the predator and superpredator. On the evolutionary time scale, de novo trait variation is caused by a rare genetic mutation. The current phenotypes determine the ecological equilibrium of the food chain, and hence the selective pressures acting on variants of the traits. Under the assumption that mutations have very small effects, the long-term coevolutionary process can be modelled as a trait substitution sequence in each species (Metz *et al.* 1992, 1996), the dynamics of which are captured by a set of three deterministic differential equations, one per trait (Dieckmann & Law 1996). When reduced to the classical two-trait, predator–prey coevolutionary model, the system is known to drive trait evolution towards a stable equilibrium or towards a Red Queen cycle (if not towards extinction; Dieckmann *et al.* 1995; Dercole *et al.* 2003, 2006).

As in Dieckmann *et al.* (1995), Lotka–Volterra equations are used to describe the ecological dynamics of the food chain:

$$\frac{\mathrm{d}n_1}{\mathrm{d}t} = n_1(r - cn_1 - a_2n_2), \tag{2.1a}$$

$$\frac{\mathrm{d}n_2}{\mathrm{d}t} = n_2(e_2a_2n_1 - d_2 - a_3n_3) \tag{2.1b}$$

$$\text{and} \quad \frac{\mathrm{d}n_3}{\mathrm{d}t} = n_3(e_3a_3n_2 - d_3), \tag{2.1c}$$

where n_1, n_2 and n_3 are prey, predator and superpredator densities, respectively; r and c are prey intrinsic *per capita* growth rate and sensitivity to intraspecific competition, respectively; and a_i, e_i and d_i are the attack rate, efficiency and intrinsic death rate in the predator ($i = 2$) and superpredator ($i = 3$), respectively. Each species is characterized by one genetic trait x_i ($i = 1$–3). The genetic system is one-locus haploid; the genetic traits can influence the prey competition function c and the attack rates a_2 and a_3, and trait dependencies are modelled using the following functional forms:

$$c = c_0 + c_2(x_1 - c_1)^2, \tag{2.2a}$$

$$a_2 = \exp\left(-\left(\frac{x_1 - a_{24}}{a_{21}}\right)^2 + 2a_{23}\frac{x_1 - a_{24}}{a_{21}}\frac{x_2 - a_{25}}{a_{22}}\right.$$
$$\left. - \left(\frac{x_2 - a_{25}}{a_{22}}\right)^2\right) \tag{2.2b}$$

$$\text{and} \quad a_3 = \exp\left(-\left(\frac{x_2 - a_{34}}{a_{31}}\right)^2 + 2a_{33}\frac{x_2 - a_{34}}{a_{31}}\frac{x_3 - a_{35}}{a_{32}}\right.$$
$$\left. - \left(\frac{x_3 - a_{35}}{a_{32}}\right)^2\right) \tag{2.2c}$$

(with $0 < a_{23}$, $a_{33} < 1$ and c_0, c_2, a_{21}, a_{22}, a_{31}, a_{32} all positive). Prey competition is minimum at $x_1 = c_1$, where prey are best adapted to their environment, while the attack rates a_2 and a_3 are bidimensional Gaussian functions with elliptic contour lines centred at (a_{24}, a_{25})

and (a_{34}, a_{35}), respectively, and controlled in amplitude and orientation by parameters a_{21}–a_{23} and a_{31}–a_{33}, respectively. Differences $(x_1 - a_{24})$ and $(x_2 - a_{25})$ (respectively, $(x_2 - a_{34})$ and $(x_3 - a_{35})$) measure the degree to which the predator (superpredator) 'matches' the prey (predator); that is, the attack rate is maximum when $x_1 = a_{24}$ and $x_2 = a_{25}$ (respectively, $x_2 = a_{34}$ and $x_3 = a_{35}$), while parameters a_{21}–a_{23} (respectively, a_{31}–a_{33}) control the sensitivity of the attack rate to the mismatch.

When a mutation occurs in trait x_1 and generates a new value x'_1, the ecological system becomes

$$\frac{dn_1}{dt} = n_1\big(r - c(x_1)n_1 - c(x_1)n'_1 - a_2(x_1, x_2)n_2\big), \quad (2.3a)$$

$$\frac{dn'_1}{dt} = n'_1(r - c(x'_1)n_1 - c(x'_1)n'_1 - a_2(x'_1, x_2)n_2), \quad (2.3b)$$

$$\frac{dn_2}{dt} = n_2\big(e_2a_2(x_1, x_2)n_1 + e_2a_2(x'_1, x_2)n'_1 \\ - d_2 - a_3(x_2, x_3)n_3\big) \quad (2.3c)$$

and $\quad \dfrac{dn_3}{dt} = n_3(e_3a_3(x_2, x_3)n_2 - d_3), \quad (2.3d)$

so that the fitness function of mutant x'_1 is given by

$$f_1(x_1, x_2, x_3, x'_1) = \frac{1}{n'_1}\frac{dn'_1}{dt}\bigg|_{\substack{n=\bar{n} \\ n'_1=0}} \\ = r - c(x'_1)\bar{n}_1(x_1, x_2, x_3) \\ - a_2(x'_1, x_2)\bar{n}_2(x_1, x_2, x_3), \quad (2.4)$$

where $n = (n_1, n_2, n_3)$ and $\bar{n}$ denotes the ecological equilibrium of model (2.1) at which the food chain stabilizes in the absence of mutants (§3).

Similar equations can be written when a mutation arises in the predator (trait x_2) or superpredator (trait x_3; see appendix S1 in the electronic supplementary material) and yields the fitness functions of mutants x'_2 and x'_3:

$$f_2(x_1, x_2, x_3, x'_2) = \frac{1}{n'_2}\frac{dn'_2}{dt}\bigg|_{\substack{n=\bar{n} \\ n'_2=0}} \\ = e_2a_2(x_1, x'_2)\bar{n}_1(x_1, x_2, x_3) - d_2 \\ - a_3(x'_2, x_3)\bar{n}_3(x_1, x_2, x_3) \quad (2.5)$$

and

$$f_3(x_1, x_2, x_3, x'_3) = \frac{1}{n'_3}\frac{dn'_3}{dt}\bigg|_{\substack{n=\bar{n} \\ n'_3=0}} \\ = e_3a_3(x_2, x'_3)\bar{n}_2(x_1, x_2, x_3) - d_3. \quad (2.6)$$

The long-term coevolution of traits x_1, x_2 and x_3 on the evolutionary time scale obeys the so-called canonical equation of adaptive dynamics (Dieckmann & Law 1996); that is, the three-dimensional system of ODEs,

$$\frac{dx_1}{dt} = \frac{1}{2}\mu_1\sigma_1^2\,\bar{n}_1\frac{\partial f_1}{\partial x'_1}\bigg|_{x'_1=x_1}, \quad (2.7a)$$

$$\frac{dx_2}{dt} = \frac{1}{2}\mu_2\sigma_2^2\,\bar{n}_2\frac{\partial f_2}{\partial x'_2}\bigg|_{x'_2=x_2} \quad (2.7b)$$

and $\quad \dfrac{dx_3}{dt} = \dfrac{1}{2}\mu_3\sigma_3^2\,\bar{n}_3\dfrac{\partial f_3}{\partial x'_3}\bigg|_{x'_3=x_3} \quad (2.7c)$

The right-hand sides are the product of mutation rates (μ_i, $i = 1$–3), mutational steps variances (σ_i^2), equilibrium densities ($\bar{n}_i$) and selection gradients (fitness derivatives). The latter explicit expressions are cumbersome and were always generated and handled by means of symbolic computation.

3. MODEL ANALYSIS AND RESULTS
The ecological model (2.1) has a unique non-trivial equilibrium,

$$\bar{n}_1 = \frac{r}{c} - \frac{a_2 d_3}{ce_3a_3}, \quad (3.1a)$$

$$\bar{n}_2 = \frac{d_3}{e_3a_3} \quad (3.1b)$$

and $\quad \bar{n}_3 = \dfrac{e_2a_2}{a_3}\left(\dfrac{r}{c} - \dfrac{a_2 d_3}{ce_3a_3}\right) - \dfrac{d_2}{a_3}, \quad (3.1c)$

which is positive if and only if $\bar{n}_3 > 0$. When positive, the equilibrium $\bar{n}$ is globally stable (in the positive orthant). Thus, the ecological model (2.1) is only viable within the region of the trait space defined by the condition $\bar{n}_3 > 0$.

If the superpredator and the predator are able to simultaneously match the predator and the prey, respectively (i.e. $a_{25} = a_{34}$), and if, at the same time, the prey is able to minimize its sensitivity to intraspecific competition (i.e. $c_1 = a_{24}$), then $\bar{x}_1 = c_1$, $\bar{x}_2 = a_{25}$, $\bar{x}_3 = a_{35}$ is an equilibrium of the evolutionary model (2.7). Starting from these conditions, and fixing parameters at values corresponding to evolutionary cycles in the ditrophic model (Dieckmann *et al.* 1995), we performed the numerical continuation of the equilibrium $\bar{x}$ with respect to several parameters.

As expected, evolutionary stability was sensitive to the mutation rate μ_1 of the prey. As μ_1 increases, the evolutionary equilibrium loses stability through a supercritical Hopf bifurcation that yields a small-amplitude stable evolutionary cycle (see appendix S3 in the electronic supplementary material). Starting from the Hopf bifurcation, we numerically continued the cycle, while monitoring its stability through the computation of the associated Floquet multipliers (i.e. the three eigenvalues of the linearized Poincaré map associated with the cycle; one of them is structurally equal to 1, and therefore its estimated value is a measure of computation accuracy; the other two determine the stability of the cycle). Again, by increasing μ_1, evolutionary stability was easily lost through a series of period-doubling bifurcations (a negative Floquet multiplier passing through -1; see appendix S3 in the electronic supplementary material). At each period-doubling bifurcation, the cycle becomes unstable, and a new stable cycle (which traces twice the bifurcating cycle) appears. Switching to the continuation of the new stable cycle allowed us to find the next period-doubling bifurcation. Because the sequence of bifurcation parameter values μ_1^i, $i = 1, 2, \ldots$, accumulates geometrically at the frontier μ_1^∞ of the chaotic region of the Feigenbaum period-doubling cascade, only a limited number of bifurcations in the sequence could be detected (μ_1^i, $i = 1, 2, 3$, are reported in figure 1). The robustness of the cascade has been checked through the continuation of the period-doubling bifurcations with respect to various pairs of parameters (details will be published elsewhere).
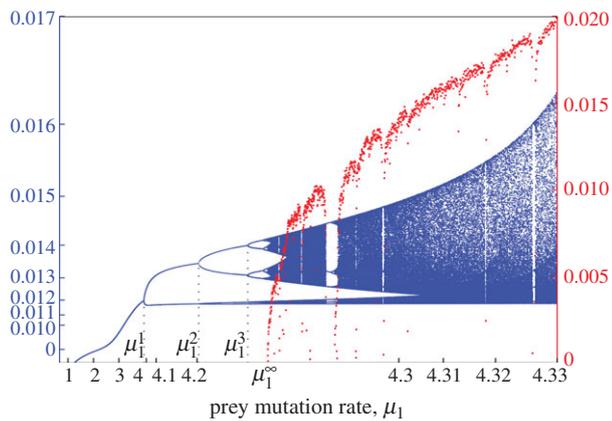
Figure 1. Period-doubling route towards genetically driven Red Queen chaos in a three-species food chain. Peak values of the superpredator trait, x_3 (blue), in the corresponding evolutionary attractor and the largest Lyapunov exponent, L_1 (red), as functions of the prey mutation rate, μ_1. The value μ_1^∞ indicates the lower limit of the chaotic range. Parameter values: $\mu_2 = 1$, $\mu_3 = 1$, $\sigma_1^2 = 0.3$, $\sigma_2^2 = 2$, $\sigma_3^2 = 2$, $r = 0.5$, $d_2 = 0.05$, $d_3 = 0.02$, $e_2 = 0.14$, $e_3 = 0.14$, $a_{21} = 0.22$, $a_{22} = 0.25$, $a_{23} = 0.6$, $a_{24} = 0$, $a_{25} = 0.04$, $a_{31} = 0.22$, $a_{32} = 0.25$, $a_{33} = 0.6$, $a_{34} = 0$, $a_{35} = -0.04$, $c_0 = 0.5$, $c_1 = 0$, $c_2 = 3$.

In order to estimate μ_1^∞, we computed the full spectrum of the attractor's Lyapunov exponents $L_1 \geq L_2 \geq L_3$ for finely incremented values of μ_1 (step 10^{-5}; see appendix S2 in the electronic supplementary material). $L_1 > 0$ implies that μ_1 is in the chaotic region, whereas $L_1 = 0$ in periodic windows (figure 1); in the chaotic region, L_2 is structurally equal to 0 (its estimated value measures computation accuracy), while L_3 is negative. The attractor's fractal dimension then follows from the Kaplan–Yorke formula (figure 2). In this example, the dominant Lyapunov exponent equals $+0.0081321$ and the fractal dimension of the attractor is 2.0176 (the attractor lies roughly on a two-dimensional Möbius strip). Typically, the prey and predator characters oscillate with small irregular fluctuations in amplitude and frequency, while variation in the amplitude of the oscillations in the superpredator trait is more pronounced.

Our analysis shows that the genetically driven Red Queen turns chaotic under conditions similar to those leading to genetically driven Red Queen cycles, provided that the mutation time scale of the prey is short enough compared with the mutation time scales of the predator and the superpredator. That is (Dieckmann *et al.* 1995; Dercole *et al.* 2003), the predator efficiency should be great enough to drive the prey away from its genetic optimum; and there should be sufficient need for the predator to track the prey's character change. As the prey departs from its optimum, its population density drops, which causes a reversal of selection on the predator's trait, followed by a reversal of selection on the prey's character. If the prey evolves fast enough, it will not be 'caught up' by the predator and permanent trait oscillations will evolve; the system ends up in chaos because the predator is also engaged in a coevolutionary chase with the superpredator. Broad comparative analyses (e.g. Martin & Palumbi 1993) have established a strong relationship between nucleotide substitution rate and body size. For

instance, rates of nuclear and mtDNA evolution are slow in whales, intermediate in primates and fast in rodents, and a similar effect of body size also exists in poikilothermic vertebrates. Thus, trophic chains with smaller prey (and hence faster mutagenesis) may be more prone to coevolutionary chaos.

4. DISCUSSION

Even though quantitative data on long-term predator–prey coevolutionary dynamics remain elusive (Barnosky 2001), the fossil record supports the view that predation is an important driver of evolutionary change (Kelley *et al.* 2003). Moreover, palaeontological and phylogenetic analyses gather increasing evidence for the role of three-level chain interactions in coevolution (Currie *et al.* 2003; Kelley *et al.* 2003). These empirical findings have been paralleled by extensions of coevolutionary theory beyond pairwise interactions (Abrams 1996; Caldarelli *et al.* 1998; Loeuille *et al.* 2002; Gandon 2004; Nuismer & Doebeli 2004; Loeuille & Loreau 2005; Kisdi & Liu 2006; Bell 2007; Ferrière *et al.* 2007; Shoresh *et al.* 2008; Jones *et al.* 2009; Stegen *et al.* 2009), but so far the complexity of evolutionary dynamics among more than two species coevolving in a multi-dimensional trait space has been little explored. As a step forward in that direction, we added a superpredator, as a third coevolving species, to coevolution between a prey and a predator.

Prey–predator–superpredator trophic chains have long attracted the attention of ecologists as they occur by diverse mechanisms, can cross ecosystem boundaries and have practical importance; for example, in the management of fisheries or biological control of crop pests (Cohen *et al.* 2009). Our model descends from the lineage of two-species models that addressed genetically driven predator–prey coevolution (Stenseth & Maynard Smith 1984; Rosenzweig *et al.* 1987; Rand & Wilson 1991; Marrow *et al.* 1992; Dieckmann *et al.* 1995; Doebeli 1997; Gavrilets 1997; Khibnik & Kondrashov 1997; Dercole *et al.* 2003, 2006) and specifically extends the analysis of Dieckmann *et al.* (1995), where stable cycles in adaptive dynamics were first documented.

We searched for strange attractors in the three-trait, three-species coevolutionary model by weaving intuition and theory. Theory was telling us that in third-order dynamical systems the most common route to chaos is the Feigenbaum period-doubling cascade (see appendix S3 in the electronic supplementary material), and we knew that evolutionary stability in predator–prey models was especially sensitive to the mutation rate of the prey (Dieckmann *et al.* 1995; Dercole *et al.* 2003). Thus, our analysis of the tritrophic evolutionary dynamics was organized by looking for parameters that caused evolutionary cycles in the lower ditrophic model (and such that increasing the prey mutation rate could trigger doubling of the cycle period) and then tracking the period-doubling cascade. The strategy was successful at detecting transitions towards evolutionary chaos in the three-species system.

Our analysis of three-species coevolution was intended as an extension of Dieckmann *et al.*'s (1995) two-species model. This is the technical motivation for our choice of the type I functional response to describe trophic interactions, hence the Lotka–Volterra structure of the
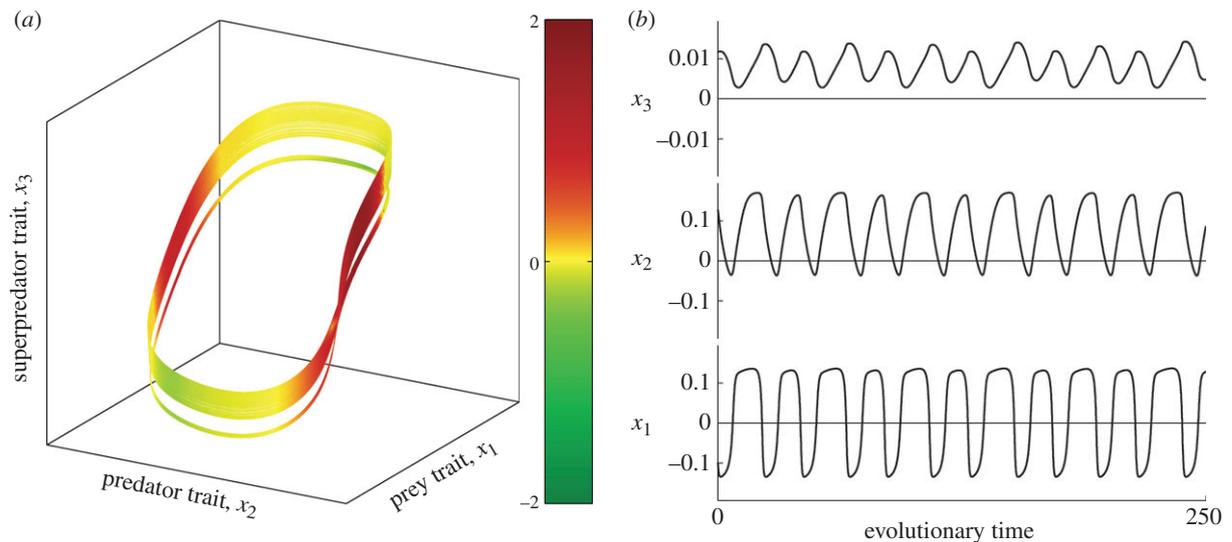
Figure 2. Genetically driven chaotic Red Queen in a three-species food chain. (*a*) Evolutionary strange attractor. The estimated Lyapunov exponents are $L_1 = 8.1321 \times 10^{-3}$, $L_2 = -2.3923 \times 10^{-6}$, $L_3 = -4.6270 \times 10^{-1}$, and the fractal dimension is $2 - L_1/L_3 = 2.0176$ (Kaplan–Yorke formula). Colour codes the largest local Lyapunov exponent (see appendix S2 in the electronic supplementary material). (*b*) Chaotic time series of prey, predator and superpredator traits. Parameter values as in figure 1 and $\mu_1 = 4.2667$.

ecological model. This has the important consequence of ensuring that the food chain always stabilizes at an equilibrium on the ecological time scale. Therefore, oscillations predicted by the evolutionary model could only be due to nonlinear interactions between selective pressures acting on genetic variation in the adaptive traits—not to trait variation induced by instabilities in the ecological dynamics (Abrams & Matsuda 1997*a*). More realistic food chain models with, for example, saturating (type II) functional responses or self-limitation at higher trophic levels can also stabilize at ecological equilibria, though ecological cycles and ecological chaos are also expected in viable regions of the trait space. This opens the possibility of Red Queen chaotic dynamics that would be 'ecogenetically driven' (*sensu* Khibnik & Kondrashov 1997; for the two-species case see Dercole *et al.* 2003; Dercole *et al.* 2006).

Another fundamental feature of the model is the definition of the adaptive traits. We keep the 'matching model' used in Dieckmann *et al.* (1995), which has long been popular in the theory of predator–prey coevolution (Cohen *et al.* 1993; Abrams 2000; Loeuille & Loreau 2005; Stegen *et al.* 2009). The matching model assumes that the traits of a species and its prey jointly determine the attack (and capture) rate on the former by the latter, and that the attack rate is maximized when the two traits match. Scaled body size is a commonly used surrogate measure for such traits (Williams & Martinez 2000). Defining the adaptive traits according to the matching model is known to promote genetically driven Red Queen cycles in the two-species predator–prey coevolutionary model (Marrow *et al.* 1992, 1996; Dieckmann *et al.* 1995; Abrams & Matsuda 1997*b*; Doebeli 1997; Gavrilets 1997), and thus provided us with the appropriate framework to answer our main question—how are two-species Red Queen cycles affected by the coevolution of a third species?

Several well-studied antagonistic pairwise interactions seem to conform to the matching model. This includes

parasitic cuckoos and their hosts, in which the probability that a parasitic egg be rejected depends on the similarity of host and parasite egg morphologies (Robert & Sorci 1999); crossbills and lodgepole pines, for which fitnesses are influenced by matching between bill size and cone structure (Benkman 1999); feather lice and dove hosts, in which louse fitness at least is influenced by matching size with host size (and host size correlates with parasite size across species; Clayton *et al.* 2003). Other equally well-studied systems, however, better fit an alternative model in which the strength of between-species interactions is a monotonic function of the difference between the predator and prey's traits. This is the case of parsnip web-worms and wild parsnips, in which feeding efficiency of defended plants increases with higher production of detoxifying enzymes (Berenbaum & Zangerl 1992). Likewise, the rate of successful attack in the Japanese-camellia–camellia-weevil system is a monotonic function of the difference between camelia fruit wall thickness and weevil mouthpart size (Toju & Sota 2006, 2009). The 'difference model' so defined also fits the trophic interaction between toxic newts as prey and potentially toxin-resistant garter snakes as predators (Brodie *et al.* 2002; Hanifin *et al.* 2008).

Nuismer *et al.*'s (2007) theoretical analysis of antagonistic coevolution under the difference model of attack rate shows that coevolutionary cycles are still possible with this model, provided that selection is strong enough and stabilizing selection acts on the traits. Thus, genetically driven coevolutionary cycles in pairwise antagonistic interactions appear to be at least possible under relatively broad conditions when the attack rate is described by the difference model. The question of whether coevolutionary cycles turn into chaos in the three-species food chain is open to investigation. Future models should also examine the coevolution of alternative or additional traits besides the attack rate. Dercole *et al.* (2003) and Kisdi & Liu (2006), for example, considered the evolution of handling time, a key factor of the functional response. As an

extension of our model, it would be interesting to account for genetic variation in predator and superpredator handling times, track the evolution of the functional responses themselves as a byproduct and monitor the potential bifurcations experienced by the coevolutionary dynamics as a consequence.

The possibility that natural selection acting on extant genetic variation drives community dynamics into chaos has been known since early analyses of host–pathogen models (May & Anderson 1983), and is not unexpected given that competition between multiple species or genotypes can easily destabilize population dynamics (Hofbauer & Sigmund 1998; Turchin 2003). This type of chaotic evolutionary dynamics has been found in theoretical studies of genetic polymorphisms under frequency-dependent selection (e.g. May & Anderson 1983; Seger 1992; Ferrière & Fox 1995; Solé & Sardanyés 2007), strategy frequencies in evolutionary games (Nowak & Sigmund 2003) and rapid evolution of a continuous trait in interaction with population dynamics (Abrams & Matsuda 1997a). All these are instances of evolutionary chaos on the ecological time scale. The system considered here is different since the time scales of ecology and evolution are separated: the population dynamics of different alleles stabilize on a monomorphic state over a time scale that is fast compared with the slow evolutionary time scale over which the dynamics of the adaptive traits develop. Thus, our analysis uncovers the first example of genetically driven chaotic Red Queen.

The genetically driven chaotic Red Queen implies that nonlinear interactions of selective pressures can drive phenotypic changes that are unpredictable over the slow time scale of long-term evolution, even in a perfectly constant abiotic environment. (Note that with chaos in allele or strategy frequencies driven by negative frequency dependence, there is unpredictability in the dynamics of frequencies, but the identity of alleles or strategies never changes.) This has implications for our understanding of the role of 'chance' in evolution (Travisano *et al.* 1995; Beatty 2006). Chance manifests itself when the evolutionary trajectories of adaptive traits diverge between replicated populations that were initiated in similar phenotypic and genotypic states. Experimental tests on bacterial systems have provided some of the best evaluations of the role that chance may play in evolution. Although founded by the same clone, and evolving in identical conditions, replicate populations often diverge from one another in their relative growth rate, demographic traits, morphological features and performance in other environments (Elena & Lenski 2003 and references therein). The conventional explanation for evolutionary divergence 'by chance' involves genetic stochasticity (the randomness of mutation and drift owing to demographic stochasticity) and environmental stochasticity (random changes in environmental conditions; Lenormand *et al.* 2008). However, models of adaptive trait dynamics derived from individual-level 'first principles' have shown that the effect of genetic stochasticity is often 'smoothed out' in the long term, with traits converging towards the attractor of a deterministic dynamical system, provided that there is some minimal separation between the time scales of mutation and selection (Champagnat *et al.* 2006). The present study shows that even if the randomness of genetic stochasticity is

smoothed out, uncertainty can arise from the selection component of the evolutionary process: adaptive trait trajectories converge towards a deterministic attractor, yet the chaotic nature of the attractor renders the trait dynamics unpredictable beyond a short evolutionary time horizon. Thus, the nonlinearity of the selection gradient offers an alternative to genetic or environmental stochasticity to explain the chance component of evolutionary trajectories in real populations.

Further examples of genetically driven chaotic Red Queen dynamics are likely to be discovered in models of long-term evolution in which the adaptive process operates in a three- (or more) dimensional trait space—even if all traits (e.g. behavioural or life-history traits) pertain to the same single species. Genetically driven chaos might also arise in two-trait adaptive dynamics models, or even in one-trait systems showing ecological multistability (Dercole *et al.* 2002), that are subject to externally driven periodic fluctuations in mutation or selection. Besides its conceptual value, the genetically driven chaotic Red Queen suggests three new hypotheses (discussed below) about coevolutionary dynamics. Each hypothesis opens an avenue for future theoretical work.

(a) The intrinsic unpredictability of coevolutionary dynamics is widespread

In view of the general theory of dynamical systems, the existence of chaotic evolutionary attractors over some parameter region can affect the coevolutionary dynamics broadly outside that region. Even when the coevolutionary attractor of the food chain is an equilibrium or a cycle, the 'shadow' of evolutionary chaos will be seen in the form of long erratic transients (Hastings 2004). Genetic noise—owing, for example, to random drift or stochastic gene flow—or stochastic environmental fluctuations on the slow evolutionary time scale may actually maintain these transients for arbitrarily long evolutionary times. Such 'noise-induced chaos' illustrates the general fact that small amounts of exogenous noise can have disproportionate qualitative impacts on the long-term dynamics of a nonlinear system in which chaotic structures exist for some parameter values (Rand & Wilson 1991; Lai *et al.* 2003; Ellner & Turchin 2005).

(b) Coevolution can drive population dynamics to the edge of chaos

Looking at evolution on a slow time scale, in contrast with, or even completely separated from, the fast time scale of ecology, does not mean that the coevolutionary process has no effect on the ecological state of the system. In fact, the genetically driven chaotic Red Queen implies that the population size of each species also fluctuates chaotically, but these fluctuations develop on the slow, evolutionary time scale, because at each point in evolutionary time, the food chain model analysed here is at ecological equilibrium. In other food chain models, ecological cycles and chaos occur readily (Hastings & Powell 1991; Gross *et al.* 2005). In the light of this and other studies (Khibnik & Kondrashov 1997; Dercole *et al.* 2006), the trait domain corresponding to ecological chaos may contain part or all of the coevolutionary attractor (ecogenetically driven Red Queen). A sharp change in the selective regime at the

boundary between chaotic and non-chaotic ecological dynamics is expected in general (Ferrière & Gatto 1995; Dercole *et al.* 2006), and may poise the food chain near that boundary for long evolutionary times, in a process called 'evolutionary sliding' (Dercole *et al.* 2006). This would provide an evolutionary explanation for the standing puzzle that the abundance of many natural populations seemingly fluctuates 'at the edge of chaos' (Ellner & Turchin 1995; Turchin 2003).

(c) *The chaotic Red Queen promotes genetic divergence in metacommunities*

There is considerable interest in better understanding how coevolutionary processes work in geographically structured habitats (Thompson 2005). The arising of genetically driven chaos has direct implications for the origin and maintenance of genetic diversity in spatially extended communities. Let us consider the metaphor of a fragmented landscape in which all patches are identical and isolated. Genetically driven chaotic Red Queen dynamics imply that each local trophic chain evolves along the same strange attractor, but small ancestral differences in the genetic make-up of local communities will result in permanent genetic differences between patches. The magnitude of these differences will vary over time and sometimes be as large as the coevolutionary attractor. In contrast, small ancestral differences remain small in the case of periodic Red Queen dynamics (and the same would be true if the Red Queen were ecologically driven). In other words, local genetically driven coevolutionary chaos promotes spatial genetic divergence, even in the absence of environmental differences between patches. Red Queen dynamics in general can explain phenotypic mismatches between coevolving species even in the absence of spatial structure, gene flow or genetic drift (Berenbaum *et al.* 1986; Hanifin *et al.* 2008); the chaotic Red Queen, in particular, predicts the persistence of different degrees of mismatches between local communities, even if environmental conditions are spatially uniform.

Furthermore, general results on the synchronization of dynamical systems subject to common fluctuating exogenous forces warn that the genetic divergence between local populations can be lost in the presence of long-term environmental fluctuations (this is known in ecology as the Moran effect; see Royama 1992 for a review). However, recent results (Colombo *et al.* 2008) show in great generality that this is possible only if environmental fluctuations are large and tuned specifically to the endogenous dynamics of the system. Genetically driven coevolutionary chaos could therefore play an important role in promoting genetic diversity in ecological communities threatened by environmental homogenization (Olden *et al.* 2004). We conclude that genetically driven Red Queen chaos might explain genetic differentiation of local communities without invoking local adaptation to different habitat conditions or to multiple steady states of local populations in the metacommunity. This points to the possibility that, in sexual species, the genetic divergence of local populations induced by complex adaptive dynamics might favour the evolution of reproductive isolation and hence parapatric speciation—even across relatively uniform habitats, as in

marine species (Palumbi 1994). Extension of speciation models along ecological gradients (Doebeli & Dieckmann 2003) will help examine this hypothesis further.

5. CONCLUDING REMARKS

Here, we have extended Dieckmann *et al.*'s (1995) model of predator–prey genetically driven coevolution by adding a coevolving superpredator to the system. When Red Queen periodic cycles develop in the two-species model, the adaptive dynamics of the three coevolving species are often chaotic. A general condition for this to happen is that the evolutionary rate of the prey be large enough. The greatest irregularity is then predicted in the dynamics of the superpredator trait. Because the ecological model of the food chain is always at equilibrium throughout the trait space, instability in the ecological dynamics plays no role in generating this chaotic Red Queen, which is thus entirely driven by nonlinear interactions between the selective pressures acting on rare genetic variation of the traits.

The specificities of the model and the new hypotheses arising from the results call for continued theoretical investigation of chaotic dynamics in genetically driven coevolutionary processes. This theoretical endeavour should be paralleled by an empirical effort focusing on the patterns of temporal unpredictability and spatial heterogeneity of antagonistic coevolution and the consequences for population dynamics, genetic differentiation in metacommunities and macroevolutionary processes, including speciation.

A key difference between coevolutionary cycles and coevolutionary chaos lies in the expectation that geographically distinct communities subject to homogenizing factors of their environment (e.g. large-scale climatic fluctuations) should exhibit similar degrees of phenotype mismatching when coevolving cyclically, and persistently dissimilar degrees of mismatching when coevolving chaotically. Spatially heterogeneous mismatches have been documented recently in the camellia–weevil (Toju 2009) and newt–garter snake (Hanifin *et al.* 2008) systems. In the light of our results, the fine-scale divergence of coevolution in the former may not require geographical variation of environmental factors (Toju 2009). Molecular data supporting the role of beneficial mutations, rather than standing genetic variation, as fuelling coevolution between newts and their snake predators (Feldman *et al.* 2009) offer promising evidence for the relevance of genetically driven Red Queen models to deepen our understanding of geographical patterns of coevolution in nature.

Besides trophic interactions, the Red Queen is expected to reign in many exploiter–victim systems (Lythgoe & Read 1998). Biomedical science has already revealed the potential ubiquity of the Red Queen in parasitic and pathogenic interactions (Moya *et al.* 2004). Experimental coevolution in host–pathogen systems is being used successfully to evidence the patterns and dissect the processes of ecologically driven Red Queen dynamics in laboratory systems (Koskella & Lively 2009) and in nature (Decaestecker *et al.* 2007). On the evolutionary time scale, antagonistic coevolutionary dynamics fuelled by de novo genetic variation have been studied experimentally using bacterial systems

(Lenski & Levin 1985; Bohannan & Lenski 2000; Buckling & Rainey 2002; Gallet *et al.* 2009). The time-shift experimental design (Gaba & Ebert 2009) implemented to study ecologically driven Red Queen dynamics could be applied to measure how predictable genetically driven coevolutionary trajectories are under different experimental treatments, and thus to search for the essential property of chaotic dynamics—exponentially declining predictability of trajectories. Combining experiments with sufficiently detailed mathematical models of the study systems will be instrumental to identify relevant experimental treatments, to design data collection and analysis and to interpret the results (Decaestecker *et al.* 2007). If it were supported by such experiments on microbial systems, the genetically driven chaotic Red Queen might contribute to our understanding of the rapid and indeterminate evolution of viral pathogens (Kirkwood & Bangham 1994; Moya *et al.* 2004), and perhaps influence the study and control of emergent pathogens on large temporal and spatial scales.

Ultimately, the important question raised by the genetically driven chaotic Red Queen is unlikely to be whether or not long-term evolution in any specific ecological system is chaotic—a question that makes sense only in the realm of mathematical models. Population ecologists have long gone beyond that question—chaos versus non-chaos—to draw stunning insights from the nonlinear dynamics theory into how environmental forces and internal dynamics shape species abundance and distribution in nature (Allen *et al.* 1993; Ellner & Turchin 1995; Dixon *et al.* 1999; Turchin 2003). The same move could take place in evolutionary biology, as genetically driven Red Queen chaos challenges our ability to measure, compare and interpret coevolutionary patterns and processes in the real world.

REFERENCES

Abrams, P. A. 1991 The effects of interacting species on predator–prey coevolution. *Theor. Popul. Biol.* **39**, 241–262. (doi:10.1016/0040-5809(91)90022-8)

Abrams, P. A. 1996 Evolution and the consequences of species introduction and deletions. *Ecology* **77**, 1321–1328. (doi:10.2307/2265529)

Abrams, P. A. 2000 The evolution of predator–prey interactions: theory and evidence. *Annu. Rev. Ecol. Syst.* **31**, 79–105. (doi:10.1146/annurev.ecolsys.31.1.79)

Abrams, P. A. & Matsuda, H. 1997a Prey evolution as a cause of predator–prey cycles. *Evolution* **51**, 1742–1750. (doi:10.2307/2410997)

Abrams, P. A. & Matsuda, H. 1997b Fitness minimization and dynamic instability as a consequence of predator–prey coevolution. *Evol. Ecol.* **11**, 1–20. (doi:10.1023/A:1018445517101)

Allen, J. C., Schaffer, W. M. & Rosko, D. 1993 Chaos reduces species extinction by amplifying local population noise. *Nature* **364**, 229–232. (doi:10.1038/364229a0)

Barnosky, A. 2001 Distinguishing the effects of the Red Queen and Court Jester on Meiocene mammal evolution in the northern Rocky Mountains. *J. Vertebr. Paleontol.* **21**, 172–185. (doi:10.1671/0272-4634(2001)021[0172:DTEOTR]2.0.CO;2)

Beatty, J. 2006 Replaying life's tape. *J. Phil.* **103**, 336–362.

Bell, G. 2007 The evolution of trophic structure. *Heredity* **99**, 494–505. (doi:10.1038/sj.hdy.6801032)

Benkman, C. W. 1999 The selection mosaic and diversifying coevolution between crossbills and lodgepole pine. *Am. Nat.* **154**, S75–S91. (doi:10.1086/303213)

Berenbaum, M. R. & Zangerl, A. R. 1992 Genetics of physiological and behavioral resistance to host furanocoumarins in the parsnip webworm. *Evolution* **46**, 1373–1384. (doi:10.2307/2409943)

Berenbaum, M. R., Zangerl, A. R. & Nitao, J. K. 1986 Constraints on chemical coevolution—wild parsnips and the parsnip webworm. *Evolution* **40**, 1215–1228. (doi:10.2307/2408949)

Bohannan, B. J. M. & Lenski, R. E. 2000 Linking genetic change to community evolution: insights from studies of bacteria and bacteriophage. *Ecol. Lett.* **3**, 362–377. (doi:10.1046/j.1461-0248.2000.00161.x)

Brodie Jr, E. D., Ridenhour, B. J. & Brodie III, E. D. 2002 The evolutionary response of predators to dangerous prey: hotspots and coldspots in the geographic mosaic of coevolution between garter snakes and newts. *Evolution* **56**, 2067–2082.

Buckling, A. & Rainey, P. B. 2002 Antagonistic coevolution between a bacterium and a bacteriophage. *Proc. R. Soc. Lond. B* **269**, 931–936. (doi:10.1098/rspb.2001.1945)

Caldarelli, G., Higgs, P. G. & McKane, A. J. 1998 Modeling coevolution in multispecies communities. *J. Theor. Biol.* **193**, 345–358. (doi:10.1006/jtbi.1998.0706)

Champagnat, N., Ferrière, R. & Méléard, S. 2006 Unifying evolutionary dynamics: from individual stochastic processes to macroscopic models. *Theor. Popul. Biol.* **69**, 297–321. (doi:10.1016/j.tpb.2005.10.004)

Clayton, D. H., Bush, S. E., Goates, B. M. & Johnson, K. P. 2003 Host defenses reinforce host–parasite cospeciation. *Proc. Natl Acad. Sci. USA* **100**, 15 694–15 699. (doi:10.1073/pnas.2533751100)

Cohen, J. E., Pimm, S. L., Yodzis, P. & Saldan, J. 1993 Body sizes of animal predator and animal prey in food webs. *J. Anim. Ecol.* **62**, 67–78. (doi:10.2307/5483)

Cohen, J. E., Schittler, D. N., Raffaelli, D. G. & Reuman, D. C. 2009 Food webs are more than the sum of their tritrophic parts. *Proc. Natl Acad. Sci. USA* **106**, 2335–2340.

Colombo, A., Dercole, F. & Rinaldi, S. 2008 Remarks on metacommunity synchronization with application to prey–predator systems. *Am. Nat.* **171**, 430–442. (doi:10.1086/528959)

Currie, C. R., Wong, B., Stuart, A. E., Schultz, T. R., Rehner, S. A., Mueller, U. G., Sung, G. H., Spatafora, J. W. & Straus, N. A. 2003 Ancient tripartite coevolution in the attine ant–microbe symbiosis. *Science* **299**, 386–388. (doi:10.1126/science.1078155)

Decaestecker, E., Gaba, S., Raeymaekers, J. A. M., Stoks, R., Van Kerckhoven, L., Ebert, D. & De Meester, L. 2007 Host–parasite 'Red Queen' dynamics archived in pond sediment. *Nature* **450**, 870–873. (doi:10.1038/nature06291)

Dercole, F., Ferrière, R. & Rinaldi, S. 2002 Ecological bistability and evolutionary reversals under asymmetrical competition. *Evolution* **56**, 1081–1090.

Dercole, F., Irisson, J.-O. & Rinaldi, S. 2003 Bifurcation analysis of a prey–predator coevolution model. *SIAM J. Appl. Math.* **63**, 1378–1391. (doi:10.1137/S0036139902411612)

Dercole, F., Gragnani, A., Ferrière, R. & Rinaldi, S. 2006 Coevolution of slow–fast populations: evolutionary sliding, evolutionary pseudo-equilibria and complex Red Queen dynamics. *Proc. R. Soc. B* **273**, 983–990. (doi:10.1098/rspb.2005.3398)

Dieckmann, U. & Law, R. 1996 The dynamical theory of coevolution: a derivation from stochastic ecological processes. *J. Math. Biol.* **34**, 579–612. (doi:10.1007/BF02409751)

Dieckmann, U., Marrow, U. & Law, R. 1995 Evolutionary cycling in predator–prey interactions: population dynamics and the Red Queen. *J. Theor. Biol.* **176**, 91–102. (doi:10.1006/jtbi.1995.0179)

Dixon, P. A., Milicich, M. J. & Sugihara, G. 1999 Episodic fluctuations in larval supply. *Science* **283**, 1528–1530. (doi:10.1126/science.283.5407.1528)

Doebeli, M. 1997 Genetic variation and the persistence of predator–prey interactions in the Nicholson–Bailey model. *J. Theor. Biol.* **188**, 109–120. (doi:10.1006/jtbi.1997.0454)

Doebeli, M. & Dieckmann, U. 2003 Speciation along environmental gradients. *Nature* **421**, 259–264. (doi:10.1038/nature01274)

Ebert, D. 2008 Host–parasite coevolution: insights from the Daphnia–parasite model system. *Curr. Opin. Microbiol.* **11**, 290–301. (doi:10.1016/j.mib.2008.05.012)

Elena, S. F. & Lenski, R. E. 2003 Evolution experiments with microorganisms: the dynamics and genetic bases of adaptation. *Nat. Rev. Genet.* **4**, 457–469. (doi:10.1038/nrg1088)

Elena, S. F., Cooper, V. S. & Lenski, R. E. 1996 Punctuated evolution caused by selection of rare beneficial mutations. *Science* **272**, 1802–1804. (doi:10.1126/science.272.5269.1802)

Ellner, S. P. & Turchin, P. 1995 Chaos in a noisy world: new methods and evidence from time series analysis. *Am. Nat.* **145**, 343–375. (doi:10.1086/285744)

Ellner, S. P. & Turchin, P. 2005 When can noise induce chaos and why does it matter: a critique. *Oikos* **111**, 620–631. (doi:10.1111/j.1600-0706.2005.14129.x)

Feldman, C. R., Brodie Jr, E. D., Brodie III, E. D. & Pfrender, M. E. 2009 The evolutionary origins of beneficial alleles during the repeated adaptation of garter snakes to deadly prey. *Proc. Natl Acad. Sci. USA* **106**, 13 415–13 420. (doi:10.1073/pnas.0901224106)

Ferrière, R. 2000 Adaptive responses to environmental threats: evolutionary suicide, insurance and rescue. *Options* **Spring 2000**, 12–16.

Ferrière, R. & Fox, G. A. 1995 Chaos and evolution. *Trends Ecol. Evol.* **10**, 480–485. (doi:10.1016/S0169-5347(00)89194-6)

Ferrière, R. & Gatto, M. 1995 Lyapunov exponents and the mathematics of invasion in oscillatory or chaotic populations. *Theor. Popul. Biol.* **48**, 126–171. (doi:10.1006/tpbi.1995.1024)

Ferrière, R., Gauduchon, M. & Bronstein, J. L. 2007 Evolution and persistence of obligate mutualists and exploiters: competition for partners and evolutionary immunization. *Ecol. Lett.* **10**, 115–126. (doi:10.1111/j.1461-0248.2006.01008.x)

Futuyma, D. J. 1983 Evolutionary interactions among herbivorous insects and plants. In *Coevolution* (eds D. J. Futuyma & M. Slatkin), pp. 207–231. Sunderland, MA: Sinauer Associates.

Gaba, S. & Ebert, D. 2009 Time-shift experiments as a tool to study antagonistic coevolution. *Trends Ecol. Evol.* **24**, 226–232. (doi:10.1016/j.tree.2008.11.005)

Gallet, R., Tully, T. & Evans, M. E. K. 2009 Ecological conditions affect evolutionary trajectory in a predator–prey system. *Evolution* **63**, 641–651. (doi:10.1111/j.1558-5646.2008.00559.x)

Gandon, S. 2002 Local adaptation and the geometry of host–parasite coevolution. *Ecol. Lett.* **5**, 246–256. (doi:10.1046/j.1461-0248.2002.00305.x)

Gandon, S. 2004 Evolution of multihost parasites. *Evolution* **58**, 455–469.

Gavrilets, S. 1997 Coevolutionary chase in exploiter–victim systems with polygemic characters. *J. Theor. Biol.* **186**, 527–534. (doi:10.1006/jtbi.1997.0426)

Gross, T., Ebenhoh, W. & Feudel, U. 2005 Long food chains are in general chaotic. *Oikos* **109**, 135–144. (doi:10.1111/j.0030-1299.2005.13573.x)

Hamilton, W. D., Axelrod, R. & Tanese, R. 1990 Sexual reproduction as an adaptation to resist parasites (a review). *Proc. Natl Acad. Sci. USA* **35**, 3566–3573. (doi:10.1073/pnas.87.9.3566)

Hanifin, C. T., Brodie Jr, E. D. & Brodie III, E. D. 2008 Phenotypic mismatches reveal escape from arms-race coevolution. *PLoS Biol.* **6**, e60. (doi:10.1371/journal.pbio.0060060)

Hastings, A. 2004 Transients: the key to long-term ecological understanding? *Trends Ecol. Evol.* **19**, 39–45. (doi:10.1016/j.tree.2003.09.007)

Hastings, A. & Powell, T. 1991 Chaos in a three species food chain. *Ecology* **72**, 896–903. (doi:10.2307/1940591)

Hofbauer, J. & Sigmund, K. 1998 *Evolutionary games and population dynamics*. Cambridge, UK: Cambridge University Press.

Jones, E. I., Ferrière, R. & Bronstein, J. L. 2009 Eco-evolutionary dynamics of mutualists and exploiters. *Am. Nat.* **174**, 780–794. (doi:10.1086/647971)

Kelley, P. H., Kowalewski, M. & Hansen, T. A. (eds) 2003 *Predator–prey interaction in the fossil record*. New York, NY: Plenum.

Khibnik, A. I. & Kondrashov, A. S. 1997 Three mechanisms of Red Queen dynamics. *Proc. R. Soc. Lond. B* **264**, 1049–1056. (doi:10.1098/rspb.1997.0145)

Kirkwood, T. B. L. M. & Bangham, C. R. M. 1994 Cycles, chaos, and evolution in virus cultures: a model of defective interfering particles. *Proc. Natl Acad. Sci. USA* **91**, 8685–8689. (doi:10.1073/pnas.91.18.8685)

Kisdi, E. & Liu, S. 2006 Evolution of handling time can destroy the coexistence of cycling predators. *J. Evol. Biol.* **19**, 49–58. (doi:10.1111/j.1420-9101.2005.00993.x)

Koskella, B. & Lively, C. M. 2009 Evidence for negative frequency-dependent selection during experimental co-evolution of a freshwater snail and a sterilizing trematode. *Evolution* **63**, 2213–2221. (doi:10.1111/j.1558-5646.2009.00711.x)

Lai, Y. C., Liu, Z. & Billings, L. 2003 Noise-induced unstable dimension variability and transition to chaos in random dynamical systems. *Phys. Rev. E* **67**, 026210. (doi:10.1103/PhysRevE.67.026210)

Lenormand, T., Roze, D. & Rousset, F. 2008 Stochasticity in evolution. *Trends Ecol. Evol.* **24**, 157–165.

Lenski, R. E. & Levin, B. R. 1985 Constraints on the coevolution of bacteria and virulent phage: a model, some experiments, and predictions for natural communities. *Am. Nat.* **125**, 585–602. (doi:10.1086/284364)

Lively, C. M. 1999 Migration, virulence, and the geographic mosaic of adaptation by parasites. *Am. Nat.* **153**, S34–S47. (doi:10.1086/303210)

Loeuille, N. & Loreau, M. 2005 Evolutionary emergence of size-structured food webs. *Proc. Natl Acad. Sci. USA* **102**, 5761–5766. (doi:10.1073/pnas.0408424102)

Loeuille, N., Loreau, M. & Ferrière, R. 2002 Consequences of plant–herbivore coevolution on the dynamics and functioning of ecosystems. *J. Theor. Biol.* **217**, 369–381. (doi:10.1006/jtbi.2002.3032)

Lythgoe, K. A. & Read, A. F. 1998 Catching the Red Queen? The advice of the Rose. *Trends Ecol. Evol.* **13**, 473–474. (doi:10.1016/S0169-5347(98)01486-4)

Marrow, P., Law, R. & Cannings, C. 1992 The coevolution of predator–prey interactions: ESSs and Red Queen dynamics. *Proc. R. Soc. Lond. B* **250**, 133–141. (doi:10.1098/rspb.1992.0141)

Marrow, P., Dieckmann, U. & Law, R. 1996 Evolutionary dynamics of predator–prey systems: an ecological perspective. *J. Math. Biol.* **13**, 556–578. (doi:10.1007/BF02409750)

Martin, A. P. & Palumbi, S. R. 1993 Body size, metabolic rate, generation time, and the molecular clock. *Proc. Natl Acad. Sci. USA* **90**, 4087–4091. (doi:10.1073/pnas.90.9.4087)

Matsuda, H. & Abrams, P. A. 1994 Runaway evolution to self-extinction under asymmetrical competition. *Evolution* **48**, 1764–1772. (doi:10.2307/2410506)

May, R. M. & Anderson, R. M. 1983 Epidemiology and genetics in the coevolution of parasites and hosts. *Proc. R. Soc. Lond. B* **219**, 281–313. (doi:10.1098/rspb.1983.0075)

Metz, J. A. J., Nisbet, R. M. & Geritz, S. A. H. 1992 How should we define fitness for general ecological scenarios? *Trends Ecol. Evol.* **7**, 198–202. (doi:10.1016/0169-5347(92)90073-K)

Metz, J. A. J., Geritz, S. A. H., Meszéna, G., Jacobs, F. J. A. & van Heerwaarden, J. S. 1996 Adaptive dynamics: a geometrical study of the consequences of nearly faithful reproduction. In *Stochastic and spatial structures of dynamical systems* (eds S. J. van Strien & S. M. Verduyn Lunel), pp. 183–231. Burlington, MA: Elsevier Science.

Moya, A., Holmes, E. C. & Gonzalez-Candelas, F. 2004 The population genetics and evolutionary epidemiology of RNA viruses. *Nat. Rev. Microbiol.* **2**, 279–288. (doi:10.1038/nrmicro863)

Nowak, M. A. & Sigmund, K. 2003 Chaos and the evolution of cooperation. *Proc. Natl Acad. Sci. USA* **90**, 5091–5094.

Nuismer, S. L. & Doebeli, M. 2004 Genetic correlations and the coevolutionary dynamics of three-species systems. *Evolution* **58**, 1165–1177.

Nuismer, S. L., Ridenhour, B. J. & Oswald, B. P. 2007 Antagonistic coevolution mediated by phenotypic differences between quantitative traits. *Evolution* **61**, 1823–1834. (doi:10.1111/j.1558-5646.2007.00158.x)

Olden, J. D., LeRoy Poff, N., Douglas, M. R., Douglas, M. E. & Faush, K. D. 2004 Ecological and evolutionary consequences of biotic homogenization. *Trends Ecol. Evol.* **19**, 18–24. (doi:10.1016/j.tree.2003.09.010)

Palumbi, S. R. 1994 Genetic divergence, reproductive isolation, and marine speciation. *Annu. Rev. Ecol. Syst.* **25**, 547–572. (doi:10.1146/annurev.es.25.110194.002555)

Rand, D. A. & Wilson, H. B. 1991 Chaotic stochasticity—a ubiquitous source of unpredictability in epidemics. *Proc. R. Soc. Lond. B* **246**, 179–184. (doi:10.1098/rspb.1991.0142)

Robert, M. & Sorci, G. 1999 Rapid increase of host defence against brood parasites in a recently parasitized area: the case of village weavers in Hispaniola. *Proc. R. Soc. Lond. B* **266**, 941–946. (doi:10.1098/rspb.1999.0727)

Rosenzweig, M. L., Brown, J. S. & Vincent, T. L. 1987 Red Queen and ESS: the coevolution of evolutionary rates. *Evol. Ecol.* **1**, 59–94. (doi:10.1007/BF02067269)

Royama, T. 1992 *Analytical population dynamics*. London, UK: Chapman & Hall.

Salathe, M., Kouyos, R. D. & Bonhoeffer, S. 2008 The state of affairs in the kingdom of the Red Queen. *Trends Ecol. Evol.* **23**, 439–445. (doi:10.1016/j.tree.2008.04.010)

Seger, J. 1992 Evolution of exploiter–victim relationships. In *Natural enemies: the population biology of predators, parasites and diseases* (ed. M. J. Crawley), pp. 3–25. Oxford, UK: Blackwell Scientific.

Shoresh, N., Hegreness, M. & Kishony, R. 2008 Evolution exacerbates the paradox of the plankton. *Proc. Natl Acad. Sci. USA* **105**, 12 365–12 369. (doi:10.1073/pnas.0803032105)

Solé, R. V. & Sardanyés, J. 2007 Red Queen strange attractors in host–parasite replicator gene-for-gene coevolution. *Chaos Soliton. Fract.* **32**, 1666–1678.

Stegen, J. C., Enquist, B. J. & Ferrière, R. 2009 Advancing the metabolic theory of biodiversity. *Ecol. Lett.* **12**, 1001–1015. (doi:10.1111/j.1461-0248.2009.01358.x)

Stenseth, N. C. & Maynard Smith, J. 1984 Coevolution in ecosystems: Red Queen evolution or stasis. *Evolution* **38**, 870–880. (doi:10.2307/2408397)

Strauss, S. Y., Sahli, H. & Conner, J. K. 2005 Toward a more trait-centered approach to diffuse (co)evolution. *New Phytol.* **165**, 81–89. (doi:10.1111/j.1469-8137.2004.01228.x)

Thompson, J. N. 1994 *The coevolutionary process*. Chicago, IL: Chicago University Press.

Thompson, J. N. 2005 *The geographic mosaic of coevolution*. Chicago, IL: Chicago University Press.

Thrall, P. H., Hochberg, M. E., Burdon, J. J. & Bever, J. D. 2007 Coevolution of symbiotic mutualists and parasites in a community context. *Trends Ecol. Evol.* **22**, 120–126. (doi:10.1016/j.tree.2006.11.007)

Toju, H. 2009 Natural selection drives the fine-scale divergence of a coevolutionary arms race involving a longmouthed weevil and its obligate host plant. *BMC Evol. Biol.* **9**, 273–292. (doi:10.1186/1471-2148-9-273)

Toju, H. & Sota, T. 2006 Imbalance of predator and prey armament: geographic clines in phenotypic interface and natural selection. *Am. Nat.* **167**, 105–117. (doi:10.1086/498277)

Toju, H. & Sota, T. 2009 Do arms races punctuate evolutionary stasis? Unified insights from phylogeny, phylogeography and microevolutionary processes. *Mol. Ecol.* **18**, 3940–3954. (doi:10.1111/j.1365-294X.2009.04340.x)

Travisano, M., Mongold, J. A., Bennett, A. F. & Lenski, R. E. 1995 Experimental tests of the role of adaptation, chance, and history in evolution. *Science* **267**, 87–90. (doi:10.1126/science.7809610)

Turchin, P. 2003 *Complex population dynamics: a theoretical/empirical synthesis*. Princeton, NJ: Princeton University Press.

Van Valen, L. 1973 A new evolutionary law. *Evol. Theory* **1**, 1–30.

Vermeij, G. J. 1982 Unsuccessful predation and evolution. *Am. Nat.* **120**, 701–720. (doi:10.1086/284025)

Vermeij, G. J. 1994 The evolutionary interaction among species—selection, escalation, and coevolution. *Annu. Rev. Ecol. Syst.* **25**, 219–236. (doi:10.1146/annurev.es.25.110194.001251)

Williams, R. J. & Martinez, N. D. 2000 Simple rules yield complex food webs. *Nature* **404**, 180–183. (doi:10.1038/35004572)