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When do optimisation arguments make evolutionary sense?

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1. Introduction: philosophical preliminaries

The primary goal of science is understanding, i.e., creating orderly pictures of the world compatible with our observations. The test for, as well as the usefulness of such pictures is that they allow extrapolating from previous observations. This predictive capability is the basis for the hypothetico-deductive method (our main save-guard from wishful self-delusion) as well as for applications. Of course, regression analysis performs the same feats. The difference is that the predictive capabilities of good scientific theories extend far beyond the experimental situations from which they were inductively derived. Yet, contrary to the standard tenet, predictive capabilities are not the only proof of quality. For example, population genetics has made immense contributions to our understanding by showing that Mendel's particulate mechanism is compatible with the observations made on quantitative traits and that imperceptible selective pressures can have large effects over geologically very short time periods, thus overthrowing earlier naïve ideas to these effects. Yet, the reach of both pieces of theory in the cases where they can be tested in concrete instances effectively go but little beyond that of mere regression. What mattered is that the theory introduced a proper way of looking at the problems, and thereby made extrapolation possible. By now an impressive population genetical theory of short term adaptive evolution has been constructed, as surveyed in the Chapters by Warren Ewens [16] and Reinhard Bürger [6]. At the predictive end this theory does not reach much beyond the earlier feats. Still, these theoretical developments have greatly contributed to our understanding, largely since they allow analysing "what if" scenarios on a mechanistically founded and further essentially tautological basis, and thus enrich our naturally rather poor intuition.

The main reason for the limited predictive reach of population genetics theory is that genotypic fitnesses come in as unspecified phenomenological parameters, or, more recently, are specified through an assumed special genotype to phenotype map combined with a simple phenomenological ecological model. Only in the theory of neutral evolution there is a specification based on assumed strong first principles: all fitnesses are equal to one (or zero in the continuous time perspective to which we shall adhere below). The latter assumption leads to both that theory's mathematical strength and its limitations as a tool: although used in all sorts of inferences, the outcomes of those inferences hinge on this specific, often poorly tested, and moreover very non-generic, assumption.

To get further at the predictive side we need a more realistic handle on fitness. Mechanistically, fitness is determined by how the traits of phenotypes influence their population dynamical performance. Not only that, often those traits have our main interest in the first place. To link that primary focus on traits to population genetics we have to assess the map from genotype to phenotype and the ecological background for the population dynamical

performance. In general, little can still be said about the former, notwithstanding the great molecular advances. Hence, most theoretical efforts concentrate on deriving predictions based only on the relation between traits and performance, sidestepping the need for population genetical modeling and the concomittant need for knowing the genetic basis of those traits. The main tool were initially, and to a large extent still are, optimisation models in which a presumed fitness proxy, like the strength of a bone or an energy intake rate is maximised as a function of a set of trait vectors delimited by constraints, justified by their users with a hand-waving reference to Fisher's fundamental theorem (c.f. the Chapters by Ewens [16] and Bürger [6]).

Better thought out approaches are based on ESS theory (see the Chapter by Metz [34, Subsection 2.2]). ESS is an abbreviation of Evolutionarily Stable Strategy. However, as the definition of an ESS does not bring with it that an ESS is stable in the standard mathematical sense (see e.g. [34, Figure 2.4]), we prefer to interpret the abbreviation as Evolutionarily Steady Strategy. ESSes are the traps of any evolutionary process driven by the invasion of new mutants. They are defined as such values of the trait vector, here called strategies, that no mutant playing an alternative strategy can invade in the environment produced by them. The important difference with the optimisation approach is that the ESS argument accounts for the fact that fitnesses not only depend on the traits of phenotypes but also on the environment in which those phenotypes live, and that this environment is not constant but codetermined by the phenotypes that are currently around (for if this were not the case populations would either grow to infinity or go extinct, except when the fitnesses are zero).

The ESS approach not only is better founded, it also has the advantage that we do not need an ever applicable fitness concept, which may exist in the simplified world of population genetical theory but does not do so so readily in the more messy ecologies of real life. Instead it suffices to have a fitness concept that characterises the potential for population growth of mutant phenotypes in an environment set by resident phenotypes, not yet influenced by the mutant.

In this Chapter we investigate how optimisation approaches fit with the ESS viewpoint. There are three reasons for embarking on such an effort. The first one is practical. We want to develop a feel for the reach of the optimisation results. In general, optimisation approaches appear to work rather well, notwithstanding their basically flawed methodology. This paradox will be resolved at the end of Subsection 2.3. The optimisation literature is not so much wrong as imprecise in that its reach is far less than suggested, and its results have been put only to correspondingly restricted tests. The next two reasons come into play when we want to do away with those strong implicit restrictions. The first one is again practical. Given the relative simplicity of optimisation procedures we want a handle on how to rig an eco-evolutionary model so that its ESSes can be calculated from an optimisation principle, as well as insight into the robustness of the results from such limited models.

Finally, on the fundamental side there is the wish for insight on a meta-level. In precisely what manner do the various approaches in the literature fit together? In the textbooks one can find various hand-waving answers. We aim for precise ones.

2. Setting the stage: technical preliminaries

As the concepts and their technical implementation described below are not only needed as context for the discussion of evolutionary optimisation arguments but are of considerable importance in their own right, we have strived for a self-contained exposition. We assume throughout that populations are large and relatively well mixed, i.e., any individual is directly or indirectly affected by a large number of other individuals each of which on average has but a small effect on the demographic behaviour of the focal individual. (For some further extensions, see [33, Section 'Aggregates'].) Moreover, for the ease of the exposition we assume till Subsection 2.4 that reproduction is clonal.

2.1. Fitness and fitness proxies

The effectiveness of ESS calculations is based on the close to universal existence of a scalar quantity, called fitness, characterising the speed at which a phenotype can invade in a given environment. (NB: This fitness concept is essentially different from the population genetical one; see the Chapter by Metz [34, Subsection 2.2.].) Here we define environment as anything outside an individual that influences its population dynamical behaviour, which by definition consists of impinging on the environment, giving birth and dying (see e.g. [33, 35, 41, 40]). We then can construct a Markovian representation of that behaviour in terms of a state space, transition probabilities that depend on the course of the environment and outputs that either deterministically depend on, or occur in a Poisson (or Poisson cluster) process with rates that depend on the individual's state and the condition of the environment. Given the course of the environment, individuals independently move through their state spaces, the population state is a measure over this space, and the expectation of this measure, which is again a measure, moves according to a positive linear evolutionary system. The theory of such systems then tells that generally the expected size of a population in an ergodic environment will in the long run on average grow or decline exponentially (for details, see [33, 17]). The per capita rate of this growth, to be denoted generally as ρ, or in accordance with standard custom in the special case of constant environments as r, is the sought after fitness.

Example. Finite state individuals and clonal reproduction. The following example lacks in biological realism but is the simplest one demonstrating the basic principles. A partial justification may be the hunch that any useful model has to be approximable (uniformly for all relevant environments) by a finite state model.

The expected growth of a population of independent finite state individuals in continuous time in a constant environment is given by

$$\frac{\mathrm{d}}{\mathrm{d}t}N = (\mathbf{R} + \mathbf{T})N,$$

N the vector of spatial densities of individuals in different states. (In our present context N refers to the mutants. To keep the notational burden low we suppress dependences on the trait vector of the mutants and resident environment.) The matrices $\mathbf{R}$ and $\mathbf{T}$ are built up from per capita rates. The off-diagonal components of $\mathbf{T}$ equal the transition rates between the corresponding states, the diagonal components equal minus the overall rates of transitions from the states minus the state dependent death rates. The components of $\mathbf{R}$ equal the average per capita birth rates in dependence on the state of the parent split according to the state of the offspring. The fitness of the type of individuals under consideration corresponds to the rightmost eigenvalue of $\mathbf{R}+\mathbf{T}$ (which is necessarily real and goes with a positive eigenvector since otherwise the trajectory would leave the positive cone).

The theory of branching processes moreover tells that when a population is started with a single individual it will, barring some technical conditions, eventually either go extinct or grow exponentially, with the probability of the latter being positive if and only if its fitness is so (see [27, 3, 4, 21]).

From any more general perspective ρ necessarily is a function of two variables, the trait vector of the individuals Y and the environment E, to be written as $\rho(Y|E)$. (Depending on the context we may suppress one or both of these arguments.)

The existence of such a fitness is the basis for all deliberations below. However, given its existence it is often possible to replace ρ by some more easily determined quantity that leads to the same outcome for the calculations that have our interest. For example, in optimisation calculations ρ can be replaced with any quantity that is monotonically related to it, and in many other types of ESS calculation one may replace ρ with any sign-equivalent quantity. We shall refer to such quantities as fitness proxies. An example of an often used fitness proxy of the first type is the average rate of energy intake. It should be noted though, that being a fitness proxy is always predicated on additional assumptions. For instance, it may help a forager little to increase its average energy intake in an environment where doing so drastically increases its exposure to predation. An important fitness proxy of the second type, restricted to non-fluctuating environments, is the logarithm of the average life-time offspring number $\ln(R_0)$, where R_0 is defined as the dominant eigenvalue (or more generally spectral radius, or, still more generally, Perron root) of the next generation operator. (The advantage of using births as reference points in the life cycle is that usually the set of birth states is considerably smaller than the full complement of states necessary to describe how an individual passes through its life. However, if that happens to be more convenient, other points in the life cycle where the individual by

necessity can be in but a few states may also be used as basis for the bookkeeping.) This next generation operator is constructed by calculating from a model for the behaviour of individuals how many offspring are born on average in different birth states dependent on the birth state of the parent (see e.g. [10]).

Example. Finite state individuals and clonal reproduction, continued. Order the individual states so that the birth states come first. To step back and forth between a population state and a birth based approach we need a matrix $\mathbf{K}$ injecting the vector of birth rates into the space of changes in densities of all individuals, young and old alike:

$$\mathbf{K}^T = \begin{pmatrix} 1 & 0 & \cdots & 0 & 0 & \cdots & \cdots & 0 \\ 0 & \ddots & \ddots & \vdots & \vdots & & & \vdots \\ \vdots & \ddots & \ddots & 0 & \vdots & & & \vdots \\ 0 & \cdots & 0 & 1 & 0 & \cdots & \cdots & 0 \end{pmatrix}.$$

As can be seen from this formula, $\mathbf{K}^{\mathrm{T}}$ maps the space of population rates back onto the space of birth rates.

Arguing from first principles one can calculate the next generation matrix by first calculating the expected times that the Markov chain stays in each possible state before the individual dies as $\mathbf{T}^{-1}\mathbf{K}$, after which the next generation matrix can be expressed as $\mathbf{L} = \mathbf{K}^{\mathrm{T}}\mathbf{R}\mathbf{T}^{-1}\mathbf{K}$.

For the general case we observe that due to our assumption of environmental constancy the state of an individual can always be replaced by a proxy state consisting of age together with the state at birth, giving the production of offspring dependent on the state at birth as an operator valued function $\mathbf{\Lambda}(a)$. Integrating out over age gives the next-generation operator $\mathbf{L}$.

Example. Finite state individuals and clonal reproduction, continued. In our finite state model the state of an individual moves according to a Markov chain with killing, and the probability that individuals born in certain birth states at age a are alive and reside in certain states is given by the matrix $\mathrm{e}^{a\mathbf{T}}\mathbf{K}$. Hence the average birth rate at age a split according to the birth state of the parent and that of the kids is $\mathbf{\Lambda}(a) = \mathbf{K}^{\mathrm{T}}\mathbf{R}\mathrm{e}^{a\mathbf{T}}\mathbf{K}$. This expression shows that given the mechanism as embodied in the matrices $\mathbf{R}$ and $\mathbf{T}$ it is possible to calculate the average birth rate of an individual from only its state at birth and age. Hence, age and birth state together are a proxy state for the goal of calculating the average birth rates of individuals.

The vector of population birth rates B satisfies a vectorial version of Lotka's integral equation from mathematical demography

$$B(t) = \int_0^{t-t_0} \mathbf{\Lambda}(a)B(t-a)\mathrm{d}a + \mathbf{K}^{\mathrm{T}}\mathbf{R}e^{(t-t_0)\mathbf{T}}N(t_0)$$

(if the initial datum is given in terms of the population composition at time t_0), which for $t_0 \to -\infty$ reduces to

$$B(t) = \int_0^\infty \mathbf{\Lambda}(a)B(t-a)\mathrm{d}a.$$

Substitution of an exponential trial solution $B(t) = \mathrm{e}^{rt}U$ gives that the invasion fitness r can be calculated from $\mathbf{\Lambda}$ by solving the characteristic equation:

$$\text{dominant eigenvalue of } \tilde{\mathbf{\Lambda}}(r) = 1, \quad r \in \mathbb{R}$$

(or equivalently $\det(\mathbf{I} - \tilde{\mathbf{\Lambda}}(r)) = 0$, $r \in \mathbb{R}$), with

$$\begin{aligned}\tilde{\mathbf{\Lambda}}(z) := \int_0^\infty \mathrm{e}^{-za}\mathbf{\Lambda}(a)\mathrm{d}a &= \mathbf{K}^{\mathrm{T}}\mathbf{R}(\mathbf{T} - z\mathbf{I})^{-1}\mathbf{K} \quad \text{for } z > z_0,\\ &= \infty \qquad\qquad\qquad\qquad\quad \text{for } z \le z_0,\end{aligned}$$

with z_0 the rightmost root of $\det(\mathbf{T} - z\mathbf{I})$, and that U equals the, positive, eigenvector with eigenvalue 1 of $\tilde{\mathbf{\Lambda}}(r)$. The general theory of renewal equations tells that for $t_0 \to -\infty$ indeed $B(t)$ will grow like $\mathrm{e}^{rt}U$. From the fact that also $B(t) = \mathbf{K}^{\mathrm{T}}\mathbf{R}N(t)$ it follows that the r found in this manner is equal to the dominant eigenvalue r of $\mathbf{R} + \mathbf{T}$.

To prove the sign equivalence of r and $\ln(R_0)$ note that

$$\mathbf{L} := \int_0^\infty \mathbf{\Lambda}(a)\mathrm{d}a = \tilde{\mathbf{\Lambda}}(0) \quad \text{and} \quad R_0 = \text{dominant eigenvalue of } \mathbf{L}.$$

Since all components of $\tilde{\mathbf{\Lambda}}(z)$ are positive and decrease with z, also its dominant eigenvalue decreases with z. Hence, r is positive when $\ln(R_0) > 0$ and is negative when $\ln(R_0) < 0$.

The following argument shows that R_0 rightfully can be interpreted as an average lifetime offspring number. The average lifetime numbers of offspring by individuals born in different states equals $\mathbf{1}^{\mathrm{T}}\mathbf{L}$, where $\mathbf{1}$ is a vector that has all its components equal to 1. The natural probability distribution to average these numbers over is the stationary distribution generated by the generation process itself, i.e., the right eigenvector U of $\mathbf{L}$ corresponding to R_0, normalised such that $\mathbf{1}^{\mathrm{T}}U = 1$. Doing so gives $\mathbf{1}^{\mathrm{T}}\mathbf{L}U = \mathbf{1}^{\mathrm{T}}R_0U = R_0\mathbf{1}^{\mathrm{T}}U = R_0$.

A further, partial, proxy for $\ln(R_0)$ in cases where the next generation operator is representable by a matrix $\mathbf{L}$ is $Q := -\det(\mathbf{I} - \mathbf{L})$. Q is sign equivalent to $\ln(R_0)$ where it counts most, that is, close to $\ln(R_0) = 0$. Moreover, for path connected trait spaces and an $\mathbf{L}$ that depends continuously on the traits, if $r(X_i) = 0$ for $i = 1, \ldots, k$ then $Q(X_i) = 0$ for the same X_i and if moreover $Q(Y) < 0$ for all Y different from those X_i then also $r(Y) < 0$ for those Y (see [37]).

2.2. Resident environments and invasion fitness

The only environments that matter in ESS calculations are environments generated by the attractors of some, so-called resident, community. In nature populations are necessarily bounded. If this bound were too small our population would go extinct in too short a time for it to reach an ESS. Hence we assume that the population is infinite in numbers although bounded in density, i.e., number of individuals per unit of area or volume. The community then follows a deterministic dynamics with as state space for each population a closed bounded subset of the cone of positive measures over the state space of the individuals, and as total state space the product of the state spaces of the comprising species, plus the state spaces of the dynamics of any inanimate resources. With an infinitesimal amount of noise the states of such communities will approach an "extinction preserving chain attractor" (see [26, 19]); with larger amounts of noise the community will in general end up in a stochastic attractor, that is, a stationary distribution of community states. We will throughout assume that the community attractor generates an ergodic environment (to all appearances exceptions to this assumption are rare).

Notation. The environment generated by a coalition of clones $C = \{X_1, ..., X_k\}$ will be written as

$$E_{\text{attr}}(C)$$

with the convention that we write just X for $C = \{X\}$.

Convention 2.1. We take the use of the expression $E_{\text{attr}}(C)$ as implying that for a community starting with all types $X \in C$ there exists an attractor with the densities of all those types nonzero.

For ease of exposition we moreover proceed on the

Assumption 2.2. $E_{\text{attr}}(C)$ is unique.

The domain of the function E_{attr} is therefore the space of all realisable coalitions.

Assumption 2.2 is not necessary for any of the developments below. All statements can be extended to the general case with but minor modifications, which, however, would tally up to a considerable amount of verbal clutter.

A combination of the preceding arguments leads to the

Definition 2.3. The quantity

$$s_C(Y) := \rho\,(Y | E_{\text{attr}}(C)) \tag{2.1}$$

is called the *invasion fitness* of a new type Y in a C-community.

An essential observation is

Proposition 2.4.

$$s_C(X) = 0 \quad \text{for all } X \in C. \tag{2.2}$$

Proof. The presence of X as a resident means that the density of X does not go to zero and neither can it go to infinity. Therefore its average per capita growth rate is zero. □

Hence for all $X \in C$, $s_C(X) = s_X(X) = 0$, and $s_C(Y) > s_C(X)$ whenever $s_C(Y) > 0$, and similarly with $>$ replaced by $<$ or $=$.

2.3. Calculating ESSes

The usual way of calculating ESSes is by devising some procedure to maximise $\rho(X|E)$ over all potential trait values for any feasible E, resulting in a function $X_{\rm opt}(E)$. As a next step one determines for each trait value the environment that it generates as a resident, $E_{\rm attr}(X)$. Finally one varies X to find an evolutionarily unbeatable value X^*, i.e., an X^* such that

$$X_{\rm opt}(E_{\rm attr}(X^*)) = X^*. \tag{2.3}$$

For more complicated trait spaces and ecologies solving such a combined optimisation problem and equation tends to be far from easy. Hence, determining a single optimisation principle that has to be satisfied by the ESS can be a great help.

After calculating an ESS one should preferrably ascertain that the set of trait values X_0 from which it is approximated with non-zero probability through a sequence $X_0, X_1, X_2, \ldots$ such that $s_{X_i}(X_{i+1}) > 0$, possibly interspersed with polymorphisms, is sufficiently large to warrant consideration of X^* as a potential evolutionary prediction. Although the last condition is not part and parcel of the ESS concept (it should have been!), only the attracting ESSes, customarily called CSSes , are relevant as evolutionary predictions (c.f. [11, 44, 12, 18]). (The acronym CSS is an abbreviation of the not overly informative phrase 'Continuously Stable Strategy'.) One further advantage of showing the existence of an optimisation principle is that this implies that the corresponding ESSes are globally attractive in the case of clonal and haploid organisms and may be expected to have a fair attainability in diploid Mendelian ones.

An alternative way of spotting ESSes, which also immediately gives insight in their evolutionary attractivity, is through the plotting of a so-called Pairwise Invasibility Plot (PIP), i.e., a plot of the sign of the invasion fitness of potential mutants with the potential resident trait values on the abscissa and the potential mutant trait values on the ordinate. See Figure 3.2 and the Chapter by Metz [34, Section 2, in particular Figure 2.4]. As we shall see in Subsection 3.2, such plots also provide us with an easy diagnostic for the presence or non-presence of an optimisation principle.

For higher dimensional trait spaces it is not possible to work with PIPs, but the basic idea that underlies the determination of an ESS from a PIP still goes through. Any ESS satisfies

$$G(X^*) = 0 \quad \text{with} \quad G(X) := \left.\frac{{\rm d}s_X(Y)}{{\rm d}Y}\right|_{Y=X}, \tag{2.4}$$

together with the condition that

$$\mathbf{H}(X^*) := \left.\frac{\mathrm{d}^2 s_X(Y)}{\mathrm{d}Y^2}\right|_{Y=X=X^*} \text{ is negative definite.} \tag{2.5}$$

(In adaptive dynamics the vector G^{T} is known as the selection gradient and the matrix $\mathbf{H}$ as the selection Hessian.)

In addition to ESSes, which are by definition monomorphic, there may exist Evolutionarily Steady Coalitions (ESCs), i.e., combinations of phenotypes C such that $s_C(Y) < 0$ for all $Y \notin C$. Finding non-monomorphic ESCs through an extension of the optimisation route is tricky. However, the adaptive dynamics toolbox works almost unchanged. It lets us calculate candidate ESCs by intersecting the adaptive "isoclines" (see the Chapter by Metz [34, Subsection 2.4]) defined by setting the selection gradients equal to 0,

$$G_i(C^*) = 0 \quad \text{for } i = 1, \ldots, k, \quad \text{with} \quad G_i(C) = \left.\frac{\mathrm{d}s_C(Y)}{\mathrm{d}Y}\right|_{Y=X_i}, \tag{2.6}$$

followed by a check that a so-found singular point indeed corresponds to local fitness maxima

$$\mathbf{H}_i(C^*) := \left.\frac{\mathrm{d}^2 s_{C^*}(Y)}{\mathrm{d}Y^2}\right|_{Y=X_i^*} \text{ is negative definite,} \quad \text{for } i = 1, \ldots, k. \tag{2.7}$$

The local attractivity of any found ESS or ESC can be gauged by approximating the trait substitution process with the so-called canonical equation of adaptive dynamics (see [34, Subsection 2.5]).

Combining the previous considerations shows that a good recipe for numerically finding possibly attracting candidate ESSes is running the canonical equation for a reasonable sample of initial conditions and mutational covariance matrices. Necessary and sufficient conditions for a guaranteed local convergence, independent of the mutational covariance matrix can be found in [29, 30, 31].

Resolution of the paradox from Section 1: As a final issue we point to the fact that an ESS maximises the invasion fitness in the environment as set by that strategy. Hence, if we just measure the environment we may predict the evolutionarily steady trait values that go with that environment by maximising fitness in that environment. This is why the predictions from optimisation theory work so well. Optimisation theory may not predict the outcome of evolution, for that would entail also predicting the environment that goes with the ESS, but it often very satisfactorily predicts the strategies that may be present in that environment. However, such limited predictions are of little practical use when it comes to gauging the potential consequences of purposeful or inadvertent environmental manipulation like controlled fishing regimes or human induced global warming.

2.4. Genetics

Not many species that have our interest reproduce clonally. Luckily many results from the clonal theory go through almost unaltered under Mendelian

inheritance. For the community dynamics one has to distinguish individuals according to their genotypes, and incorporate their mating opportunities with different genotypes into the description of the environment (c.f. [9]; this in the case of casual matings, with more extended pair formation it becomes necessary to extend the state space of individuals to keep track of their marriage status). Alleles reproduce clonally and as such have invasion fitnesses. So in principle we can calculate ESSes based on the population dynamics of hypothetical mutant alleles affecting the phenotype.

To link with the usually encountered arguments that tend to be implicitly based on the assumption of clonal reproduction, we define for Mendelian diploids a mock fitnesss of phenotypes by introducing a parallel clonal model with individuals passing through their lives like their Mendelian counterparts and having a reproduction equal to the average of the contributions through the micro- and macro-gametic routes (for humans semen and ova) of those counterparts. The definition of R_0 can be similarly extended. As an example we give the recipe for the calculation of R_0 when, except possibly for a sex difference, there is but a single birth state.

Example. Mendelian diploids with everybody born equal. In the case of diploid hermaphrodites with but a single birth state, R_0 equals half the sum of the average numbers of offspring fathered or mothered. The factor $1/2$ comes from the wish to define R_0 such that the outcome from naive evolutionary calculations based on this "offspring number" for individuals matches the outcome from more detailed genetically based calculations.

When the sexes are separate, the sex difference comes on top of the physiological structure, spatial position, etc.. In diploids, if everybody is born equal but for their sex, the corresponding next-generation operator is

$$\mathbf{L} = \tfrac{1}{2}\begin{pmatrix} \ell_{\mathrm{ff}} & \ell_{\mathrm{fm}} \\ \ell_{\mathrm{mf}} & \ell_{\mathrm{mm}} \end{pmatrix}.$$

with ℓ_{ff} the lifetime number of daughters of a female, ℓ_{fm} the lifetime number of daughters of a male, ℓ_{mf} the lifetime number of sons of a female, and ℓ_{mm} the lifetime number of sons of a male, all for the mutant, as they happen to occur in the environmental and genetic background provided by the resident population.

The simplest case is when the sex determination is independent of the trait in which the mutant differs from the resident as then we can write $\ell_{\mathrm{ff}} = p_{\mathrm{f}} f$, $\ell_{\mathrm{mf}} = p_{\mathrm{m}} f$, $\ell_{\mathrm{fm}} = p_{\mathrm{f}} m$, $\ell_{\mathrm{mm}} = p_{\mathrm{m}} m$, with m and f the numbers of offspring fathered and mothered over a lifetime, and p_{m} and p_{f} the probability of being born a male or a female. Therefore $\mathbf{L}$ has rank one and

$$R_0 = \tfrac{1}{2}\left(p_{\mathrm{f}} f + p_{\mathrm{m}} m\right).$$

This result could also have been obtained more directly by observing that everybody is born stochastically equal, having the same probabilities of being born male or female. We then get R_0 by just averaging over the possibilities.

As a curiosity we mention that when the trait in which the mutant differs has an influence on the sex determination we can still end up with the same

formula by defining p_{m} and p_{f} to be the asymptotic probabilities of being born a male or a female, i.e., by choosing for p_{m} and p_{f} the components of the right eigenvector U of $\mathbf{L}$, and defining m and f again as the number of offspring fathered or mothered over a lifetime, i.e., $f = \ell_{\mathrm{ff}} + \ell_{\mathrm{mf}}$, $m = \ell_{\mathrm{fm}} + \ell_{\mathrm{mm}}$. Then, by using $R_0 = \mathbf{1}^{\mathrm{T}}\mathbf{L}U$, exactly the same formula for R_0 is obtained. Only the similarity of the expressions is pleasing: to calculate p_{m} and p_{f} we first have to calculate R_0.

With the above definitions various fitness-based deductions for the clonal case go through for Mendelian inheritance. In particular, for genetically homogeneous populations the fitness of a resident equals zero (since genetically homogeneous populations breed true and resident populations by definition do not in the long run grow or decline). Moreover, the invasion of a new mutant in a homogeneous population is correctly predicted, as that mutant initially only occurs in heterozygotes that breed true by backcrossing with the homogeneous resident.

The situation for ESCs is more complicated as there may be so-called genetic constraints. So it may happen, for example, that an invading mutant heterozygote also has a positive fitness in the environmental background provided by its own homozygote. Luckily, in the so-called Ideal Free (IF) case, as in the clonal case, all phenotypes comprising an ESC have fitness zero, at least when there is only a single birth state and the ESC engenders a community dynamical equilibrium. This IF case is defined by the requirement that there are no genetic constraints whatsoever, that is, mutants can occur that produce any feasible type as heterozygotes in the genetic backgrounds supplied by the resident population. Unfortunately at the present state of knowledge about genotype to phenotype maps there is no way of predicting for what traits persistent genetic constraints can indeed be ruled out.

Remark. Whether in general the conditions $s_C(X) = 0$ for all $X \in C$ and $s_C(Y) < 0$ for all $Y \notin C$ imply that C is uninvadable by any non-neutral mutant is still unresolved. For constant environments and individuals that can be born in only one state the proof is easy as there the invasion R_0 of a mutant allele can be written as a positively weighted sum of the R_0 of the phenotypes of all the different heterozygotes in which it may occur [9, 14, 32]. Hence, if the fitness of all $X \in C$ equals zero and no alternative phenotype has positive fitness, any non-neutral genetic mutant has negative invasion fitness. It would be nice to have the issue resolved more generally.

Haploids basically follow the clonal rules. See [37] for haplo-diploids like hymenopterans in which the females are diploid and the males haploid (the supplementary material to [42] lists the many known haplo-diploid taxa). Polyploids as well as more complicated life cycles with both haploid and diploid phases as seen in mosses, ferns and various sorts of algae still remain to be studied.

3. Results

3.1. General considerations

Assumption 3.1. For any ESCs

$$\rho\,(X|E_{\rm attr}(C)) = 0 \quad \text{for all } X \in C, \tag{3.1}$$

that is, the ESCs that we consider are of clonal or haploid organisms, or in the case of Mendelian diploids they are ESSes, or more generally ESCs in which the fitness of all resident phenotypes is zero as is the case under the IF assumption at least in cases where there is only one state at birth as well as population dynamical equilibrium.

Notation. $\mathcal{X}$ will denote the set of potential trait vectors, $\mathcal{C}$ the set of all coalitions $C \in \text{domain}(E_{\rm attr})$ satistying equation (3.1), and $\mathcal{E} := E_{\rm attr}(\mathcal{C})$.

Convention 3.2. When we speak of all C this is meant to refer only to $C \in \mathcal{C}$, and when we speak of all E this is meant to refer only to $E \in \mathcal{E}$.

that is, we focus only on feasible E, i.e., E in the range of $E_{\rm attr}$, and as far as these E are concerned we restrict the discussion to clonal or haploid organisms, or in the case of Mendelian diploids to monomorphisms while for polymorphisms we restrict ourselves to environments that go with ESCs satisfying Assumption 3.1.

Definition 3.3. We call a function $\phi : \mathcal{X} \to \mathbb{R}$ an optimisation principle when under any constraint the outcome of evolution can be determined by maximising ϕ.

We shall below abbreviate this as ϕ being maximised by evolution. The proviso "for any constraint" in Definition 3.3 mirrors the usual practice of combining an optimisation principle, derived from the population dynamics, with a discussion of the dependence of the evolutionary outcome on the possible constraints.

Remark. The above definition of an optimisation principle may seem unduly restrictive. Why not just ask for an optimisation principle to have its maxima coinciding with any ESSes? The point is that mathematically speaking the latter sort of optimisation principles always exist but are totally uninformative. Just calculate the ESSes for a model and take any function that has its maxima at those points.

Optimisation principles correspond more or less to the textbook intuition for the meaning of fitness, which generally fails to account for the fact that the fitnesses of all possible types are bound to change with any change in the character of the residents.

By letting the constraint set consist of just two possible trait values it follows that

Lemma 3.4. *If ϕ is an optimisation principle then*

$$\phi(Y) < \phi(X) \Leftrightarrow s_X(Y) < 0 \quad \text{and} \quad \phi(Y) = \phi(X) \Leftrightarrow s_X(Y) = 0.$$

As a consequence, in the clonal and haploid cases the existence of an optimisation principle ϕ allows one to rule out mutual exclusion ($s_X(Y) < 0$ and $s_Y(X) < 0$) and protected polymorphisms ($s_X(Y) > 0$ and $s_Y(X) > 0$) since both lead to the contradictory conclusion $\phi(Y) < \phi(X) < \phi(Y)$.

So far we only considered ϕ as a function on $\mathcal{X}$. For many models with an optimisation principle (to wit, all those models that we have encountered) it is possible to extend ϕ to the whole of $\mathcal{C}$. Such models are characterised by the fact that for $C = \{X_1, \ldots, X_n\}$ one has $\phi(X_1) = \cdots = \phi(X_n)$. Therefore we can set $\phi(C)$ equal to the common value of the $\phi(X_i)$. This extension is consistent in that one still has $\phi(Y) > \phi(C) \Leftrightarrow s_C(Y) > 0$ for any $Y \in \mathcal{X}$ and $C \in \mathcal{C}$. A consequence is that in the clonal and haploid cases invasion implies substitution: if $s_X(Y) > 0$ then we know that X and Y cannot form a stable coalition C, since this would lead to the contradiction that $\phi(X) = \phi(C) = \phi(Y)$ while $\phi(X) < \phi(Y)$. Thus the outcome of a successful invasion can only be that the resident dies out and is replaced by the invading type. Therefore each successful mutant X increases $\phi(X)$, and hence any ESS attracts.

In Mendelian diploids the argument given for convergence to an ESS does not work, as invasion needs not imply substitution. Population dynamically stable coalitions may arise when a heterozygote mutant invades that also enjoys positive fitness in the environment set by a population of the corresponding homozygotes. This is usually excluded for smooth genotype to phenotype maps when only small mutational steps are allowed as then the genotype to phenotype map is locally additive and invasion fitness is necessarily smooth in the invader trait, and will usually be smooth in the resident traits away from population dynamical bifurcation points. Moreover, in the case of potentially larger mutational steps, under the IF assumption a further mutant may appear that realises the phenotype of the heterozygote in any genetic background. In that case, at least when the environment is constant and the organisms have but a single birth state, such an evolutionary stalemate will after a while be broken up again. It may be expected that this results in an eventual convergence to the ESS. The strongest expression of this conviction can be found in [23]. However, whether this is indeed the case without exception is not fully clear yet. See further [12, 22, 45, 15, 13]; note though that all these authors restrict themselves to non-fluctuating environments and single birth states.

3.2. Highest abstraction level: traits only

Let us now turn to the most general context in which a discussion of optimisation is meaningful. Thus we forget about the maps $E_{\text{attr}} : \mathcal{X} \to \mathcal{E}$ and $\rho : \mathcal{X} \times \mathcal{E} \to \mathbb{R}$ and simply suppose that $(X, Y) \mapsto s_X(Y)$ is a given invasion fitness function $\mathcal{X} \times \mathcal{X} \to \mathbb{R}$ on which we impose, a priori, only the condition $s_X(X) = 0$ for all $X \in \mathcal{X}$. The question then arises what conditions are necessary and sufficient for the existence of a function $\phi : \mathcal{X} \to \mathbb{R}$ such that

the conclusion

$$\phi(Y) < \phi(X) \Leftrightarrow s_X(Y) < 0 \quad \text{and} \quad \phi(Y) = \phi(X) \Leftrightarrow s_X(Y) = 0.$$

of Lemma 3.4 holds. For the purposes of this section we forget about any interpretation in terms of evolutionary outcomes and call any such function ϕ an optimisation principle for s. The question of characterising those s which admit an optimisation principle is addressed in [39] and [20] the results of which we now briefly discuss.

Clearly we are only interested in the sign of the invasion fitness function $s_X(Y)$. In the case of one dimensional traits it is customary to represent the sign of the invasion fitness function by means of a pairwise invasibility plot (PIP) as in Figure 3.1. If x and y (we denote here, as in the rest of the paper, real numbers by lowercase letters and general vectors by uppercase letters) are plotted on the customary axes, then points (x, y) where $s_x(y) > 0$ are coloured grey, points where $s_x(y) < 0$ white and the neutral boundaries $s_x(y) = 0$ black. Since a PIP contains all information about the sign of the invasion fitness function s, any conditions we impose on s should only depend on the corresponding PIP.

The first observation, already mentioned implicitly in Subsection 3.1, is the following:

Lemma 3.5 (Sign-antisymmetry). *If there exists an optimisation principle for s then*

$$s_X(Y) > 0 \Leftrightarrow s_Y(X) < 0.$$

In terms of PIPs this is the property of skew-symmetry: by flipping the diagram with respect to the line $x = y$ the positive grey regions are mapped to negative white ones and vice versa, while the neutral black lines are mapped onto themselves.

A second observation that is also a straightforward consequence of the definitions is:

Lemma 3.6 (Sign-transitivity). *If there exists an optimisation principle for s then*

$$s_X(Y) \geq 0 \text{ and } s_Y(Z) \geq 0 \quad \Rightarrow \quad s_X(Z) \geq 0.$$

The content of Lemma 3.6 can be reformulated using the familiar game of rock-scissors-paper.

Definition 3.7. An ordered triple $(X, Y, Z) \in \mathcal{X}^3$ is called a *rock-scissors-paper-cycle* if

$$s_X(Y) > 0, \quad s_Y(Z) > 0, \quad \text{and} \quad s_Z(X) > 0. \tag{3.2}$$

The triple (X, Y, Z) is called a *weak rock-scissors-paper-cycle* if it satisfies (3.2) with two of the three $>$ signs replaced by $\geq$ signs.

Under the additional assumption of sign-antisymmetry, sign-transitivity for s is exactly the statement that there are no weak rock-scissors-paper-cycles. The advantage of this viewpoint is that it emphasises that one can prove the

nonexistence of an optimisation principle by exhibiting just a single (weak) rock-scissors-paper-cycle.

We note that the simple proofs of Lemmas 3.5 and 3.6 are based on the properties of antisymmetry and transitivity enjoyed by the order relation of the real numbers. Also note that, for the purposes of the discussion on the existence of optimisation principles, we are only interested in the sign of the invasion fitness function. These observations motivate us, following [20], to consider directly the binary relation $\preceq_s$ on $\mathcal{X}$ defined as follows:

Definition 3.8. Given an invasion fitness function $s : \mathcal{X} \to \mathcal{X}$ define the weak invadability relation $\preceq_s$ by $X \preceq_s Y \Leftrightarrow s_X(Y) \geq 0$.

We note that our assumption $s_X(X) = 0$ implies that this relation is always reflexive, i.e., $X \preceq_s X$. The content of Lemma 3.6 is then that if s admits an optimisation principle then $\preceq_s$ must be a transitive relation. A binary relation $\sim$ is called total, if for any X, Y in the relevant domains at least one of the alternatives $X \sim Y$ or $Y \sim X$ holds. The relation $\preceq_s$ defined above is total if and only if there is no case of mutual exclusion $s_X(Y) < 0$, $s_Y(X) < 0$. A total and transitive binary relation is called a total preorder.

On an abstract level we now see that an optimisation principle can be considered a representation of the relation $\preceq_s$ by means of an order preserving map $\phi : \mathcal{X} \to \mathbb{R}$ so that $X \preceq_s X \Leftrightarrow \phi(X) \leq \phi(Y)$.

One of the theorems that led to the birth of order theory was Georg Cantor's theorem that a countable dense (between any two points lies a third) linear order without a largest or smallest element is isomorphic to the rational numbers as an ordered set. By extending and adapting the proof of this fact, it can be shown that the necessary conditions we have listed, along with the rather nonrestrictive requirements that the trait space be a separable metric space and the invasion fitness is at least separately continuous, are also sufficient for the existence of an optimisation principle [20]. More specifically, one has:

Theorem 3.9. *Let $\mathcal{X}$ be a trait space which is a separable metric space. Let $s : \mathcal{X} \times \mathcal{X} \to \mathbb{R}$ be a given invasion fitness function which is continuous in each variable separately. Then, if there exists no optimisation principle for s, at least one of the following alternatives holds:*

(i) *There is a pair of traits X, Y satisfying mutual exclusion $s_X(Y) < 0$, $s_Y(X) \leq 0$, or*
(ii) *there is a weak rock-scissors-paper-cycle.*

Note the minor technicality that for mutual exclusion we must allow one "$\leq$" sign just as we were forced to introduce weak rock-scissors-paper-cycles. If in stead we just ruled out $s_X(Y) < 0$, $s_Y(X) < 0$ then one could give the counterexample consisting of $\mathcal{X} = \{X, Y\}$ and $s_X(Y) = -1$, $s_Y(X) = 0$. Also note that protected coexistence $s_X(Y) > 0$, $s_Y(X) > 0$ is technically covered by (iii), since if X, Y enjoy protected coexistence, then (X, X, Y) is a weak rock-scissors-paper-cycle.

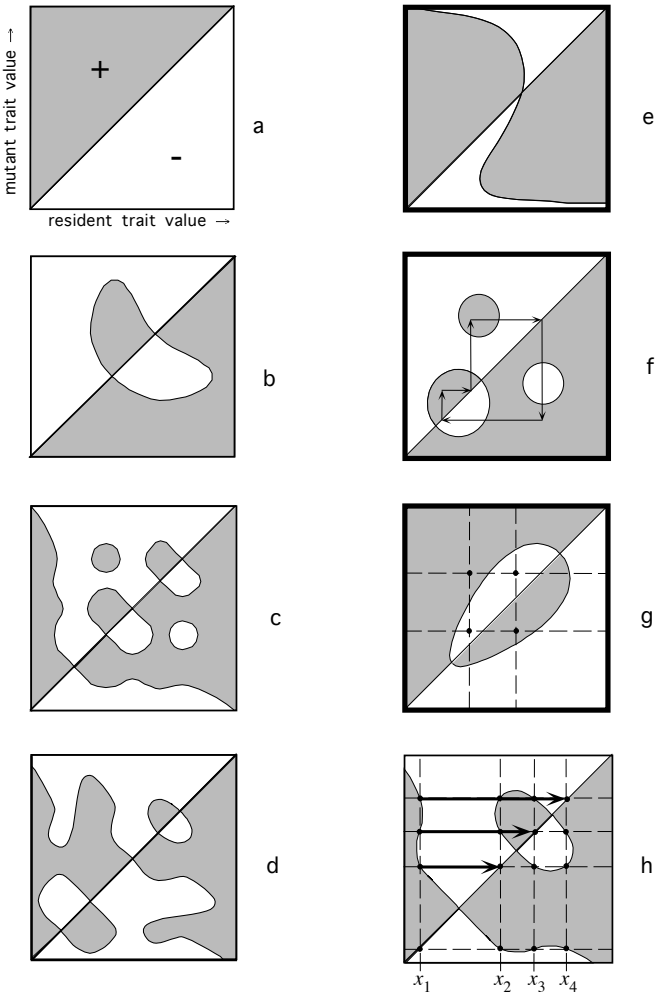

FIGURE 3.1. How the presence of an optimisation principle can be detected from PIPs. Panels (a) to (d) and (h) are examples of PIPs for models with an optimisation principle. Panel (e) is not skew symmetric and therefore there is no optimisation principle. In Panel (f) the presence of a rock-scissors paper trait triple is indicated by arrows. Panel (g) shows the 'opposite corners' obstruction to optimisation: an axis parallel rectangle with one pair of opposite corners in the positive region of the PIP while the other pair lies in the negative region. (You are encouraged to find yourself an opposite corners configuration in Panel (f) and a rock-scissors-paper triple in Panel (g).) Finally Panel (h) shows how the transitivity condition directly manifests itself in the PIP: above any resident trait value x_1 there is a certain alternation of plus and minus regions. If we read of the trait values that are selectively neutral relative to x_1 then these trait values should have exactly the same pattern of plus and minus regions above them. (Check for yourself that this condition is not fulfilled in Panels (e) to (g).)

We note now a second criterion for PIPs which follows from the order theoretic viewpoint. For any trait X one may consider the set $Inv(X)$ of trait types Y satisfying the weak invadability condition $Y \preceq_s X$. One can show easily that $\preceq_s$ is a total preorder if and only if the sets $Inv(X)$ (some of which may coincide) are totally ordered by inclusion. Given X and Y, if neither $Inv(X) \subseteq Inv(Y)$ nor vice versa, there must be elements X', Y' such that $X' \in Inv(X)$, $X' \notin Inv(Y)$, $Y' \notin Inv(X)$ and $Y' \in Inv(Y)$. In terms of PIPs this means that some four points (x, x'), (x, y'), (y, x'), (y, y') form the corners of an axis parallel rectangle such that one pair of opposite corners lies in the white region of the PIP while the other pair lies in the grey/black region; see Figure 3.1, Panel (g). Hence, a PIP accords with the existence of an optimisation principle if and only if it is skew symmetric and there exist no such 'opposite corner' configurations.

Theorem 3.9 has a useful corollary. We begin with a preliminary one-dimensional formulation:

Corollary 3.10. *Suppose the trait space is an interval I on the real line and suppose $s : I \times I \to \mathbb{R}$ is separately continuous in the resident and invader traits. Then there is an optimisation principle for s if and only if the following two conditions hold for all $x, y \in I$:*

(i) $\operatorname{sign}(s_x(y)) = -\operatorname{sign}(s_y(x))$,
(ii) *if* $s_x(y) = 0$ *then* $\operatorname{sign}(s_x(z)) = \operatorname{sign}(s_y(z))$ *for all* z.

Proof. The necessity of the two conditions given is clear: the first condition is just a restatement of the conclusion of Lemma 3.5 while the failure of the second implies the presence of a weak rock-scissors-paper-cycle.

To prove the sufficiency of the stated conditions, we show that the presence of a weak rock-scissors-paper triple leads to a contradiction under the assumptions (i) and (ii). First note that the presence of "=" in the weak rock-scissors-paper inequalities immediately implies a contradiction to (i)-(ii).

We are left to consider the following case: $s_x(y) > 0, s_y(z) > 0, s_z(x) > 0$. For definiteness we assume that on the real line one has $x < y < z$, the other cases being similar. Let $x' = \sup\{t < y : s_x(t) = 0\}$. By continuity $s_x(x') = 0$. Now using (i) and (ii) one sees that $s_{x'}(z) < 0$. Thus by Rolle's theorem one has $s_{z'}(z) = 0$ for some $x' < z' < y$. Similar reasoning shows that $s_x(x'') = 0$ for some $x' < z' < x'' < y$, which contradicts the definition of x'. □

In terms of PIPs the conditions mentioned in Corollary 3.10 mean that whenever (x, y) lies on the black neutral line $s_x(y) = 0$, and one draws vertical lines passing through the points x and y on the horizontal axis, the patterns of white and grey on these lines are identical; see Figure 3.1, Panel (h). Of course, due to skew symmetry, one observes the same pattern in corresponding horizontal lines. Together these conditions are thus necessary and sufficient for the existence of an optimisation principle.

The next Corollary generalises 3.10 to higher dimensional trait spaces.

Corollary 3.11. *Suppose $\mathcal{X}$ is a path connected trait space which is separable and metrisable and the invasion fitness function s is separately continuous in the resident and invader traits. Then the following two conditions for all $X, Y \in \mathcal{X}$ are together necessary and sufficient for the existence of an optimisation principle:*

(i) $\operatorname{sign}(s_X(Y)) = -\operatorname{sign}(s_Y(X))$,
(ii) *if* $s_X(Y) = 0$ *then* $\operatorname{sign}(s_X(Z)) = \operatorname{sign}(s_Y(Z))$ *for all* $Z \in \mathcal{X}$.

Proof. It is enough to show that under condition (i) the presence of a weak rock-scissors-paper-cycle leads to a violation of condition (ii). Thus suppose condition (i) holds and that there is a weak rock-scissors-paper-cycle in $\mathcal{X}$. By path connectedness one can find a path $\gamma : I \to \mathcal{X}$ parametrised by an interval $I \subset \mathbb{R}$ such that the cycle is contained in the image γI. Applying Corollary 3.10 to the trait space I equipped with the invasion fitness function $(x, y) \mapsto s_{\gamma(x)}(\gamma(y))$ we find $x, y, z \in I$ such that $s_{\gamma(x)}(\gamma(y)) = 0$, but $\operatorname{sign}(s_{\gamma(x)}(\gamma(z))) \neq \operatorname{sign}(s_{\gamma(y)}(\gamma(z)))$, so (ii) fails for $X = \gamma(x)$ and $Y = \gamma(y)$. □

The following derived criterion will prove useful in Section 4, Application 4.3.

Corollary 3.12. *If there is an optimisation principle and $s_X(Y) = 0$, then the sets $\{Z | s_X(Z) = 0\}$ and $\{Z | s_Y(Z) = 0\}$ are equal.*

As a final point we note that when the trait space is multidimensional, the presence of an optimisation principle implies that all of the information necessary for deducing evolutionary outcomes is captured by a one-dimensional quantity. Even for a one dimensional trait, s has to satisfy very strict requirements. From a mathematical viewpoint such a situation is highly nongeneric. Yet, the practical fact is that optimisation models are frequently encountered in the literature, in textbooks in particular. The cause is no doubt our penchant for making simplifications to ease the math, but one should not be misled into believing that optimisation is the default scenario in the real world.

3.3. Medium abstraction level: traits and environments

Delving deeper into the determining factors for having an optimisation principle we consider as first step in the mechanistic direction the separate functions $(Y, E) \mapsto \rho(Y|E)$ and $X \mapsto E_{\text{attr}}(X)$. This problem was first investigated for the clonal and haploid cases in [38]. [39] covers also the diploid case under the same assumptions as made in this chapter. Below we give a summary of the results. Proofs can be found in the references.

The first result in [39, 38] may not come entirely unexpected given the results from Subsection 3.2. An eco-evolutionary model has an optimisation principle if and only if "the trait values affect fitness effectively in a one-dimensional monotone manner". The term "effectively" here means that the specified properties only need to pertain to the range of fitness values closely surrounding the change from negative to positive. More precisely,

Proposition 3.13. *An eco-evolutionary model has an optimisation principle if and only if*

A. *there exists a function* $\phi : \mathcal{X} \to \mathbb{R}$ *and a function* $g : \mathbb{R} \times \mathcal{E} \to \mathbb{R}$*, increasing in its first argument, such that*

$$\text{sign } \rho(X|E) = \text{sign } g(\phi(X), E). \tag{3.3}$$

In that case ϕ, or any increasing function of ϕ, is an optimisation principle.

A perhaps more surprising result is

Proposition 3.14. *Condition* **A** *is equivalent to*

B. *there exists a function* $\psi : \mathcal{E} \to \mathbb{R}$ *and a function* $h : \mathcal{X} \times \mathbb{R} \to \mathbb{R}$*, increasing in its second argument, such that*

$$\text{sign } \rho(X|E) = \text{sign } h(X|\psi(E),) \tag{3.4}$$

which can be paraphrased as "the environment acts effectively in a one-dimensional monotone manner".

Propositions 3.13 and 3.14 show once again that optimisation principles, although frequently encountered in the literature, are exceptions rather than the rule.

Definition 3.15. We shall call a function $\psi : \mathcal{E} \to \mathbb{R}$ as in condition **B** a pessimisation principle.

Conditions **A** and **B** are related to each other by

Proposition 3.16. *If an optimisation principle, or equivalently a pessimisation principle, exists, it is possible to choose the functions* ϕ *and* ψ *such that*

$$\text{sign } \rho(X|E) = \text{sign } (\phi(X) + \psi(E)), \tag{3.5}$$

where ϕ *and* ψ *are connected through the relation*

$$\phi(X) = -\psi(E_{\text{attr}}(X)). \tag{3.6}$$

(Optimisation and pessimisation principles are only uniquely determined up to increasing transformations.) Hence, given a pessimisation principle ψ it is possible to construct a matching optimisation principle ϕ via the construction (3.6) and vice versa.

Corollary 3.17. *When a pessimisation principle* ψ *exists, evolution minimises* $\psi(E_{\text{attr}}(X))$ *under any constraint on* X*.*

Better still, $\psi(E_{\text{attr}}))$ decreases with each increase in its matching $\phi(X)$. Moreover, fitness increases with ψ where it counts, i.e., around zero. Hence the choice of the term "pessimisation principle". When a pessimisation principle exists, in the end the worst attainable world remains, together with the type(s) that can just cope with it. The following example may give a more concrete feel for the issue.

Example. The textbook scenario for so-called r-selection Consider a structured population in continuous time regulated through an additional death rate d_{E} which is the same for all states of an individual and with all other demographic parameters independent of E. Then minus the mean death rate, $-\langle d_{\mathrm{E}}(E(t))\rangle_{\mathrm{time}}$, associated with an environment provides a pessimization principle (i.e., evolution maximises the mean death rate), with the asymptotic relative growth rate ρ_0 calculated on the assumption that $d_{\mathrm{E}} = 0$ as matching optimization principle. A special case is where the environment is constant except for occasional instantaneous decimating catastrophes, provided the latter kill totally indiscriminately (so that ρ_0 equals the intrinsic rate of population increase or Malthusian parameter r for that constant environment). But for the (essential, but generally unmentioned) indiscriminateness, this is the condition touted in the textbooks as supporting r-maximisation.

The practical importance of Proposition 3.16 is that, while condition **A** is close to trivial, the equivalent condition **B** and relation (3.6) often provide a useful tool for either deriving optimisation principles or proving the non-existence of such principles for large families of eco-evolutionary models.

The two optimisation principles most frequently touted in the evolutionary ecology literature are the intrinsic rate of population increase r and the lifetime offspring number R_0. The results discussed above can be used to characterise the ecological scenarios for which evolution "just maximises" r or R_0. Here "just maximising a function of X and E" should be interpreted as maximising that function by varying X for an unspecified choice of E (the latter as reflection of the absence of any mention of E in the usual statements in the non-epidemiological literature).

Remark. A convention of logic is that when a statement is not explicitly indicated as pertaining to a specific individual case, or subset of cases, it should be interpreted as pertaining to all possible cases. This convention is itself but a formalisation of the human habit of interpreting open statements like "raven are black" as meaning that all raven are black and not as some raven being black, or raven being black only under certain circumstances.

Under the presupposition that the community dynamics engenders constant environments so that the Malthusian parameter r and the lifetime offspring number R_0 are well defined, the following results hold good.

Proposition 3.18. *Evolution just maximises r if and only if*

C. *the combination of life histories and ecological embedding is such that r can be written as*

$$r(X|E) = g\left(r(X|E_0), E\right)$$

for some function g that increases in its first argument, and E_0 some fixed, but otherwise arbitrary, environment,

Proposition 3.19. *Evolution just maximises R_0 if and only if*

D. *the combination of life histories and ecological embedding is such that* $\ln(R_0)$ *can be written as*

$$\ln\left(R_0(X|E)\right) = g\left(\ln\left(R_0(X|E_0)\right), E\right)$$

for some function g *that increases in its first argument, and* E_0 *some fixed, but otherwise arbitrary, environment.*

In contrast to criteria **A** and **B**, criteria **C** and **D** are relatively easy to check in specific situations.

A fair fraction of textbook statements, if taken literally, applies only when condition **C** or **D** is fulfilled.

3.4. Lowest abstraction level: life histories

The next step in the mechanistic direction is to consider how invasion fitness ρ is built up from demographic parameters (in an evolutionary context usually referred to as life-history traits), and what conditions on those parameters correspond to the conditions that were found in Subsection 3.3.

Presently two manuscripts are floating around, by Roger Bowers [5] and by Claus Rueffler and co-workers [43], that relate life cycle structure to properties of the associated invasion fitness function. Both manuscripts deal with finite state individuals in constant environments, i.e., community dynamical equilibria. The first one considers continuous time community models with all phenotypes within a species influencing the environment in the same manner. The dependence of the demographic parameters is considered to be essentially affine in the community composition, in a manner that depends on the, bivariate, traits. The second one considers discrete time population models with separable demographic parameters, that is, parameters that can each be decomposed into an inherited parameter times possibly a scalar function of E, referred to as regulatory function and denoted as R, without any further a priori assumptions on how E is determined. Some of the inherited demographic parameters are supposed to be under evolutionary control and then are called traits. The environment is supposed to be organised as the cartesian product of one-dimesional components, and the regulatory functions are supposed to be monotone in the order relation imposed by the positive cone. We only review the material that pertains to the existence or non-existence of optimisation principles.

We start with the plethora of sufficient conditions for the existence of an optimisation principle derived by Rueffler et al. [43]. Two immediate trivial cases are

Proposition 3.20. *If there exists a scalar function* $\psi : \mathcal{E} \to \mathbb{R}$ *such that all regulatory functions can be written as monotonically increasing functions of* $\psi(E)$*, then evolution maximises* $-\psi(E_{\mathrm{attr}}(X))$.

Proposition 3.21. *If all demographic parameters come with the same* R*, then evolution minimises* $R(E_{\mathrm{attr}}(X))$ *and maximises the dominant eigenvalue of the population projection matrix for* R *set equal to some arbitrary fixed value.*

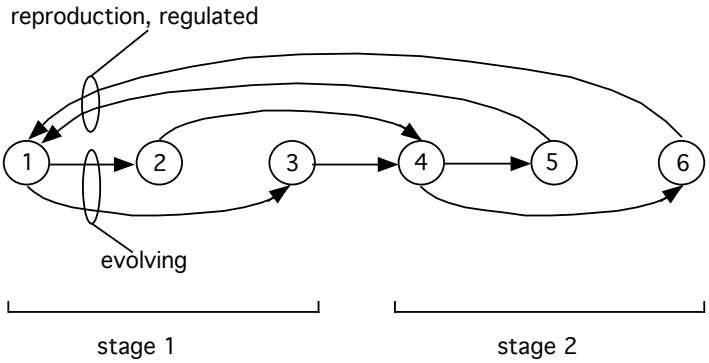

FIGURE 3.2. Life cycle that satisfies the conditions of Proposition 3.22 (and also condition 1 of Proposition 3.25).

An example is when the only influence of the environment is through a death probability that is independent of the state or type of an individual on top of any state- or type-dependent ones. In that case evolution maximises that death probability and the value of r for any given fixed value of that probability.

In the next Proposition we use the term *transition ratio* to cover both the state-transition-and-survival probabilities and the fertilities, and refer to both types of event together as *'transitions'*.

Proposition 3.22. *If*

(i) *the states can be partitioned into n disjunct classes G_i, $i \in \mathbb{N} \bmod n$, n even, with each class only connecting to the following class through a single state that may be an element of either class,*
with the transition ratios from states in odd numbered classes not being regulated and the transition ratios from states in the even numbered classes not evolving (the unregulated *resp.* non-evolving classes*), and*

(ii) *the transition ratios from the classes that can only be left from a single state satisfy the following restrictions*
for non-evolving classes: the transition ratios to the next class are all regulated in the same way, and
for unregulated classes: if the class does not connect to the next class only through single states in both classes, the transition ratios to the next class are non-evolving,

then there is an optimisation principle.

This optimisation principle can be calculated by treating the entrance stream into any of the classes as "births" for which we calculate R_0. This R_0 (i) is a fitness proxy and (ii) can be written as a product of a function of the traits times a function of E. The result then follows from Proposition 3.19.

Remark. Without further restrictions on the models described in Propostion 3.22 the R_0 referred to above may be infinite. This happens as soon as one

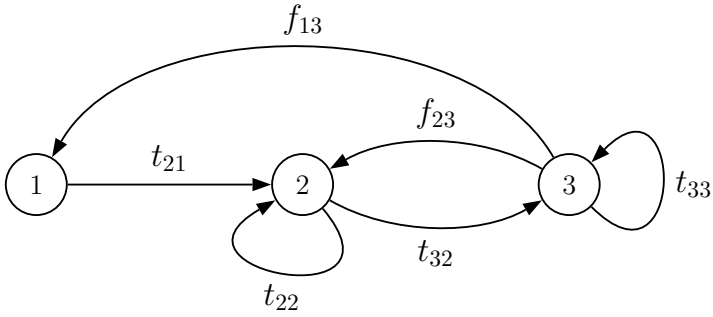

FIGURE 3.3. Life cycle of a perennial plant with a seedling (1), juvenile (2) and flowering (3) state. Q for this life cycle equals $-1 + (t_{22} + t_{33} + t_{21}t_{32}f_{13} + t_{32}f_{23}) - (t_{22}t_{33})$, with the bracketed terms corresponding to the first and second sums in (3.7). From [43].

of the classes if disconnected from the rest of the life cycle has a nonnegative growth rate. However, such a biological anomaly is implicitly excluded as this would lead to a contradiction with the assumption that the models allow a community dynamical equilibrium including the species under consideration.

Models of this type frequently occur in the literature. For examples see Figure 3.2 and Application 4.2.

The remainder of the results of Rueffler and coworkers are based on the fitness proxy Q described at the end of Subsection 2.1. The advantage of Q over r or R_0 is that Q is affine in each of the separate heritable demographic parameters (baseline state transition cum survival probabilities t_{ij} and fertilities times survival probabilites f_{ij}) and regulatory functions ($R_{\mathrm{t},ij}$ and $R_{\mathrm{f},ij}$, supposedly lying between zero and one), together collected in the demographic projection matrix $\mathbf{A}$. More in particular, it is possible to write

$$Q = -1 + \sum_{\mathcal{L}_\mathbf{A}} L - \sum_{\mathcal{L}_\mathbf{A}^{2*}} LM + \sum_{\mathcal{L}_\mathbf{A}^{3*}} LMN - \dots \,, \tag{3.7}$$

with (i) L, M and N so-called loop transmissions, where a loop is a sequence of demographic parameters that lead from a given state to itself without passing more than once through some other state, with the loop transmission the product of the demographic parameters along the loop, (ii) $\mathcal{L}_\mathbf{A}$ the set of all loops associated with $\mathbf{A}$, and (iii) a $*$ hung on to an n-fold cartesian power of $\mathcal{L}_\mathbf{A}$ indicating that only n-tuples are considered in which the sets of states occurring in those loops are mutually exclusive (c.f. Figure 3.3). A loop is called evolving if it contains at least one trait, and regulated if it contains at least one demographic parameter affected by E.

Rueffler and co-workers use (3.7) together with

Proposition 3.23. *If it is possible to find functions* $g_1 : \mathcal{X} \to \mathbb{R}$, $g_2 : \mathcal{X} \to \mathbb{R}$, $e_1 : \mathcal{E} \to \mathbb{R}$ *and* $e_2 : \mathcal{E} \to \mathbb{R}$ *with* $\operatorname{sign}[g_2]$ *and* $\operatorname{sign}[e_1]$ *constant such that*

$$Q(X|E) = g_1(X)e_1(E) + g_2(X)e_2(E), \tag{3.8}$$

then $\operatorname{sign}[e_1(E)]g_1(X)/|g_2(X)|$ *is an optimisation principle.*

to delineate a zoo of special classes of sufficient conditions for the existence of an optimisation principle in terms of the life cycle graph. The taxonomy of this zoo is not simple. Moreover, it is not clear yet whether possibly any further special cases are still out in the wild. Hence we give only one simple example and refer to [43] for the details.

Example. Evolution of fertility patterns in perennial plants. Assume that we are interested in the evolution in the two fertility parameters in the life-cycle graph in Figure 3.3. The biological interpretation is that f_{13} corresponds to fall reproduction, and f_{23} to spring reproduction so that the seeds can already germinate in the same year and appear as a juvenile at the next sampling time. Now assume that the environment influences only the winter seed survival, and by that f_{31}, and seedling survival t_{21}. Then we get, with a bar above a life history parameter indicating that it is constant and a tilde that it evolves,

$$Q(\tilde{f}_{13}, \tilde{f}_{12}|E) = \\ [\bar{t}_{32}\tilde{f}_{23} + \bar{t}_{22} + \bar{t}_{33} - \bar{t}_{22}\bar{t}_{33} - 1] + [\bar{t}_{21}\bar{t}_{32}\tilde{f}_{13}][R_{\mathrm{t},21}(E)R_{\mathrm{f},13}(E)].$$

Hence evolution maximises $(\bar{t}_{21}\bar{t}_{32}\tilde{f}_{13})/(1 + \bar{t}_{22}\bar{t}_{33} - \bar{t}_{22} - \bar{t}_{33} - \bar{t}_{32}\tilde{f}_{23})$. (The term $(\bar{t}_{32}\tilde{f}_{23} + \bar{t}_{22} + \bar{t}_{33} - \bar{t}_{22}\bar{t}_{33} - 1)$ is negative since otherwise the population would not have an equilibrium.)

The problem of determining sufficient conditions for the existence of an optimisation principle can also be turned on its head in the form of deriving necessary conditions for the non-existence of an optimisation principle, or, in the terminology of [25], for selection to be frequency dependent. We start with a condition presented by Roger Bowers [5], although phrased in a different language.

Proposition 3.24. *Let the community be described by*

$$\frac{\mathrm{d}N}{\mathrm{d}t} = \mathbf{M}(X|N) \tag{3.9}$$

with N *the vector of population densities of all species in the community differentiated according to the states of the individuals,* $\mathbf{M}$ *a corresponding block diagonal matrix, and the map* $N \mapsto \mathbf{M}(X|N)$ *affine. Then a necessary condition for evolution to be frequency dependent is that for each* X *the range of the map* $N \mapsto \mathbf{M}(X|N)$ *is at least two dimensional.*

Claus Rueffler and co-workers strengthen this type of condition to

Proposition 3.25. *For selection to be frequency dependent it is necessary that* E *has more than one component and that at least two evolving and two regulated loops exist which occur in one of the following combinations:*

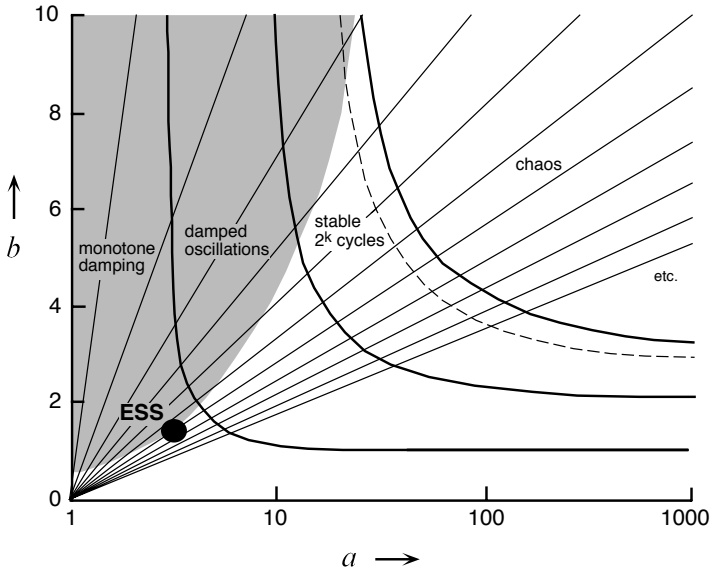

FIGURE 4.1. Types of dynamics of the monomorphic version of the model (4.1)-(4.3). The thin lines are contour lines of ϕ. A reasonable constraint set is indicated in grey together with the accompanying ESS.

(i) *A pair of loops L, M exist that are both evolving and regulated such that*

$$Q(X|E) = L(X|E) + M(X|E) + \text{remainder}. \tag{3.10}$$

(ii) *Three loops L, M, N exist where L is both evolving and regulated, M is evolving and N is regulated such that*

$$Q(X|E) = L(X|E) + M(X) + N(E) + \text{remainder}. \tag{3.11}$$

(iii) *Four loops L, M, N and O exist where L and M are evolving and N and O are regulated and where L and N are unconnected such that*

$$Q(X|E) = L(X) + M(X) + N(E) + O(E) - L(X)N(E) + \text{remainder}, \tag{3.12}$$

where X, E and $(X|E)$ are added as arguments to loops to indicate whether they contain a trait, a regulated demographic parameter or both.

The goal is, of course, to delimit the existence versus non-existence of an optimisation principle in the form of necessary and sufficient conditions. However, Figure 3.2 depicts a life cycle that satisfies requirement 1 of Proposition 3.25 as well as the requirement of Proposition 3.22, showing that that goal is not yet in sight.

4. Three applications

Application 4.1. *Evolution away from chaos*

Consider the population dynamical equations

$$n_i(t+1) = a_i \left(f\left(E(t)\right)\right)^{b_i} n_i(t) \quad \text{for } i = 0, \ldots, k, \tag{4.1}$$

with

$$E(t) = [c_0 n_0(t) + \cdots + c_k n_k(t)], \tag{4.2}$$

all a_i, b_i, and $c_i > 0$, and f decreasing from 1 to 0 for E increasing from 0 to ∞.

With the choice

$$f(E(t)) = [1 + E(t)]^{-1}, \tag{4.3}$$

and $k = 0$, this model becomes the model launched into fashion by i.a. [24] as a touchstone for the appearance of chaotic fluctuations in single species population dynamics. Figure 4.1 shows the dependence of the dynamics on the parameter values.

The trait vector appearing in (4.1) and (4.2) is

$$X = (a, b, c). \tag{4.4}$$

The parameters a, $1/b$, and c can be interpreted in individual-based terms as respectively the per capita reproduction in a boom environment, the ability to cope with a bust environment and the per capita impingement on the common environment. From (4.1) we find

$$\rho(X|E) = L[a[f(E)]^b] = \ln[a] + b\psi(E), \tag{4.5}$$

with

$$\psi(E) = L[f(E)]$$

and L the log geometric mean operator

$$L(z) \;:=\; \lim_{T\to\infty} T^{-1} \sum_{t=1}^{T} \ln(z(t)).$$

From $\rho(X|E_{\text{attr}}(X)) = 0$ we deduce that

$$\psi(E_{\text{attr}}(X)) = -b^{-1} \ln[a], \tag{4.6}$$

From Proposition 3.16 we conclude that evolution maximises

$$\phi(X) \;:=\; \frac{\ln[a]}{b} \tag{4.7}$$

In accordance with Propositions 3.13 and 3.14 we can define the functions g and h occurring in the definitions of monotone one-dimensional action as

$$g(\phi(X), E) := \phi(X) + \psi(E) =: h(X, \psi(E)). \tag{4.8}$$

Note that

$$\rho(X, E) = b(\phi(X) + \psi(E)) \tag{4.9}$$

showing that g and h are only sign equivalent to ρ, but not equal to ρ. It can even be proved that for ρ given by (4.5) it is impossible to find pairs g and ϕ, or h and ψ, for which such an equality holds good. Hence, even in the domain of stable equilibria neither of the conditions **C** or **D** is fulfilled.

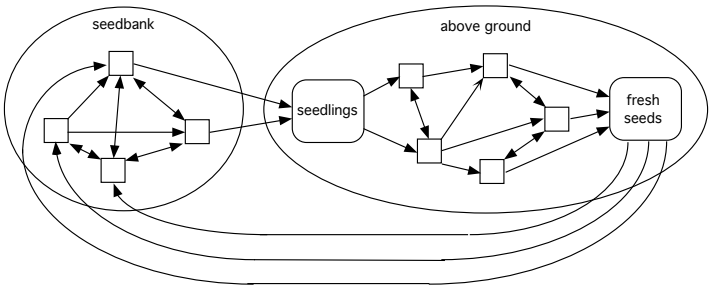

FIGURE 4.2. Life cycle of a perennial plant.

It may be expected that increasing a will in general go at the cost of increasing b. By combining the optimisation principle with a reasonable trade-off between a and b it is found that in general the ESS will lie in the region of stable equilibria (see Figure 4.1).

Application 4.2. *Evolution of germination strategies*
Figure 4.2 depicts the life history of a plant decomposed into two stages between which no information is transferred, as all seedlings are equal and so are all newly produced seeds. Within the two stages the seeds and plants are differentiated in e.g. (age, depth in soil)- respectively (age, size above ground, size below ground)-classes. Sampling is done on a yearly basis, just before germination time. Hence the boxes "seedling" and "fresh seeds" do not correspond to states of the model as those conditions are but ephemeral on the considered time scale. Yet we put them in since they provide the unique connections between the stages that by that uniqueness prevent information being transferred from one stage to another. The environmental influences on an average individual plant can be decomposed into two components $(E_\mathrm{a}, E_\mathrm{b})$, with E_b representing the influences on the seeds (consisting of e.g. seed predation pressures and fungal and bacterial attack rates at different depths; the 'b' refers to below ground), and with E_a representing influences on the plants (capturing all direct and indirect competitive influences within the community through shading, nutrient depletion and changing predation pressures; the 'a' refers to above ground). Seedlings can survive only in so-called safe sites, places that are temporarily without ground cover. Since the birth of a safe site necessarily coincides with the demise or a state change of one or more plants in the community it may be assumed that E_a also determines the fraction of the area covered by safe sites. The quantity of evolutionary interest is the germination strategy. Especially deeper buried seeds have only a partial knowledge of whether they are in a safe site. Hence we can capture the germination strategy by the dependence of the germination probability on a seed's state and whether it is in a safe site. Therefore, for a full description of the eco-evolutionary model the following quantities and functions are needed, with a $\check{}$ indicating the mutant:

N, $\check{N}$: vector of densities of seeds in different states,
P, $\check{P}$: vector of densities of plants in different states,
$U(E_\mathrm{b})$: state distribution of a new seed just prior to germination time,
$\mathbf{S}(E_\mathrm{b})$: matrix of survival and state transition probabilities of seeds,
$h(E_\mathrm{a})$: density of safe sites available at germination time,
$k(E_\mathrm{a})$: fraction of total area covered by those safe sites,
G, $\check{G}$: state dependent probabilities that a seed in a safe site germinates,
F, $\check{F}$: state dependent probabilities that a seed outside a safe site germinates,
$\theta(E_\mathrm{a})$: average number of seedlings in a safe site that survive seedling competition divided by the average density of novel seedlings in safe sites,
$J(E_\mathrm{a})$: state distribution of young plants that have survived seedling competition,
$\mathbf{A}(E_\mathrm{a})$: matrix of survival and state transition probabilities of plants,
$Y(E_\mathrm{a})$: seed production by plants in different plant states.

Note that the probability distribution of the state of a newborn seed at the next germination time, encoded in the vector U, will in general be defective (i.e., has total mass smaller than one) due to seed mortality. Note also that in most concrete instances the probability distribution of plant states after seedling competition, encoded in the vector J, will probably be concentrated on but a single plant state: small juvenile. J by definition has full mass as the probabilities of seedling death are all accounted for in $\theta(E_\mathrm{a})$.

The resident population state satisfies the following recurrences

$$\begin{aligned} N' &= \mathbf{S}(E_\mathrm{b})(\mathbf{I} - k(E_\mathrm{a})\mathbf{diag}(G) - (1 - k(E_\mathrm{a}))\mathbf{diag}(F))N \\ &\quad + U(E_\mathrm{b})Y^\mathrm{T}(E_\mathrm{a})P, \\ P' &= \mathbf{A}(E_\mathrm{a})P + J(E_\mathrm{a})h(E_\mathrm{a})\theta(E_\mathrm{a})G^\mathrm{T}N, \end{aligned} \tag{4.10}$$

In words, the seeds of next year consist of this year's seeds that neither germinate nor succumb plus the surviving new seeds from this year. The plants of next year consist of this year's surviving plants plus the new plants, the density of which is calculated as the density of safe sites times this year's average numbers of survivors of seedling competition in a site. By the same token, the mutant population state satisfies

$$\begin{aligned} \check{N}' &= \mathbf{S}(E_\mathrm{b})(\mathbf{I} - k(E_\mathrm{a})\mathbf{diag}(\check{G}) - (1 - k(E_\mathrm{a}))\mathbf{diag}(\check{F}))\check{N} \\ &\quad + U(E_\mathrm{b})Y^\mathrm{T}(E_\mathrm{a})\check{P}, \\ \check{P}' &= \mathbf{A}(E_\mathrm{a})\check{P} + J(E_\mathrm{a})h(E_\mathrm{a})\theta(E_\mathrm{a})\check{G}\mathrm{T}\check{N}. \end{aligned} \tag{4.11}$$

These equations should be combined with equations for the remainder of the community to determine $(E_\mathrm{a}, E_\mathrm{b})$.

For the calculations below we assume that the resident population dynamics converges to an equilibrium.

As it turns out, the present model is still a bit too general to allow the ES germination strategy to be determined from an optimisation principle. However, it is only by considering more general models that it is possible to delineate the crucial assumptions needed for the results from the previous Section to apply.

At the resident equilibrium the average lifetime offspring number of a resident equals 1. The calculation of this average lifetime offspring number can be broken down into a number of steps. First we calculate the average number of full seasonal cycles (measured between end-of-seedling-competition time points) that a survivor from the seedling stage lives through during its lifetime, split up according to the state the plant was in at the end-of-seedling-competition moments. From the general Markov chain results in [28] it follows that these numbers are given by the vector $(\mathbf{I} - \mathbf{A}(E_{\mathrm{a},G,F}))^{-1} J(E_{\mathrm{a},G,F})$, with $(E_{\mathrm{a}}, E_{\mathrm{b}})_{G,F}$ the equilibrium environment, to be determined from the full community dynamical equations for the resident strategy (G, F). Hence, the average number of seeds that a plant that just germinated in a safe site will produce over its lifetime is $Y^{\mathrm{T}}(E_{\mathrm{a},G,F})(\mathbf{I} - \mathbf{A}(E_{\mathrm{a},G,F}))^{-1} J(E_{\mathrm{a},G,F})\theta(E_{\mathrm{a},G,F})$. Similarly, the average number of germination moments that a seed experiences while in various seed states equals $(\mathbf{I}-\mathbf{S}(E_{\mathrm{b},G,F})(\mathbf{I}-k(E_{\mathrm{a},G,F})\mathbf{diag}(G)-(1-k(E_{\mathrm{a},G,F}))\mathbf{diag}(F)))^{-1} U(E_{\mathrm{b},G,F})$. Therefore, the probability of a seed germinating in a safe site instead of dying or germinating elsewhere equals $G^{\mathrm{T}}(\mathbf{I} - \mathbf{S}(E_{\mathrm{b},G,F})(\mathbf{I} - k(E_{\mathrm{a},G,F})\mathbf{diag}(G) - (1 - k(E_{\mathrm{a},G,F}))\mathbf{diag}(F)))^{-1} U(E_{\mathrm{b},G,F})$. Multiplying these two numbers gives an expression for $R_0(G, F|(E_a, E_b)_{G,F})$ that we have to set equal to 1 as part of the process of calculating the resident equilibrium. As it turns out, there is no need to calculate this equilibrium in full. All that is needed later is an expression for $\theta(E_{a,G,F})$ as a function of the other resident parameters which can be determined from the equation $R_0 = 1$. The calculation of $R_0(\check{G}, \check{F}|(E_a, E_b)_{G,F})$ proceeds in a similar manner. After substituting the earlier found expression for $\theta(E_{a,G,F})$ and cancelling terms in the numerator and denominator we get

$$R_0(\check{G}, \check{F}|(E_{\mathrm{a}}, E_{\mathrm{b}})_{G,F}) \tag{4.12}$$
$$= \frac{\check{G}^{\mathrm{T}}\left(\mathbf{I}-\mathbf{S}(E_{\mathrm{b},G,F})\left(\mathbf{I}-k\left(E_{\mathrm{a},G,F}\right)\mathbf{diag}(\check{G})-\left(1-k\left(E_{\mathrm{a},G,F}\right)\right)\mathbf{diag}(\check{F})\right)\right)^{-1} U\left(E_{\mathrm{b},G,F}\right)}{G^{\mathrm{T}}\left(\mathbf{I}-\mathbf{S}\left(E_{\mathrm{b},G,F}\right)\left(\mathbf{I}-k\left(E_{\mathrm{a},G,F}\right)\mathbf{diag}(G)-\left(1-k\left(E_{\mathrm{a},G,F}\right)\right)\mathbf{diag}(F)\right)\right)^{-1} U\left(E_{\mathrm{b},G,F}\right)}.$$

From this expression it can be seen that ESSes can be determined by optimising (G, F) in

$$\tilde{\phi}(G, F; k, \mathbf{S}, U) := G^{\mathrm{T}} \left(\mathbf{I} - \mathbf{S}\left(\mathbf{I} - k\mathbf{diag}(G) - (1 - k)\mathbf{diag}(F)\right)\right)^{-1} U \tag{4.13}$$

in dependence on $(k, \mathbf{S}, U)$, and solving the community dynamical equilibrium equations together with

$$(G, F) = (G, F)_{\mathrm{opt}}(k(E_{\mathrm{a},G,F}), \mathbf{S}(E_{\mathrm{b},G,F}), U(E_{\mathrm{b},G,F})). \tag{4.14}$$

If and only if G and F do not influence the equilibrium values of the seed state transition and survival probabilities and the fraction of the area covered by safe sites, i.e., $\mathbf{S}(E_{\mathrm{b},G,F}) = \bar{\mathbf{S}}$, $U(E_{\mathrm{b},G,F}) = \bar{U}$ and $k(E_{\mathrm{a},G,F}) = \bar{k}$,

the function $\phi : (G, F) \mapsto \tilde{\phi}(G, F; \bar{k}, \bar{\mathbf{S}}, \bar{U})$ is an evolutionary optimisation principle, in accordance with Proposition 3.22.

We are still in the midst of exploring this model. Some first results and more details can be found in [36].

Application 4.3. *Virulence evolution*
For a long time, it was close to dogma in epidemiological theorizing (e.g., [1, 2]) that the main basis for the study of virulence evolution should be sought in the maximisation of R_0, defined in epidemiology as the number of secondary infections engendered by a primary infection *in an otherwise infection-free population.* To this end, R_0 is considered as a function of the disease's demographic parameters, which in turn are envisaged as functions of some underlying trait vector that is supposed to be under evolutionary control. Here we consider, following [8], how this dogma fares in the light of Propositions 3.13 and 3.14 (see also [7]).

The epidemiological models that we consider below have been chosen for the simplicity of the calculations they engender. In particular, their community dynamics possess unique internal point attractors.

We start by giving a full population dynamical description of the ecological context, before reverting to considerations focusing on infected individuals. It is the individual-based dynamics of the latter that provides the basis for the classification of the environmental feed-back loop relative to its consequences for the ESSs of disease traits. The details of the population dynamics surrounding infected individuals is relevant only in so far as it acts as an environment affecting the population dynamical behavior of the infected individuals.

To characterise the instantaneous environmental conditions to which infected individuals may be exposed, we follow standard custom by letting S denote the density of susceptible individuals and I the density of infected individuals. After specifying the dynamics of this instantaneous environment, the corresponding evolutionary environments can be calculated from the attractors of this dynamics. Infections occur according to the law of mass action, with a fixed rate constant β. Infected individuals do not recover but die at a per capita rate α, acting on top of the per capita death rate experienced by susceptible and infected individuals alike. In the absence of the disease, $I = 0$, the population grows in a density-dependent manner, with per capita birth rate $b_0 - h_\mathrm{b}(S, 0)$ and per capita death rate $d_0 + h_\mathrm{d}(S, 0)$, with $b_0 > d_0 > 0$. The functions h_b and h_d both increase in S and I, with $h_\mathrm{b}(0, 0) = h_\mathrm{d}(0, 0) = 0$. The full population dynamical equations are then given by

$$\frac{\mathrm{d}S}{\mathrm{d}t} = [b(S, I) - d(S, I) - \beta I]S, \quad \frac{\mathrm{d}I}{\mathrm{d}t} = [\beta S - \alpha - d(S, I)]I, \tag{4.15}$$

with

$$b(S, I) = b_0 - h_\mathrm{b}(S, I), \quad d(S, I) = d_0 + h_\mathrm{d}(S, I). \tag{4.16}$$

(The implicit assumption that infected individuals are not allowed to reproduce greatly simplifies the proofs of the attractivity of the equilibria.)

The parameters α and β are assumed to be under evolutionary control by the disease (evolution in host-controlled traits is not considered here). In agreement with the standard custom, we assume α and β to be connected by a constraint: β cannot become too high and α simultaneously not too low, which can be expressed as $g(\alpha, \beta) \leq m$ with g increasing in β and decreasing in α. As evolution acts to increase β and decrease α, it will quickly run into this constraint. From there on, evolution will effectively be restricted to the curve $g(\alpha, \beta) = m$, alternatively parameterised as $\beta = \beta(\alpha)$, or as $(\alpha(x), \beta(x))$ for some scalar physiological trait x.

Within the general class of models (4.15), we consider four special cases,

$$\begin{array}{lll} \text{(i)} & h_{\mathrm{b}}(S,I) = \kappa(S+I), & h_{\mathrm{d}}(S,I) = 0, \\ \text{(ii)} & h_{\mathrm{b}}(S,I) = 0, & h_{\mathrm{d}}(S,I) = \kappa S, \\ \text{(iii)} & h_{\mathrm{b}}(S,I) = 0, & h_{\mathrm{d}}(S,I) = \kappa S^2, \\ \text{(iv)} & h_{\mathrm{b}}(S,I) = 0, & h_{\mathrm{d}}(S,I) = \kappa(S+I). \end{array} \tag{4.17}$$

These model families have been rigged so that for model (i) and (ii) the environmental feedback for the disease is one-dimensional monotone. According to Propositions 3.13 and 3.14, these models thus support an optimisation principle. For model (i) the optimisation principle is equivalent (i.e., monotonically related) to R_0, while for model (ii) this is not the case. For model (iii) the environment feedback acts one-dimensionally but not monotonically, and for model (iv) it acts two-dimensionally.

It should be understood that we chose the specific examples in Equation (4.17) primarily for didactical purposes. For their individual-based underpinning one may think of population regulation through fighting. For models (i) and (iv) fighting may be initiated by all individuals, whereas for models (ii) and (iii) infected individuals are assumed to suffer from fights without being able to initiate such fights themselves. Model (iii) is based on the assumption of aggression increasing linearly with aggressor density. Fighting, of course, may here be replaced by any other form of interference competition.

In this application we will distinguish the customary evolutionary R_0, i.e., the infection-time production of new disease cases by a mutant disease case introduced into a resident disease in equilibrium with its host, from the customary epidemiological R_0 by denoting the former as R. We start by expressing R as a general function of the mutantt traits $\check{X} = (\check{\alpha}, \check{\beta})$ and of the variables (S, I) parameterising the potential environmental conditions,

$$\begin{array}{ll} \text{(i)} & R(\check{\alpha}, \check{\beta}|S,I) = \frac{\check{\beta}S}{\check{\alpha}+d_0}\,, \\ \text{(ii)} & R(\check{\alpha}, \check{\beta}|S,I) = \frac{\check{\beta}S}{\check{\alpha}+d_0+\kappa S}\,, \\ \text{(iii)} & R(\check{\alpha}, \check{\beta}|S,I) = \frac{\check{\beta}S}{\check{\alpha}+d_0+\kappa S^2}\,, \\ \text{(iv)} & R(\check{\alpha}, \check{\beta}|S,I) = \frac{\check{\beta}S}{\check{\alpha}+d_0+\kappa(S+I)}\,. \end{array} \tag{4.18}$$

It is only later that we will confine attention to the realisable environments, given by the equilibrium values $(\hat{S}(\alpha, \beta), \hat{I}(\alpha, \beta))$ produced by the possible residents $X = (\alpha, \beta)$.

For model (i), R increases with S. So the optimisation principle can be constructed directly from (3.6). Minimising $\hat{S}$, which can easily be seen from (4.15)-(4.17) to yield $\hat{S} = (\alpha+d_0)/\beta$, should thus be equivalent to maximising $\phi(\alpha,\beta) = -\hat{S} = -(\alpha+d_0)/\beta$. To calculate R_0 for this model, we observe that $R_0(\alpha,\beta) = R(\alpha,\beta|S_0,0) = \beta S_0/(\alpha+d_0)$, with S_0 denoting the equilibrium value for S in the absence of the disease. It is not difficult to see that R_0 and the ϕ resulting from our general construction are indeed monotonically related, independent of the value of S_0.

For model (ii), R is again monotone in S. From $E_{\mathrm{attr}}(X) = \hat{S} = (\alpha + d_0)/(\beta-\kappa)$, we find the matched optimisation principle $\phi = -(\alpha+d_0)/(\beta-\kappa)$. However, maximising ϕ is not equivalent to maximising $R_0 = \beta S_0/(\alpha + d_0 + \kappa S_0) = \beta(b_0 - d_0)/[\kappa(\alpha + b_0)]$, where we used $S_0 = (b_0 - d_0)/\kappa$ as for model (i). To see this non-equivalence, it suffices to observe that the contour lines, defined by $R_0(\alpha,\beta) = R_0(\alpha_0,\beta_0)$ and $\phi(\alpha,\beta) = \phi(\alpha_0,\beta_0)$ for given (α_0,β_0), differ, as can be seen from the lack of coincidence in their derivatives at (α_0,β_0), calculated via an implicit differentiation of the defining relations: $\mathrm{d}\alpha/\mathrm{d}\beta = (\kappa\alpha_0 + b_0)/(\beta_0\kappa)$ for R_0, which differs from $\mathrm{d}\alpha/\mathrm{d}\beta = (\alpha_0 + d_0)/(\beta_0 - \kappa)$ for ϕ.

The fact that invasion fitness in model (iii) is non-monotone in any scalar summary of the condition of the environment, and that the evolutionary environment in model (iv) is essentially two-dimensional, can already be guessed from (4.18). However, for a proof we have to deal with the fact that, for instance, in model (iii) R should be non-monotone relative to whatever summary variable even when its domain is restricted to the realisable values of S and in addition to an infinitesimal neighborhood of those combinations of $(\check{\alpha},\check{\beta})$ and $\hat{S}(\alpha,\beta)$ for which $R(\check{\alpha},\check{\beta}|\hat{S}(\alpha,\beta)) = 1$. The necessary technicalities can be found in [8, Appendix A]. For the present exposition it suffices to note that in cases (iii) and (iv) the directions $\mathrm{d}\alpha/\mathrm{d}\beta$ in (α_0,β_0) of the contour lines $\{(\alpha,\beta)|R(\alpha,\beta|S(\alpha_0,\beta_0),I(\alpha_0,\beta_0)) = 1\}$ and $\{(\alpha,\beta)|R(\alpha_0,\beta_0)|S(\alpha,\beta),I(\alpha,\beta)) = 1\}$, which can be determined by implicit differentiation, are generically different. Hence, by Corollary 3.12 neither case allows an optimisation principle.

5. Discussion

The title question was interpreted by us as: find necessary and sufficient conditions on eco-evolutionary models such that the ESSes for these models for all possible constraints on the trait space $\mathcal{X}$ can be calculated by optimising some function $\phi : \mathcal{X} \to \mathbb{R}$. At the highest level of abstraction this question was answered by naming two conditions that should be satisfied by the invasion fitness function $(X,Y) \mapsto s_X(Y)$: (i) it should be sign-antisymmetric, that is, $s_X(Y) > 0 \Leftrightarrow s_Y(X) < 0$, and (ii) there should be no weak rock-scissors-paper configurations, that is, triples (X,Y,Z) such that $s_X(Y) \geq 0$, $s_Y(Z) \geq 0$, and $s_Z(X) > 0$. At a lower level of abstraction

this was found to be equivalent to both the trait and the environment acting effectively in a one-dimensional monotone manner. On a still lower level, that of life history parameters, the picture becomes more diffuse. As even for reasonably delimited classes of models no necessary and sufficient conditions are available yet, all we could do is give a brief summary of the insights that are available at the current time. This clearly is a highly interesting area for further research.

Of course, there exist still lower levels of abstraction, like the organisation of foraging or the construction of bones. Although quite a lot of optimisation modelling is done here in concrete applications, it for the time being appears inopportune to extend our approach to these levels, primarily since those applications customarily leave open the full eco-evolutionary context that would be the ultimate justification of the presumed optimisation principle. The best that one can say is that these applications should probably be considered as attempts at predicting not so much evolutionary outcomes as well as predicting properties of individuals from the environment in which they are observed to live, on the supposition that the combination of realised trait value and environment is currently sitting at an ESS, and that the optimised quantity is a fair fitness proxy.

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