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Using stage-structured evolutionary game theory to model the experimentally observed evolution of a genetic polymorphism

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ABSTRACT

Aim: Our aim is to show the utility of evolutionary game theory (EGT) methods in describing and predicting the outcome of experiments for which genetic data are available in the absence of phenotypic data. As an example we use experimental data from genetically perturbed cultures of the flour beetle *Tribolium castaneum*. Using natural selection, the theory provides a theoretical basis for the observed population dynamics and evolution of a polymorphism of wild-type and corn oil sensitive alleles.

Method of analysis: We derive an EGT version of a well-validated model for the population dynamics of *Tribolium* (the LPA model) using a version of EGT developed for stage-structured populations given by Vincent and Brown (2005). We use the wild-type allele frequency as the strategy in this model. We estimate model parameters and conduct simulations using the parameterized model.

Experimental data: We use population and genetic data from an experiment conducted by Desharnais (1979). Cultures of *T. castaneum* homozygous for corn oil sensitivity are perturbed by adding homozygous wild-type individuals. The data include population densities of larvae, pupae, and adults as well as allele frequencies obtained from genetically perturbed cultures.

Conclusions: The parameterized EGT version of the LPA model fits reasonably accurately the population and genetic data in both the control and genetically perturbed treatments. For both treatments, the model predicts an oscillatory 2-cycle asymptotic attractor. The predicted oscillations match well with the oscillations observed in the population data, even capturing observed increases in amplitude and population densities in the genetically perturbed treatment. Interestingly, the model also predicts a periodic oscillation in allele frequency, although the amplitude is too small to be detected in the data. We conclude that the EGT-predicted evolution of a polymorphism by natural selection is obtained in agreement with the experimental data. Our results illustrate two points concerning EGT: first, the theory can be successful in application to real data and, second, it can be applied in a context that allows a connection to classical population genetics theory.

Keywords: allele frequency, evolutionary game theory, genetic polymorphism, LPA stage-structured model, *Tribolium*.

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INTRODUCTION

Evolutionary game theory (EGT) models phenotypic and population dynamics simultaneously, allowing one to study evolutionary changes in a biological population as they are influenced by changing population densities. The three main elements of an evolutionary game are players, strategies, and payoffs. The players are the individual organisms, the strategies are phenotypes with a heritable (genetic) component, and the payoffs are relative fitness gains or losses, usually expressed as net reproductive rates of individual organisms. Thus, EGT provides a quantitative prediction of an evolutionarily attainable strategy in addition to the population density when this strategy is reached. It also provides a context for the study of an evolutionarily stable strategy (ESS) that, unlike classical population genetics techniques, also models the population dynamics.

The evolutionary game has two parts. One part involves population dynamics as influenced by the strategy that governs fitness; the other part involves the evolutionary process resulting in changes in mean strategy (Vincent and Brown, 2005). The modelling methodology based on EGT couples the two parts: the population dynamics portion of the game, which is influenced by changes in mean strategy, and the strategy dynamics portion, which models changes in mean strategy over time as they are influenced by the population dynamics. Both population density and mean strategy influence payoffs to individuals.

In this paper, we use EGT to model an experimental study of the evolution of corn oil sensitivity in the flour beetle *Tribolium castaneum* (Herbst) (Moffa and Costantino, 1977; Desharnais and Costantino, 1980; Costantino and Desharnais, 1991; Dennis *et al.*, 1995). We base our EGT model on a population dynamic model that has been rigorously validated, by numerous experimental studies, for the dynamics of *T. castaneum* (Dennis *et al.*, 1995; Cushing *et al.*, 2003; Costantino *et al.*, 2005). This model – called the LPA model – has the form of a (discrete time) Leslie matrix model. The model describes the dynamics of three life-cycle stages of *Tribolium*; namely, the larval, pupal, and adult stages. [For a study based on a more simplistic model based on total population numbers, see Rael *et al.* (2007).] Vincent and Brown (2005) developed the EGT methodology for general matrix models, and it is this approach that we adopt here in application to the LPA model for *Tribolium*. We show how the resulting EGT version of the LPA model can accurately describe the experimental data from the evolutionary study of *T. castaneum*.

Although one usually uses EGT to model dynamics in a Darwinian sense in which strategies are phenotypes, we use the theory here in a context in which the strategies are allele frequencies. We do this because genetic data, rather than phenotypic data, are available in the *Tribolium* experiment. This approach could be used in other applications in which genetic information is more readily available than suitable phenotypic data. In our application to the *Tribolium* experiment, the strategy is a genetically determined phenotype in *T. castaneum* that results from variation in a single gene that controls sensitivity to corn oil. Individuals homozygous for the *cos* allele are unable to properly digest unsaturated fatty-acid present in corn oil (Costantino *et al.*, 1966). This imparts variable rates of fecundity and mortality when cultured on a corn oil substrate (Desharnais and Costantino, 1980). Individuals may have one of three possible genotypes: *cos*/*cos*, *cos*/+, or +/+ , where + represents the wild-type allele. We define strategy as a numerical representation of genotype where *cos*/*cos* individuals have strategy 0, *cos*/+ individuals strategy 0.5, and +/+ individuals strategy 1. This allows us to interpret the mean strategy of the population as the frequency of the +allele.

AN EVOLUTIONARY LPA MODEL

The flour beetle goes through three distinct life-history stages upon emerging from an egg: larval, pupal, and adult. Individuals spend approximately 2 weeks in both the larval and pupal stages. An important characteristic of these organisms is cannibalism. Adults eat eggs and pupae, and larvae consume eggs. These cannibalistic interactions impart density-dependent regulation on the population dynamics (Costantino and Desharnais, 1991). The LPA (Larva–Pupa–Adult) model developed by Dennis *et al.* (1995) has the form of a non-linear Leslie matrix model (Caswell, 2001):

$$\mathbf{x}' = \mathbf{x}\mathbf{H}^{\mathrm{T}}(\mathbf{w}, \mathbf{x}),$$

where

$$\mathbf{x} = [L\ P\ A]$$

denotes the demographic vector of larval, pupal, and adult numbers, $\mathbf{x}'$ denotes the demographic vector one time unit later, $\mathbf{H}(\mathbf{w}, \mathbf{x})$ is the fitness matrix

$$\mathbf{H}(\mathbf{w}, \mathbf{x}) = \begin{bmatrix} 0 & 0 & b\exp(-c_{el}L - c_{ea}A) \\ 1-\mu_l & 0 & 0 \\ 0 & \exp(-c_{pa}A) & 1-\mu_a \end{bmatrix}, \tag{1}$$

and

$$\mathbf{w} = [\,b\ \mu_l\ \mu_a\ c_{pa}\ c_{el}\ c_{ea}]$$

is the vector of the model parameters:

b = average number of larvae recruited per adult per unit time;
μ_l = larval probability of dying from causes other than cannibalism;
μ_a = adult probability of dying from causes other than cannibalism;
c_{pa} = strength of pupal cannibalism by adults;
c_{el} = strength of egg cannibalism by larvae;
c_{ea} = strength of egg cannibalism by adults.

For flour beetles grown under standard laboratory conditions, one time step in the model represents 2 weeks, which is the time spent in both the larval and pupal stages. The LPA model has had considerable success in describing and predicting the dynamics of *T. castaneum* in many experiments, including studies of dynamic bifurcation, chaos, the effects of demographic stochasticity, resonance properties in periodic environments, and lattice effects (Cushing *et al.*, 2003; Costantino *et al.*, 2005; Reuman *et al.*, 2006, 2008). For this reason, we take this well-validated model for the population dynamic part of our EGT model.

To cast this system into a multi-stage EGT format, we must identify which parameters can evolve over time and which are fixed constants. Sensitivity to corn oil affects reproduction and mortality rates, and as a result we assume that the recruitment, b, and the death rates, μ_l and μ_a, are functions of a scalar strategy variable used by the beetles. We assume that cannibalism is unaffected by this strategy and use the constant values for c_{pa}, c_{el}, and c_{ea}.

Before specifying the details of our model, we briefly summarize the methodology of EGT as applied to stage-structured matrix models as given by Vincent and Brown (2005). In its broadest context, EGT deals with the evolution and stability of strategies with respect to invasion by species using alternate mutant strategies. In this context, one must deal with the possibility of multiple species with population dynamics of the form

$$\mathbf{x}_i' = \mathbf{x}_i \mathbf{H}_i^{\mathrm{T}}(\mathbf{u}, \mathbf{x}),$$

where $i = 1, \ldots, n_s$; n_s is the total number of species in the community; and

$$\mathbf{u} = [u_1 \ldots u_{n_s}]$$

is the vector of the scalar strategies used by the species. If all species can be described in terms of a fitness-generating matrix (Vincent and Brown, 2005) of the form

$$\mathbf{G}(v, \mathbf{u}, \mathbf{x})|_{v=u_i} = \mathbf{H}_i(\mathbf{u}, \mathbf{x}),$$

then it is possible to find evolutionarily stable strategies (ESS) for this multi-stage evolutionary game. The variable v is a virtual variable with the property that when v is replaced by u_i, the **G**-matrix becomes identical to the $\mathbf{H}_i$-matrix for the species i. In the context of the **G**-matrix, the population dynamics for any given species is given by

$$\mathbf{x}_i' = \mathbf{x}_i \mathbf{G}^{\mathrm{T}}(v, \mathbf{u}, \mathbf{x})|_{v=u_i}. \tag{2}$$

An essential feature of evolutionary game theory is the ability to track how strategies evolve with time (strategy dynamics). Using the method provided by Vincent and Brown (2005) for stage-structured matrix models, a critical value for each $\mathbf{H}_i$-matrix is defined by

$$\mathrm{crit}\, \mathbf{H}_i(\mathbf{u}, \mathbf{x}) = \max[\mathrm{abs}(\boldsymbol{\lambda}_i)],$$

where $\boldsymbol{\lambda}_i$ is the vector of eigenvalues corresponding to $\mathbf{H}_i(\mathbf{u}, \mathbf{x})$. Thus, the critical value of $\mathbf{H}_i$ is a scalar corresponding to the maximum absolute value of the components contained in the vector $\boldsymbol{\lambda}_i$. This allows for the definition of a scalar H-function and a scalar G-function

$$H_i(\mathbf{u}, \mathbf{x}) = \mathrm{crit}\, \mathbf{H}_i(\mathbf{u}, \mathbf{x})$$

$$G(v, \mathbf{u}, \mathbf{x})|_{v=u_i} = H_i(\mathbf{u}, \mathbf{x}).$$

Strategy dynamics for stage-structured matrix models are expressed in terms of the G-function:

$$u_i' = u_i + \left[\frac{\sigma_i^2}{G(v, \mathbf{u}, \mathbf{x})|_{v=u_i}}\right] \left.\frac{\partial G(v, \mathbf{u}, \mathbf{x})}{\partial v}\right|_{v=u_i}. \tag{3}$$

This result is derived under the assumption that rate of change of a trait over time is proportional to the amount of variation present in the population, as given by σ_i^2.

In the case where the community under consideration is composed of a single species (as is the case in the *T. castaneum* laboratory experiment under consideration), we drop the subscripts. The ecological dynamics (2) with the strategy dynamics (3) give the Darwinian dynamics for the evolutionary game:

$$\mathbf{x}' = \mathbf{x}\, \mathbf{G}^{\mathrm{T}}(v, \mathbf{u}, \mathbf{x})|_{v=u}$$

$$u' = u + \left[\frac{\sigma^2}{G(v, u, x)|_{v=u}}\right] \left.\frac{\partial G(v, \mathbf{u}, \mathbf{x})}{\partial v}\right|_{v=u}. \tag{4}$$

When the projection matrix **G** is irreducible and primitive, the classic Perron-Frobenius theory implies that G is the strictly dominant eigenvalue r of **G**. This is the case when **G** is the Leslie matrix (1) defining the LPA model. We complete our EGT version of the LPA model when we identify how the model parameters b, μ_l, and μ_a depend on the strategy v. Our description of this dependence is based on experimental data.

Recall that we identify the mean strategy v in our model to be the mean frequency of the wild-type +allele in the three possible genotypes: *cos*/*cos*, *cos*/+, and +/+. The average values of b, μ_l, and μ_a are known for each of the genotypes (Moffa and Costantino, 1977; Desharnais and Costantino, 1980). A quadratic polynomial fit to these three data points results in the formulas (see Rael *et al.*, 2007)

$$\begin{aligned} b(v) &= -18v^2 + 21v + 11 \\ \mu_l(v) &= 0.10v^2 - 0.13v + 0.51 \\ \mu_a(v) &= 0.10v^2 - 0.13v + 0.11 \end{aligned} \tag{5}$$

(see Fig. 1). These are interpreted as the average recruitment and death rates in a population with a +allele frequency of v. When modelled this way, a population with a +allele frequency of approximately 0.58 has the highest reproduction rate, and one with a frequency of 0.65 has the lowest mortality rate. The range of parameter values, when given by these formulas, is consistent with the parameter estimates and confidence intervals given by Dennis *et al.* (1995).

With the parameter assignments (5), our EGT version of the LPA model becomes

$$[L\ P\ A]' = [L\ P\ A]\ \mathbf{G}^{\mathrm{T}}\ (v, L, P, A)|_{v=u} \tag{6}$$

$$u' = u + \frac{\sigma^2}{r(u, L, P, A)} \left.\frac{\partial r(v, L, P, A)}{\partial v}\right|_{v=u}$$

where

$$G = r(v, L, P, A)$$

is the dominant eigenvalue of the Leslie matrix

$$\mathbf{G}(v, L, P, A) = \begin{bmatrix} 0 & 0 & b(v)\exp(-c_{el}L - c_{ea}A) \\ 1-\mu_l(v) & 0 & 0 \\ 0 & \exp(-c_{pa}A) & 1-\mu_a(v) \end{bmatrix} \tag{7}$$

and evolving parameters $b(v)$, $\mu_l(v)$, and $\mu_a(v)$ given by (5). Dennis *et al.* (1995) also provide the parameter estimates $c_{pa} = 0.015$, $c_{el} = 0.0093$, and $c_{ea} = 0.011$ for *T. castaneum*.

The Darwinian dynamics given by our EGT LPA model (5, 6, 7) yield the temporal change in both the population densities of the larvae, pupae, and adults and the mean strategy (mean frequency of the +allele) as they influence one another.

THE EXPERIMENT

The data we use in this study are from an experiment reported by Desharnais and Costantino (1980). Cultures of the flour beetle *Tribolium castaneum* homozygous for the *cos* allele were started with identical stage distributions of 70 small larvae, 20 large larvae, 16

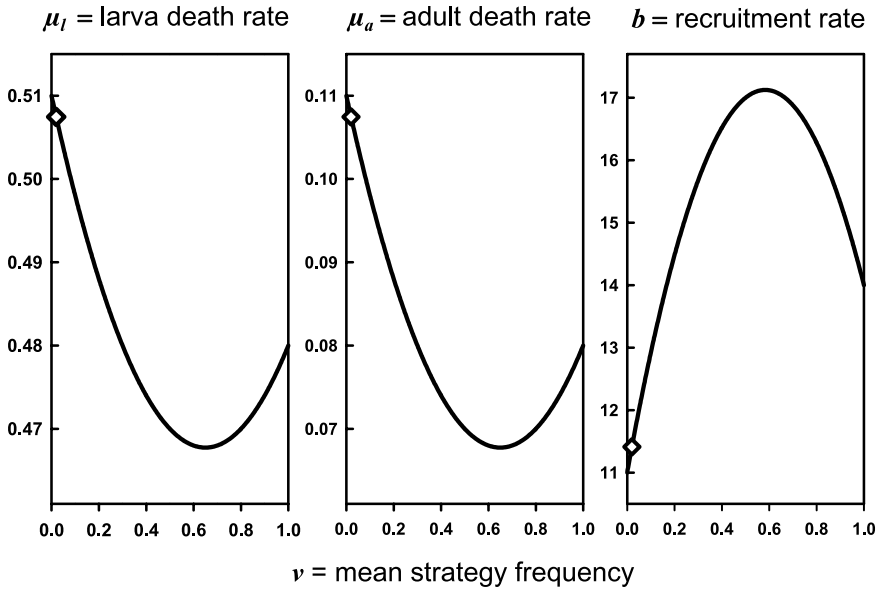

Fig. 1. The fitted recruitment and death rates b, μ_l, and μ_a as functions of strategy v as given by (5). The initial +allele frequency (denoted by the open diamond) in the genetically perturbed treatment is 0.02.

pupae, and 64 adults. Each culture was contained in a half-pint milk bottle with 20 g of corn oil medium (90% wheat flour, 5% dried brewer's yeast, 5% liquid corn oil), and kept in an unlighted incubator at 33 ± 1°C and 56 ± 11% relative humidity. Cultures were censused for larvae, pupae, and adults biweekly for 38 weeks. After 10 weeks, one female adult with the +/+ genotype was placed into the homozygous *cos*/*cos* culture. In the control treatment, no manipulations were imposed. Allele frequency data were recorded every 4 weeks for 24 weeks after the introduction of the +allele. We examined data from a control culture and a culture with one adult female with the genotype +/+ added to a homozygous *cos* culture.

RESULTS

The EGT LPA model yields results that are reasonably consistent with (and capture important features of) the data from the corn oil sensitivity experiment reported in Desharnais (1979; see also Desharnais and Costantino, 1980). Figure 2 shows comparisons of a model simulation with the experimental data for both the genetically perturbed treatment and the control treatment. The plots in Fig. 2 show both the population dynamics (namely, the dynamics of the larval, pupal, and adult stages) and that of the +allele frequency (strategy). Each plot shows the data together with a simulation of the EGT LPA model (5, 6, 7). The simulation used initial values for population size and +allele frequency corresponding to those of the experimental control treatment and the treatment upon addition of +alleles into the population, namely $[L\ P\ A\ u] = [76\ 47\ 62\ 0]$ for the control treatment and $[L\ P\ A\ u] = [13\ 136\ 62\ 0.02]$ for the perturbed treatment. The variance σ^2 in this simulation is 0.4.

In Fig. 2 we see that, in addition to a reasonably good quantitative fit of the model predictions to the experimental data, the simulation captures several important features of

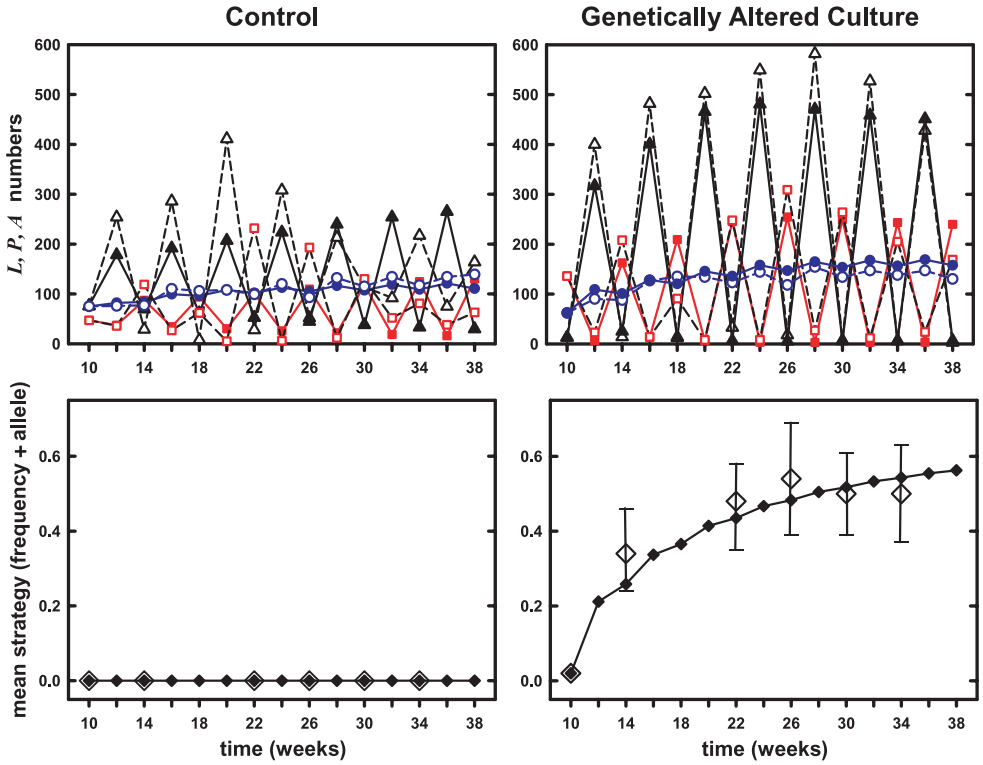

Fig. 2. The left and right columns of plots show the data and a model simulation for the control and genetically perturbed treatments respectively. Data are shown as open symbols (connected by dashed lines) and simulations are solid symbols (connected by solid lines). Triangles are numbers of larvae (L), squares are numbers of pupae (P), circles are numbers of adults (A), and diamonds are +allele frequencies (v). The +allele frequencies shown in the genetically perturbed treatment (lower right) are averages over three treatments (with error bars). The demographic data (upper plots) are for one of the control and genetically altered replicates. Parameter values used in the model simulation, in addition to those in Fig. 1, are $c_{pa} = 0.015$, $c_{el} = 0.0093$, $c_{ea} = 0.011$, and $\sigma^2 = 0.40$. Initial conditions are $[L\ P\ A\ u] = [76\ 47\ 62\ 0]$ for the control treatment and $[L\ P\ A\ u] = [13\ 136\ 62\ 0.02]$ for the perturbed treatment. The initial +allele frequency in the genetically perturbed treatment is 0.02.

the data. In the control treatment, the model predicts that the population dynamics exhibit oscillations (of period 2) in all three life-cycle stages, as one observes in the data. In this treatment, the homozygous (*cos*/*cos*) genotype remains unchanged, as shown by the horizontal plot of the mean strategy dynamic. In the genetically perturbed treatment, the model again predicts periodic oscillations in all three life-cycle stages, and these oscillations are indeed observed in the data. It is noteworthy that the model accurately predicts that the amplitude of these oscillations will increase significantly as the genetic evolution occurs, as one also observes in the experimental data. Moreover, the model predicts an evolution to a polymorphic population with +allele frequency of approximately 0.6, the same value obtained in the experiment.

The predicted asymptotic attractor of the model is a periodic 2-cycle. The amplitude of the oscillations in the population dynamic components L, P, and A of the 2-cycle attractor

are significantly large and easily observable in both the model simulation and the data. On the other hand, the 2-cycle oscillation in the mean +allele frequency component v of the attractor is approximately 0.002 with a mean of $v = 0.627$. (This attractor was not observed during the course of the experiment.) Although it is interesting that our model predicts a non-equilibrium oscillation as the evolutionary attractor, it is not possible to detect such a small amplitude in the experimental data.

Note that these model-predicted 2-cycle values for v do not maximize the recruitment rate $b(v)$ (whose maximum occurs at $v = 7/12 = 0.5833$) or minimize the mortality rates $\mu_l(v)$ and $\mu_a(v)$ (whose minima occur at $v = 13/20 = 0.65$). If the mean strategy were to reach an equilibrium value, we would expect fitness r to be maximized (at a value of 1) at the equilibrium strategy. In our case here, however, the model-predicted long-term dynamic is an oscillatory 2-cyle and, as a result, both the mean strategy v and the adaptive landscape defined by r periodically oscillate. Interestingly, for the simulation shown in Fig. 2, the two values assumed by the periodic mean strategy nearly (but not exactly) provide maximum fitness r at each phase of the 2-cycle oscillation in the fitness landscape. The maximum fitness values are alternately greater than and less than 1 (with a mean approximately equal to 1). This is predicted by the strategy equation in (6), which, when averaged over the 2-cycle, implies that the average logarithmic derivative of fitness r equals 0. Consequently, the two 2-cycle values of the mean strategy are alternately on the right and left side of (and hence below) peak fitness in the adaptive landscape. Specifically, at those times in the cycle when maximum fitness r is greater than 1, the strategy u decreases and moves away from the maximum. At the alternate times in the cycle when maximum fitness r is less than 1, the strategy increases and moves towards the maximum (see Fig. 3). However, more investigation and expanded EGT principles are needed to determine whether the strategy 2-cycle attractor has ESS properties, such as non-invasibility by mutant strategies.

DISCUSSION

Evolutionary game theory can be applied to the maintenance of polymorphisms within populations (Vincent and Brown, 2005). Although this is typically done with strategies representing phenotypes, our results show that evolutionary games are potentially useful tools for predicting long-term allele frequencies as well. Using genotype as the adaptive trait, the EGT model produced allele frequencies and population sizes similar to those found in experimental data. These data suggest that there is a stable polymorphism in the population; that is, two alleles persist due to a fitness advantage conferred by the heterozygous ($cos/+$) genotype. The population in the evolutionary game reaches a 2-cycle attractor due to the population density regulation imposed by the cannibalism in the model (Costantino and Desharnais, 1991). The predicted oscillations in the population density components of the attractor fit reasonably accurately the experimental data in both the control and genetically perturbed treatments. The predicted +allele frequency also fits the data well, although the predicted oscillation is of too small amplitude to be detected experimentally. Nonetheless, the model supports the existence of a stable polymorphism at this genetic locus.

Our model produces higher population densities when the +allele is introduced than when no manipulations are imposed, as supported by the data (Moffa and Costantino, 1977; Desharnais and Costantino, 1980). This is an important pattern to note, as it supports the theory that populations have the ability to reach larger sizes if they have a higher mean fitness (Desharnais and Costantino, 1980).

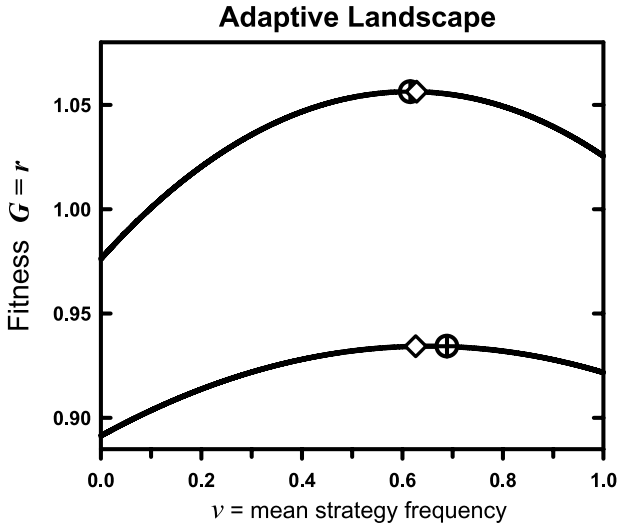

Fig. 3. The simulation shown in Fig. 2 predicts an oscillatory attractor in which both the population and strategy (+allele frequency) dynamics oscillate periodically with period 2. The resulting fitness landscape oscillates between two contours with peaks whose maxima are denoted by the crossed circles. The two values of the +allele frequency in the periodic oscillation lie on alternating sides of fitness peaks, as shown by the open diamonds.

Genetic variation in a population directly affects the rate of evolution, or change in mean strategy of a population (Vincent and Brown, 2005). Since higher amounts of variation in a population are associated with faster evolution, it is expected that introducing different amounts of the +alleles would produce different rates of evolution. The rate of evolution in the model is captured by the parameter σ^2, as strategy dynamics scale with variance (Vincent and Brown, 2005). However, there is some difficulty in estimating the variance parameter σ^2 directly from the data available for the *Tribolium* experiment. In our simulation study, we found that values of σ^2 that provide a good fit to the frequency data are at the upper end of the range of variance in the data and slightly higher. Our genetically perturbed treatments were part of the original experiment by Desharnais (1979). We also found, in studying these other treatments (see also Rael *et al.*, 2007), that the values of σ^2 in the model that produce good fits to the data correlate with the amount of variance in the initial genetic perturbation: higher σ^2 values correspond to higher initial variance.

In our model, the mean strategy corresponds to the frequency of the +allele in the population with our numerical convention for representing genotypes as strategies. This is illustrated as follows. If we let P_1, P_2, and P_3 denote the number of individuals with the *cos*/*cos*, *cos*/+, and +/+ genotypes respectively, then the frequency of the +allele (p) is given by Russell (2000):

$$p = \frac{0.5P_2 + P_3}{\sum_{i=1}^{3} P_i}.$$

This is also the mean strategy of the population given that the strategies of the above genotypes are 0, 0.5, and 1 respectively. This convention therefore allows us to apply EGT to determine long-term gene frequencies.

While classical genetic models predict equilibrium allele frequencies based on fitness, our use here of EGT methods in a genetic context provides a more detailed picture of allele frequency dynamics by incorporating population dynamics. These represent two characteristics of populations that significantly influence one another, ultimately playing a role in determining the evolutionary outcome by means of natural selection. In this study, we also demonstrate the effectiveness of EGT models in quantitatively describing and predicting evolutionary population dynamics, using genetic information, by means of an application to data from an experiment involving genetic perturbations in cultures of *T. castaneum*. Moreover, this application demonstrates that EGT can be successful in those circumstances when genetic information is available, but suitable phenotypic data are not – a circumstance that might occur often in other potential applications of EGT. In summary, our results illustrate two points concerning EGT: first, the theory can be successful in application to real data and, second, it can be applied in a context that allows a connection to classical population genetics theory.

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