The dynamics of sex ratio evolution
Dynamics of global population parameters

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HIGHLIGHTS

- ESS sex ratio models are inconsistent with population genetics sex ratio models.
- Genetic models consider also composition of male subpopulation.
- New model shows that 0.5 sex ratio is evolutionarily stable only in so called Male Subpopulation Equilibrium manifold (MSE).
- In MSE state, male subpopulation is in unique equilibrium conditional on the current composition of female subpopulation.
- 0.5 sex ratio is unstable for perturbations of male subpopulation composition.

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ABSTRACT

Classical formalizations of the Fisherian theory of sex ratio evolution are based on the assumption that the number of grand offspring of a female serves as a measure of fitness. However, the classical population genetics approach also considers the contribution of male individuals to gene proliferation. The difference between the predictions of phenotypic and genetic models is that the phenotypic approach describes the primary sex ratio of 0.5 as the ESS value, while genetic models describe the stable state of a population by a combination of the stable states of the male and female subpopulations. In this paper, we formulate an alternative model of sex ratio evolution that is focused on the dynamics and quantitative properties of this process and that combine a rigorous genetic approach with a game theoretic strategic analysis. In the new model, females are the strategic agents and males are the passive carriers on unexpressed genes. Fitness functions in the new model are derived with respect to a “fitness exchange” effect, i.e. the contribution of male individuals to female fitness and vice versa. This new model shows that the dynamics of this system are complex and consist of two phases. The first, rapid, phase converges the system to a stable manifold (termed the male subpopulation equilibrium—MSE) where the male subpopulation state is in equilibrium, conditional on the current state of the female subpopulation. Double phase dynamics occur when the population state is not compatible with the current strategic composition of the population (determined by the value of the primary sex ratio) which can be caused by ecological factors. The trajectory of convergence to the MSE can be very complicated and may contain a dramatic change in the primary sex ratio. Thus, the primary sex ratio of 0.5 is unstable for perturbations of gene frequencies among male carriers. Therefore, the new model supports predictions of genetic models that the evolutionary stability of the sex ratio should be characterized by a combination of a stable value of the primary sex ratio and the male subpopulation equilibrium.

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1. Introduction

In nature, the observed ratio of males to females is, in most cases, close to 0.5, even though a single male can fertilize many females. In many species, a group of dominant males can monopolize all of the females in the population, thus making the reproduction of other males impossible. Because not all males produce offspring, many individuals are excluded from reproduction, and such a population has a lower mean fitness than would a population in which the majority of individuals reproduce. Therefore, the origin of the sex ratio of 0.5, which is not optimal from the point of view of simple maximization of a fitness measure, such as the rate of population growth, requires explanation. Darwin considered this problem but could not come to a conclusion about it. A first attempt to clarify this phenomenon was made at the end of the 19th century by the German...
biologist Carl Dusing, which was also the first application of mathematical methods to evolutionary biology (Edwards, 2000). His result is founded on one basic assumption: the number of grand offspring is assumed to serve as a fitness measure of a female that produces offspring at the sex ratio that is encoded in her genes. Even under a situation of genetic determination of sex with males producing two classes of sperm, a female may control the sex ratio of her offspring by spontaneous aborations of fetuses of the unwanted sex or by allowing biased access of sperm to her eggs. Female control of the sex ratio is even easier to exert when two classes of eggs (male or female determined) are produced. Ronald A. Fisher made an argument similar to Dusing’s in his 1930 book, and this concept is often attributed to him (Fisher, 1930; Queller, 2006). However, female control is not explicitly assumed in his reasoning. Fisher instead considers a subpopulation of individuals (“parents”) without distinction of which sex determines the sex ratio among newborns. A similar approach that is focused on the level of the group adjusting the sex ratio among offspring can be found in a classical book on population genetics by Crow and Kimura (Crow and Kimura, 1970). However, this type of reasoning is not individual based, which is necessary for a game theoretic interpretation. The missing assumption, necessary to the introduction of the individual level, can be found in Dusing’s work. He assumed that females produce offspring according to their strategy, thus the parental population is limited to females (called “mom’s” in Charnov (1982)). The Dusing–Fisher approach gives the following predictions: in the case of a shift of the population sex ratio to an excess of males, a female that produces proportionally more female offspring than other females will have a higher number of grand offspring; similarly, when the population sex ratio is shifted toward females, the production of a higher proportion of males will ensure a higher number of grand offspring; and thus, a population sex ratio of 0.5 represents a stable, stationary state. Formalization of this reasoning can be obtained by the construction of a fitness function whose arguments include the following: (i) an individual strategy (i.e., the sex ratio in a female brood) and (ii) the population sex ratio. From a game theory perspective, the problem can be formulated in the following way: players are females, and the proportion of males in the brood of a single female, described by \( P_i \), is her individual strategy; \( P \) represents the secondary sex ratio (ratio among adult individuals that are able for reproduction), interpreted as the proportion of males; and if \( N \) describes the number of all individuals in the population, then

\[
P_n \text{ is the number of males in the population; } \quad (1-P)N \text{ is the number of females in the population; } \quad k \text{ is the number of offspring of a single female; } \quad P_i \text{ is the proportion of males in the brood of a single female.}
\]

Then the primary sex ratio is equal to the mean female subpopulation strategy

\[
P_{sp} = \sum f_i P_i
\]

where \( f_i \) is the fraction of \( P_i \) females in the population. Then, we can derive a payoff function that describes the number of grand offspring of a female with a strategy \( P_i \). A female with the strategy \( P_i \) will have \( P_i k \) sons and all females in the population will produce on average \( P_{sp} k \) sons. When the offspring grow up and become the next generation, the primary sex ratio in the mothers’ generation \( P_{sp} \) will become the secondary sex ratio in the offspring generation \( P \). The sons of a \( P_i \) female will produce \( P_i k^2 ((1-P)N/PN) \) grand offspring carrying gene encoding strategy \( P_i \). This female will also have \( (1-P_i)k \) daughters, which will produce \( k^2 (1-P_i) \) grand offspring. Thus, the total number of grand offspring carrying the gene encoding the strategy \( P_i \) equals:

\[
R(P_i, P) = \frac{1}{4} P_i k^2 \left( \frac{1-P_i}{P} \right)^N + \frac{1}{2} k^2 (1-P_i) = \frac{1}{4} k^2 (1-P) \left( \frac{P_i}{P} + \frac{1-P_i}{1-P} \right)
\]

Let us assume for simplicity that \( k=2 \); then the right-hand side of (1) reads as

\[
F(P_i, P) = \frac{P_i}{P} + 1 - P_i
\]

The function \( F(P_i, P) \) can be used to compare the numbers of grand offspring of females with different individual strategies. \( P = \sum P_i f_i \), therefore this function can be presented as \( F(P_i, \sum f_i P_i) \) whose arguments are the individual strategy and the average strategy in the population. Similar reasoning was introduced by Dusing and function (3) was obtained by Shaw and Mohler (1953). This function is a well-defined payoff function of an evolutionary game called the sex ratio game and it is a basic example of a nonlinear
fitness function in evolutionary game theory (Bomze and Potscher, 1989; Cressman, 1992; Hofbauer and Sigmund, 1988, 1990; Weibull, 1995; Maynard Smith, 1982). Therefore, function (3) we will call the Dusing–Fisher–Shaw–Mohler function (DFSM function) or the sex ratio game. When \( P = 0.5 \), all strategies will obtain equal payoffs, and hence, this is an equilibrium state. When this equilibrium is disturbed to the advantage of one sex, it is more profitable to produce more individuals of the less numerous sex. However, the above model is important not only for ESS theory. This is a null model for sex allocation theory (Charnov, 1982; West, 2009). In general, this is a basic canonical example of strategic reasoning in mathematical biology. Here we should explain the meaning of the description as “strategic”. Oster and Rocklin (1979) distinguish between “strategic” and “genetic” models. Strategic models ignore genetics and treat adaptation only at the phenotypic level. Phenotypic traits are strategies of individuals that should maximize gain defined as “fitness”. The problem is that these models, focused on individuals, can be oversimplified by using non-rigorous fitness descriptions. This may cause internal inconsistency hidden in the structure of the model. Genetic models are focused on tracking particular gene frequencies, the phenotypic level is ignored. Except for a few attempts to combine both perspectives (see for example (Bomze et al., 1983)), this dualism still exists. The sex ratio problem is a good illustration of the above model classification.

The genetic approach to sex ratio modeling can be found for example in Eshel and Feldman (1982a,b). The most rigorous analysis of this approach can be found in "Theoretical Studies on Sex Ratio Evolution" by Karlin and Lessard (1986). In the preface of this book, following statement can be found that is similar to Oster and Rocklins thoughts: "...there are two main approaches (not mutually exclusive) to understanding sex ratios under various systems of sex determination. One emphasizes the optimization and adaptive functions of sex allocation at phenotypic level, and the other emphasizes the consequences of genetic sex determination mechanisms." The genetic approach produces quantitative trajectories of gene frequencies. Those changes are driven by a mechanism conceptualized by Dusing and Fisher which should operate at some level of this process. However, rigorous mathematical relationship between Dusing and Fisher’s reasoning and population genetic models is not clear. These are the basic concepts underlying the explanation of sex ratio self-regulation in terms of Darwinian adaptation. In this paper, we will analyze relationships between these two perspectives on the level of mathematical notions, to show that they are closely related. We want to improve game theoretic methods to make them able to produce models with the level of detail comparable to population genetics. However, our main goal is to achieve a full mechanistic explanation of phenomena underlying the dynamics of sex ratio self-regulation.

1.1. Predictions of the classical theory

If we assume the point of view of the classical ESS approach, that is

(i) the initial population is homogeneous and consists of clones with a strategy \( P_1 \), and \( P = P_1 \),

(ii) then some proportion of mutants, \( P_2 \), appear, which try to invade this population.

In this situation, an individual strategy \( P_1 = 0.5 \) is an evolutionarily stable strategy (ESS). Any other strategies cannot successfully invade a population consisting of \( P_1 = 0.5 \) individuals. However, when \( P_1 < 0.5 < P_2 \) or \( P_1 > 0.5 > P_2 \), there arises a non-homogeneous evolutionarily stable state with \( P = 0.5 \), as confirmed by numerical solutions of the population genetics dynamic model (Seger and Stubblefield, 2002). Recall that the homogeneity assumption is now violated. This model describes the dynamics of selection between two alleles, \( P_1 \) and \( P_2 \), that encode different individual sex ratios. It has been shown that an analytical solution of the limited case, in which the fraction of invading mutants, \( P_2 \), goes to zero, is fully compatible with the results that are produced by the static DFSM fitness measure (Bomze and Potscher, 1989; Karlin and Lessard, 1986; Seger and Stubblefield, 2002). However, there is an intriguing inconsistency between strategic and genetic sex ratio models. Genetic models produce stability characterizations describing the composition of the female and male subpopulations (Karlin and Lessard, 1986; Eshel and Feldman, 1982a,b). This suggests that the female subpopulation (described by the primary sex ratio) can be unstable to a disturbance of the male subpopulation. DFSM model (3) cannot produce this type of prediction, because the underlying reasoning is limited to females as strategic agents.

2. Formulation of the alternative model

In the new formulation of the problem, we depart from the DFSM paradigm, which assumes that the number of grand offspring is a fitness measure. The new model explicitly considers the following aspects that are not emphasized enough or disregarded in classical DFSM theory: (i) the influence of male carriers carrying non-expressed sex ratio genes, so that we approach the problem from the perspective of a gene, not from that of a female producing offspring; (ii) we emphasize the role of diversity among individual strategies (non-homogeneous populations); (iii) we consider feedbacks between population parameters; (iv) we consider dynamic individual strategies constituting the reaction norms conditional on secondary sex ratio. These conditions require explicitly considering both primary and secondary sex ratios in the model. Fitness is described by the number of offspring (not grand offspring) that are produced during some time interval by all carriers in the population of a gene encoding the same strategy. Female is the strategic agent (“player”) but male individuals are considered as passive (“non-strategic”) carriers of a gene encoding female individual strategy. Therefore, the new model is similar to the population genetics approach (Eshel and Feldman, 1982a,b; Karlin and Lessard, 1986). However, we will use game theoretic terminology and focus explicitly on payoff analysis. The number of newly produced individuals may be treated as the per capita growth rate, because mortality of adult individuals does not affect the form of the replicator equations (according to its additive character; see Argasinski and Kozlowski (2008)). The new model represents a two-population (males and females) dynamic evolutionary game (Argasinski, 2006). This is a classical game theoretic approach (see Appendix A) that is extended by the possibility of the decomposition of the entire population into subpopulations (more details on this method are presented in Appendix B). Multi-population replicator dynamics allow modeling of the dynamics of frequencies of strategies inside arbitrarily chosen subpopulations and allow changes of relative sizes between subpopulations, in our case, of males and females.

Let us assume that there are \( y \) males and \( x \) females in the population. Each individual is the carrier of a gene that encodes an individual strategy, i.e., the sex ratio of offspring produced by a female carrying such a gene. In the entire population, there are \( u \) individual strategies described by \( P_1, \ldots, P_u \). For the \( i \)-th strategy, there are \( y_i \) male carriers and \( x_i \) female carriers (then \( x = \sum x_i \) and \( y = \sum y_i \)). Each female produces \( k \) descendants during a given time interval. The population is panmictic. The population state can then be described by the following parameters:

\[
\begin{align*}
\bar{f}_i & = \frac{y_i}{y} \quad \text{fraction of females with strategy } P_i \in \{f_1, \ldots, f_u\} \in A^u \quad \text{where } \sum f_i = 1. \\
\bar{m}_i & = \frac{x_i}{x} \quad \text{fraction of males with strategy } P_i \in \{m_1, \ldots, m_u\} \in A^u \quad \text{where } \sum m_i = 1.
\end{align*}
\]

\[ P = \frac{x}{y+x} \] fraction of males in the population (secondary sex ratio).
\[ \gamma = \frac{1-P}{P} \] mean number of females per single male individual (an auxiliary parameter).
\[ N = y + x \] the number of all individuals in the population (scaling parameter see Argasinski (2006)).

There are \( x = (1-P)/N \) females in the whole population. A single \( P_i \) female produces \( Pj \) sons. The primary sex ratio is described as in the previous section as (1), however here it can be more rigorously derived:

\[ P_{pr} = \sum P_i \frac{x_i P_j}{(1-P)N} P_i = \sum \frac{x_i}{x} P_i = \sum f_i P_i \]

The space of population states is \( A^m \times A^n \times [0.1] \). This space contains a simplex of relative frequencies of strategies in the female subpopulation (according to the general notation from Appendix B \( \sigma_i^f = f_i \)), a simplex of the male subpopulation \( (\sigma_i^m = m_i) \) and the proportion between sizes of both subpopulations \( (\gamma = P) \) (Fig. 1). Let us initially assume that individual strategies, \( P_i \), are fixed and described by the sex ratio in a brood of a females that carry the allele for this strategy. The value of each possible individual strategy may be interpreted as the probability that a single offspring produced will be male. Thus, a set of possible individual strategies is the unit interval \([0,1]\) that is the basic, simplest form of a set of individual strategies. Later in this paper, this structure will be extended to a function space describing the dynamic strategies adjusting their expression according to actual population state (reaction norms). Fitness functions for males and females can be defined on this set. However, the fitness functions will be derived, in a sense, at the statistical mechanics level. We assume that production of a single offspring is a basic statistical event, and we then average these events over the population (for a detailed derivation see Appendix C). We will explicitly consider males as passive carriers of a gene and a phenomenon that can be termed “fitness exchange”. This means that an individual not only reproduces itself (female carriers produce other female carriers) but its reproductive activity can contribute to the fitness of other type individuals (female carriers produce male carriers and male carriers transfer genes to female carriers). Then, the number of juvenile individuals produced per capita (payoff) is counted in the following way (\( x_i \) and \( y_i \) are the numbers of female and male carriers of strategy \( P_i \)). The payoff of a male individual is

\[ W_m = W_{mm} + \frac{x_i}{y_i} W_{mf} \] (4)

where \( W_{mm} \) represents the number of his sons that carry alleles for the same strategy, and \( W_{mf} \) represents the mean number of sons of female carriers of the same strategy that inherit the strategy from their mothers. The payoff of a female is

\[ W_f = W_{ff} + \frac{y_i}{x_i} W_{mf} \] (5)

where \( W_f \) represents the number of her daughters that carry the same strategy and \( W_{mf} \) represents the mean number of daughters of a male carrier of the same strategy that inherit the strategy from their fathers (see Appendix C).

Fitness functions describing the mean per capita growth rates (for example: the number of new females per single adult female which carries the same strategy) will be as follows.

The payoff function of males with strategy \( P_i \) is

\[ W_m(P_i, P, f, m) = k \left( 1-P \right) \left( \sum f_i P_j + \frac{f_i}{m_i} P_i \right) \] (6)

The payoff function of females with strategy \( P_i \) is

\[ W_f(P_i, P, f, m) = k \left( 1-P \right) \left( \sum f_i P_j \right) \] (7)

Then, the mean fitness functions will be as follows (for a derivation see Appendix D):

\[ \overline{W}_m(P, f, m) = k \Gamma P_{pr} = k \frac{P}{\sum f_i P_j} \] the mean fitness of the male subpopulation,

\[ \overline{W}_f(P, f, m) = k (1-P) \] the mean fitness of the female subpopulation,

\[ \overline{W}(P, f, m) = k (1-P) \] the mean fitness of the whole population.

The mean fitness of the whole population is proportional to the mean number of offspring produced by a single female \( k \) multiplied by the proportion of adult females in the population \( 1-P \). The mean fitness in a female subpopulation is proportional to the mean number of offspring produced by a single female, \( k \), multiplied by the mean proportion of female newborns in the brood of a single female, \( 1-\sum f_i P_i \) (1 minus the primary sex ratio). The mean fitness in the male subpopulation is proportional to the mean number of male newborns in the brood of a single female, \( k \sum f_i P_j \), multiplied by the coefficient \( I = (1-P)/P \) that describes the number of females per male individual. Now, the replicator equations can be formulated (see Appendix E):

\[ \dot{f}_i = f_i (W_f(P_i, P, f, m) - \overline{W}_f(P, f, m)) \] for \( i = 1, \ldots, u-1 \)

\[ \dot{m}_i = m_i (W_m(P_i, P, f, m) - \overline{W}_m(P, f, m)) \] for \( i = 1, \ldots, u-1 \)

\[ \dot{P} = P (\overline{W}_m(P, f, m) - \overline{W}_m(P, f, m)) \]

In effect, we obtain the following system of differential equations:

\[ \dot{f}_i = k \left( f_i \left( 1-P \right) + \frac{m_i}{2} \left( 1-\sum f_i P_j \right) \right) \] (8)

\[ \dot{m}_i = k \left( 1-P \right) \left( f_i P_j - m_i \sum f_j P_j \right) \] for \( i = 1, \ldots, u-1 \)

\[ \dot{P} = k (1-P) \left( \sum f_i P_j - P \right) \] (10)

Now, let us analyze the behavior of the replicator dynamics.
2.1. The biological domain of the model

There are two biologically irrelevant states of this system. The first of these is the stationary point $P=1$ of Eq. (9). In this case, the relative size of the subpopulation of females is zero. The second is the case when $P$ approaches 0, and the right side of Eq. (9) goes to infinity. This state occurs when the relative size of the subpopulation of males is zero. An infinite value is a mathematical artifact caused by the assumption that the number of females that can be inseminated by a single male individual is unlimited. Both of these states are repellers of the dynamics, in effect they will not affect the biological predictions of the new model. Also, cases where the primary sex ratio $\sum f_i P_j$ is equal to 0 or 1 can be excluded from further analysis. The case $\sum f_i P_j = 0$ is unstable, and its trajectory escapes to the interior of the strategy simplex if there is any variety of strategies among males. The case $\sum f_i P_j = 1$ leads to evolutionary suicide because the population converges to the state consisting of the carriers of the strategy that produce only males. Therefore, analysis of this system can be limited to the values of the primary sex ratio $\sum f_i P_j$ and the secondary sex ratio $P$ from the interior of the unit interval $(0,1)$.

2.2. Biologically relevant trajectories

From equations for the secondary sex ratio (10) and the state of the male subpopulation (9), we obtain (forward arrow means: converges to under replicator equation):

$$P \rightarrow \sum_j f_j P_j \quad \text{for } P < 1$$

and the current value of $P$ affects the pace of convergence of the dynamics determined by the value of the coefficient $(1-P)$:

$$m_i \rightarrow f_i \frac{P_i}{\sum_j f_j P_j} \quad \text{for } P < 1$$

and the current value of $P$ affects the pace of convergence of the dynamics by the average number of partners of a single male, described by the coefficient $I = (1-P)/P$. In effect, we obtain two stable equilibrium conditions:

(i) equality of primary and secondary sex ratios

$$P = \sum_j f_j P_j$$

and

(ii) the equilibrium condition for the male subpopulation that we term male subpopulation equilibrium (MSE condition)

$$f_i P_i = m_i \sum_j f_j P_j$$

This condition defines a unique stable state of the male subpopulation

$$m_{\text{MSE}} = \left[ \frac{P_1}{\sum j f_j P_j} \cdot \ldots \cdot \frac{P_u}{\sum j f_j P_j} \right]$$

This condition is termed equilibrium, because it is equivalent to the Nash equilibrium among males. From the dynamical systems point of view the MSE condition characterizes a manifold attracting the trajectories. The MSE state is conditional on the current state of the female subpopulation, $f$, and the values of individual strategies, $P_i$. The dynamics of the system are driven by changes in the primary sex ratio $\sum f_i P_j$. The parameter $P$ (the secondary sex ratio) affects the pace of convergence of equations on $m_i$ and $P$. Let us now examine trajectories of convergence in the female subpopulation.

**Lemma 1.** If MSE condition (12) is satisfied for all $i$, then the equations for the female subpopulation have the form:

$$f_i = k f_i \left( \frac{1}{2} \sum_j f_j P_j \right) \left( \frac{P_i}{\sum f_j P_j} - 1 \right) \quad \text{for } i = (1, \ldots, u-1)$$

and the evolution of parameters $f_i$ is as follows:

- $f_i$ increases when $\sum f_j P_j < 1/2$ and $\sum f_j P_j < P_i$ or $\sum f_j P_j > 1/2$ and $\sum f_j P_j > P_i$.
- $f_i$ decreases when $P_i < \sum f_j P_j < 1/2$ or $P_i > \sum f_j P_j > 1/2$.
- $f_i$ is stable when $f_i = 0$ or $f_i = 1$ or $P_i = \sum f_j P_j$.

Then we have a stable state for the entire female subpopulation when $\sum f_j P_j = 0.5$ (equations for all $i$’s are at their stable points).

For a proof, see Appendix F. **Lemma 1** shows that, when MSE condition (12) is satisfied, frequencies of females with strategies producing more (less) offspring with the rarer sex than the mean female strategy will increase (decrease). The form of the female population equations suggests that in the very close neighborhood of the MSE state there is a qualitative change of behavior of the system. After reaching the neighborhood of the MSE manifold, the dynamics are driven by changes of the averaged female strategy $\sum f_i P_j$. This suggests that the speed of convergence is slower than the speed of convergence to the MSE state. Therefore, the form of equations suggests double-phased dynamics (quantitative change of behavior after reaching MSE neighborhood). Let us characterize the stationary states of the whole system that are biologically relevant according to the aforementioned characterizations for subpopulations.

**Lemma 2.** Nontrivial stationary points of this system are

(i) all monomorphic states for all strategies, a state assigned to the $i$-th strategy is described as follows:

$$f_i = m_i = 1 \quad \text{and} \quad P_{\mu} = P = P_i,$$

(ii) when they exist, polymorphic states for which the following conditions are satisfied are represented as follows:

$$m_i = 2P f_i \quad \text{for } i = (1, \ldots, u) \quad \text{and} \quad P_{\mu} = P = 0.5.$$

For proof, see Appendix G. Note that $m_i = 2P f_i$ means that the MSE condition is satisfied under $P_{\mu} = P = 0.5$. **Lemma 2** shows that monomorphic states consisting of individuals carrying the same gene encoding strategy $P_i$ are stationary. All polymorphic states for which the primary and secondary sex ratios are equal to 0.5 and for which the MSE condition is satisfied for all strategies are also stationary.

The right sides of replicator Eqs. (8)–(10) show several important aspects of sex ratio self-regulation. During the first phase of convergence the whole system converges to the MSE state and to equality between the primary and secondary sex ratio. This phase can be interpreted as convergence to the values determined by the strategic composition of the population, however the form of the equations suggests that during convergence the strategic composition may also change dramatically. The model shows the importance of the state of the male subpopulation and suggests that the primary sex ratio of 0.5 can be unstable for perturbations of the male subpopulation. Therefore the interpretation of the sex ratio of 0.5 as an ESS becomes problematic. These predictions are consistent with the predictions of population genetics models (Karlin and Lessard, 1986; Eshel and Feldman,
The parameter $P$ is attracted by the current value of the primary sex ratio and it affects the pace of convergence of the male subpopulation (Eq. (9)) and the pace of attraction of the secondary sex ratio by the primary sex ratio (Eq. (10)). After reaching the MSE state the second phase of convergence begins. During the second, slow phase, the female subpopulation converges to 0.5, the value of the averaged female strategy. These properties will now be demonstrated by numerical examples.

3. Numerical examples

3.1. Two competing strategies

The case of two competing strategies is compatible with the classical ESS approach when one strategy is a resident in the population, and the second strategy is a rare invading mutant. A strategy that is closer to 1/2 than the resident strategy will spread in the population, and the second strategy is a rare invading mutant. A classical ESS approach when one strategy is a resident in the population converges to 0.5, if this is possible, or converges to the monomorphic male subpopulation equilibrium, the second, slow phase of evolution commences. In this longer phase, the primary sex ratio converges to 0.5, if this is possible, or converges to the monomorphic state consisting of individuals with a strategy closest to 0.5, if both strategies are either lower or higher than 0.5. The trajectory of Eq. (9) of the male subpopulation simply converges to the neighborhood of the MSE, and follows it in the slow phase according to the changes in the primary sex ratio. The trajectories of Eq. (8) of the female subpopulation after transition to the slow phase behave according to the predictions from Lemma 1.

3.2. The case of three competing strategies

Now we consider the specific case of three competing strategies $P_1=0.2$, $P_2=0.8$ and $P_3=0.5$. Initial conditions are $f_1=f_2=0.05$, $f_3=0.9$, $m_1=0.85$, $m_2=0.1$, $m_3=0.05$, which provide the equilibrium state of the female subpopulation (primary sex ratio equals 0.5), and MSE condition (12) is not satisfied (i.e., the male subpopulation is far from the equilibrium state). These initial conditions indicate that female Eq. (8) are in equilibrium and male Eq. (9) are not. Additionally, we assume that $P=0.2$. The form of the replicator equations clearly show that the value of $P$ can affect only the pace of convergence and plays a minor role in the dynamics of the system. An unstable initial value of Eq. (10) allows for the observation of the pace of convergence of parameter $P$ and comparison with the pace of convergence to the MSE.

Fig. 3(a) shows that the trajectory of the male subpopulation rapidly converges to the MSE and then follows the changes of the primary sex ratio to maintain the MSE. Fig. 3(b) shows that the female subpopulation is knocked down from the stable state and rapidly converges to the MSE. Afterward, it slowly converges to a stable state in which the primary sex ratio equals 0.5. As shown, perturbation of the MSE has very serious effects for the population and leads to deviation of the primary sex ratio. Thus the primary sex ratio is unstable for MSE perturbations.

Fig. 3(c) shows trajectories of the relative frequencies of all types of individuals, according to their sex and the strategy that they carry, without division into subpopulations of males and females (in so-called metasimplex coordinates, see Argasinski (2006)). Frequencies of females that carry the i-th strategy are $P_{fi}$, frequencies of males are $P_{mi}$ (see Appendix B and Argasinski (2006)).

Fig. 3(d) shows a plot of the deviations of the mean subpopulation from the MSE for all strategies $(f_1-f_2, f_2-f_3, f_3-f_1)$ and the deviation of the secondary sex ratio $P$ from the current value of the primary sex ratio. The border between the fast and slow phases is quite sharp, and it correlates with the convergence to the neighborhood of the MSE. Fig. 3(e) shows trajectories of mean strategies of males and females and the trajectory of the secondary sex ratio during the rapid phase. The deviation of the primary sex ratio is smaller than the corresponding deviation of the female population state. This is due to the primary sex ratio being a linear combination of the values of individual strategies.

The phase portraits (Fig. 4) of the system, in which time trajectories are depicted in Fig. 3, are also noteworthy. The trajectory of the female subpopulation has a cusp corresponding to the time point when the transition between the rapid and the slow phase occurs after reaching the MSE manifold. In addition, the trajectory of the male subpopulation is non-smooth at the point corresponding to this time point. Both the female (8) and male (9) subsystems of equations can change the direction of convergence during the transition from the rapid phase into the slow phase (for example Fig. 4).

Numerical examples show that trajectories may be very complex, but asymptotic behavior seems to be compatible with suggestions of the population genetics models. The sex ratio of 0.5 is unstable for perturbations of the MSE (disturbance of the male subpopulation). A question then arises about the relationship of the new model with the classical sex ratio game: what is the role of male individuals in sex ratio self-regulation? Recall that the classic sex ratio game is comparing the reproductive success of females measured in the number of grandoffspring. Can we interpret the DFSM function as a game theoretic payoff describing the malthusian growth rate? This problem will be examined in the next section, where the strategy maximizing DFSM payoff will be introduced to the new model.

4. A dynamic reaction norm

In this section, the new model will be extended to allow for dynamic reaction norms. By a dynamic reaction norm, we
understand a conditional strategy that can adjust its expression according to the current state of the population. In the case of our model, these will be strategies that will determine different sex ratios among offspring for different values of the secondary sex ratio $P$. Let us examine what the new model will reveal for the dynamic strategy, which is designed to maximize the classical DFSM fitness measure (3). Our model must be further extended, because the new strategy should react based on the current value of a sex ratio (assuming equality of primary and secondary sex ratios). Now, the set of possible conditional individual strategies $P(P)$ is a function space $\{P : (0.1) \rightarrow (0.1)\}$. The fixed strategies are simple constant functions with the same value for all values of $P$, therefore in the classical formulation we were limited to the subspace containing these functions. The classical fitness function

Fig. 3. A case of three competing strategies. The difference between the rapid and slow phases of convergence is evident in the trajectories of the male subpopulation (a), the female subpopulation (b) and on the plot of the relative frequencies of the whole population (c). Panel (d) shows deviations from male subpopulation equilibrium and deviation of the secondary sex ratio from the value of the primary sex ratio. Clearly visible is the correlation between the transition from rapid to slow phases and convergence to MSE. Panel (e) shows the trajectories of the mean strategies of male and female subpopulations and the trajectory of the secondary sex ratio $P$ during the rapid phase of convergence.
is maximized if

\[
P_i(P) = \begin{cases} 
0 & \text{for } P > \frac{1}{2} \\
1 & \text{for } P < \frac{1}{2} 
\end{cases}
\]  

(14)

In other words, Eq. (14) describes that the female carrier produces more of the sex that is currently in the minority. This function can be approximated by the continuous function

\[
P_i(P) = 0.5 - \frac{\arctan(100(P-0.5))}{\pi}
\]  

(15)

shown in Fig. 5 (arbitrary multiplication by 100 squeezes function (15) to approximate (14)). This is a continuous smooth function, therefore it does not cause numerical problems. This strategy always dominates the fixed strategies according to the classical fitness measure, if the sex ratio is perturbed. Numerical solutions of the replicator equations show that the reaction norm increases its relative frequency and can outcompete the fixed strategies. Fig. 6 shows an example of trajectories of gene frequencies \(P_i + (1-P_j)\) calculated from solutions of the replicator equations (according to the method from Appendix B; see also Argasinski (2006)). As we can see in Fig. 6, the relative frequency of the dynamic reaction norm, weakly dominating all fixed strategies according to the sex ratio game (DFSM function (3)), decreases at the beginning. This suggests the influence of male carriers of a sex ratio gene on changes of a gene pool and that interpretation of the FSM function as a Malthusian growth rate is problematic. It is noteworthy that the point of transition to the phase of increase correlates with the moment when the MSE is reached (the end of the rapid phase). This coincidence suggests a relationship between these two phenomena. However, the mechanistic nature of this relationship is still unclear.

5. Summary of the results obtained from the new model

5.1. The role of primary and secondary sex ratios

The new model is compatible with the predictions of classical theory. The system is robust against perturbations of the secondary sex ratio that affects only the pace of convergence (the form of Eqs. (8) and (9)). Perturbation of the parameter \(P\) (the secondary sex ratio) only changes the relative sizes of the male and female subpopulations, but the frequencies of strategies within these subpopulations remain stable. The value of \(P\) simply converges to the value of the primary sex ratio, equal to 0.5. In effect, the system rapidly converges exactly to its initial state before perturbation. The crucial role is played by the primary sex ratio. Surprisingly, the primary sex ratio is very sensitive to perturbations of the male subpopulation equilibrium (MSE).

5.2. The role of male carriers of sex ratio strategies (male subpopulation equilibrium)

The MSE phenomenon is related to the fact that when all individuals are produced by females that are residents of the population (i.e., no migration or other perturbations of population state occur), then the male subpopulation rapidly converges to a
unique state \( m^{\text{MSE}} \) given by

\[
m^{\text{MSE}} = \left[ \frac{P_1}{\sum_j P_j}, \ldots, \frac{P_q}{\sum_j P_j} \right]
\]

(13)

The mechanistic nature of the MSE phenomenon is still unclear, but the results obtained from this model show that males are involved in the process of self-regulation of the primary sex ratio (and, in effect, of the secondary sex ratio). The role of male carriers of unexpressed sex ratio genes is extremely important. Deviation from the results obtained from this model show that males are involved in the process of self-regulation of the primary sex ratio (and, in effect, of the secondary sex ratio). Therefore, the state of the male subpopulation is an important element of the evolutionarily stable state of the population as well as \( m^{\text{MSE}} \) of the male subpopulation can change the state of the female subpopulation and, subsequently, may affect the primary and secondary sex ratios. Therefore the sex ratio is unstable for perturbations of the state of the male subpopulation.

5.3. Double phased dynamics

The new model shows that dynamics of this system are very complex and are divided into two phases. During transition between phases, system can qualitatively change the behavior (e.g. change of the direction and pace of convergence). The first, rapid phase is related to convergence to the MSE and convergence of the secondary sex ratio to the value of the primary sex ratio, whereas the second slow phase is responsible for convergence of the primary sex ratio to 0.5. Rapid phase occurs when the initial state of the population is not an effect of expression of the current strategic composition. However, the new model clearly shows that the rapid phase is not a simple substitution of the initial state by the new state adjusted by the initial strategic profile. It is shown that perturbation of the frequencies among adult individuals can affect the primary sex ratio, which describes the strategic composition of the population. Therefore, genetic polymorphism is not robust against ecological factors affecting the population. The primary sex ratio may depart from the 0.5 value due to perturbation of the MSE. In addition, the example of a temporary decrease in the relative frequency of the dynamic reaction norm suggests that roles of males as passive carriers of a gene and “fitness exchange” mechanism are important. In the classical sex ratio game (3) only females are considered as strategic agents. The new approach shows that males can affect the reproductive effort of females. Therefore, classical theory considers males only as “sons”, however the new model suggests that “dad” can also matter. When the MSE condition is satisfied, the new model is fully compatible with classical theory. However, the relationship between the new model and the DFSM reasoning needs more rigorous analysis. The new model is focused on the global dynamics of the system, not on the reproductive success of individual strategies. To elucidate these relationships, we must focus on the subject of the selection of individual strategies, which will be carried out in a subsequent paper. In summary, the new model puts forward a few questions that are discussed in Section 5.4.

5.4. Open questions

Some results produced by the new model are surprising, and these will be analyzed in later papers. There are some open problems arising from the results obtained. The first question is about the status of the sex ratio models in ESS theory. In this paper we obtain new results which cannot be generated by classical game theoretic models of sex ratio evolution. Classical results that can be found in game theoretic textbooks say that the sex ratio of 0.5 is evolutionarily stable. However, results produced by the new model show that the sex ratio of 0.5 is unstable to perturbations of the male subpopulation state and the unique state of the male subpopulation conditional on the current value of the primary sex ratio. Therefore, the state of the male subpopulation is an important element of the evolutionarily stable state of the population as in population genetics models (Karlin and Lessard, 1986; Eshel and Feldman, 1982a,b). This means that we need new characterizations of evolutionary stability to describe this system. This leads on to the second question about the relationship between the DFSM function and the new model. This function is a good formalization of Fisher’s intuition related to different reproductive values. It is a good mechanistic explanation of this mechanism showing that females producing offspring in different sex ratios obtain different reproductive success measured in grand-offspring. However, the underlying reasoning disregards the influence of passive male carriers (“dads”) in the mothers’ generation via the “fitness exchange” mechanism. Males are explicitly considered only as sons of “mom”. The new model suggests that this influence can be important (decrease of the reaction norm during rapid phase). In effect the interpretation of the DFSM function as a game theoretic payoff describing the Malthusian growth rate becomes problematic. This is an important question because the sex ratio game is a basic example of nonlinear payoffs in evolutionary game theory. Therefore, we need to examine the process of selection of genes encoding individual strategies. This will be the focus of a subsequent paper, where new model will be transformed by a change of coordinates (by the method from Appendix B) to describe the evolution of a gene pool.

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Appendix A

Methods

In the most general sense, the structure of an evolutionary game can be described as the set of possible strategies \( \Omega \) and payoff functions \( W(P, \sigma) \), where \( P \) is an individual’s strategy (an element of set \( \Omega \)), and \( \sigma \) is the population state described by the distribution of individual strategies in the population. When a population with a finite number of individual strategies is considered, then a system of differential equations termed replicator dynamics can be defined. It describes changes of the population state in time and can be derived in the following way. Assume that we have a finite set of arbitrary chosen strategies \( \{P_1, \ldots, P_n\} \) where \( P_i \in \Omega \), then the population state will be described by a vector of relative frequencies of these strategies

\[
\sigma_i(t) = \frac{N_i(t)}{N(t)}
\]

(14)

where \( N(t) \) is the number of individuals with the \( i \)-th strategy, and

\[
N(t) = \sum_{i=1}^{u} N_i(t)
\]

(15)

Assume that the population growth is given by equation:

\[
\dot{N}_i(t) = N_i(t)W(P_i, \sigma(t))
\]

(16)

From this assumption, we can derive a system of ordinary differential equations (see e.g., Cressman, 1992) defined on strategy...
simplex $\Delta^{b}$:

$$\sigma_i(t) = \sigma_i(t)[W(P_i, \sigma(t)) - \overline{W}(\sigma(t))] \text{ for } i = 1, \ldots, u - 1$$

(17)

where $\overline{W}(\sigma) = \sum_{i=1}^{u} \sigma_i W(P_i, \sigma)$

These equations are now a basic dynamic approach in evolutionary game theory (Bomze and Potschker, 1989; Cressman, 1992; Hofbauer and Sigmund, 1988, 1990; Weibull, 1995; Maynard Smith, 1982). The above formulation cannot be used to model a situation in which a division of individuals into some subclasses (different species or sexes) exists because individuals from separate specific subclasses may use different sets of strategies, or they may obtain different payoffs when using the same strategy. Therefore, the ability to break down the problem into subpopulations will make the analysis of this system easier and more fruitful.

Appendix B

Change of coordinates in the space of population states

Assume that we want to break down an entire population into $z$ subgroups. Define $d^* = [d_1^*, \ldots, d_z^*]$ as a vector of indices of strategies exhibited by individuals from the $i$-th subgroup ($d_i^* \in [1, \ldots, u]$, $u_i$ the number of strategies in the $i$-th subgroup). For example, the notation $d^* = [1,3,5]$ means that in the second subgroup, there are individuals with strategies 1, 3 and 5. Every strategy should belong to a single unique subgroup (and cannot belong to two). Then, according to Argasinski (2006) using the following change of coordinates:

$$\sigma^i = [\sigma_1^i, \ldots, \sigma_u^i] = \left[ \frac{\sigma_{d_1^i}}{\sum_{j=1}^{u} \sigma_{d_j^i}}, \ldots, \frac{\sigma_{d_{u_i}^i}}{\sum_{j=1}^{u} \sigma_{d_j^i}} \right] \text{ for } i = 1, \ldots, z$$

(18)

we obtain the distribution of relative frequencies of strategies in the $i$-th subpopulation. The distribution of proportions between subpopulations has the form:

$$\gamma = [\gamma_1, \ldots, \gamma_z] = \left[ \sum_{i=1}^{u_1} \sigma_{d_1^i}, \ldots, \sum_{i=1}^{u_z} \sigma_{d_z^i} \right]$$

(19)

where $\gamma_i$ is the proportion of the $i$-th subpopulation. Every decomposition into subpopulations can be reduced again to a single population model by the opposite change of coordinates $\sigma(\gamma, \sigma_1^i, \ldots, \sigma_z^i)$ where

$$\sigma^i = \gamma_1 \sigma_1^i \text{ (20)}$$

Note that we can break down an entire population into $z$ subpopulations. When we apply the above transformations to replicator equations, we obtain a set of equations that describes the dynamics inside subpopulations (intraspecific dynamics, see Argasinski (2006)), which has the form:

$$\dot{\sigma}_i^j = \sigma_i^j [W_i^j - \overline{W}]$$

where $W_i^j$ is the fitness of the $j$-th strategy in the $i$-th subpopulation and $\overline{W}$ is the mean fitness in the $i$-th subpopulation, and a system that describes changes of relative sizes among subpopulations (interspecific dynamics) is

$$\gamma_i = \gamma_1 \sigma_1^j \text{ (21)}$$

(21)

where $\overline{W}(\sigma) = \sum_{i=1}^{n} \sigma_i W(P_i, \sigma(\gamma, \sigma_1^i, \ldots, \sigma_z^i))$ is the mean fitness in the $s$-th subpopulation. The argument of a fitness function is a set of relative frequencies of all individuals $\sigma$ (without division into subpopulations), therefore the opposite change of coordinates $\sigma(\gamma, \sigma_1^i, \ldots, \sigma_z^i)$ (20) should be applied (Argasinski, 2006). In practical applications of this method to the modeling of biological problems, replicator equations can be defined for broken down populations. This break down will simplify the formulation of the model because, when strategies are initially assigned to subpopulations, there is no need to change their indices. The choice of subpopulations is arbitrary and depends on the biological assumptions underlying the analyzed problem. The entire population may be divided into two competing subpopulations of carriers and parasites or predators and prey. It may also be divided into two subpopulations of males and females, in which case interspecific dynamics will describe the evolution of the secondary sex ratio, and intraspecific dynamics will describe changes of frequencies of strategies inside male and female subpopulations. The entire population can be divided into more than two subpopulations. The subpopulations can be divided into sub-subpopulations, and the entire population may be transformed into a complex multilevel cluster structure. However, all of these structures are equivalent to a single population replicator dynamics model.

Appendix C

Derivation of the fitness functions

Production of a single offspring contains the following statistically independent phases:

a) drawing of a mother with a probability proportional to $f_i = x_i/k$ for a female with strategy $P_i$.

b) drawing of a father with probability proportional to $m_i = y_i/y$ for a male which is a carrier of a strategy $P_i$.

c) drawing of the sex of a newly produced offspring with a probability proportional to the expression of the mother’s individual strategy $P_i$.

d) drawing of inheritance of an individual strategy gene from the mother or the father with a probability of 0.5 (haploid inheritance).

Production of a single offspring is a single trial from a binomial distribution, where success is treated as the transfer of a sex ratio gene to an individual of a given sex. Assume that indices describe the sex of parent and offspring individuals (for example $m$ means daughters of males, $f$ means daughters of females). Then the probabilities of a single success (transfer of a carried gene) in the case of a female parent are

$$\theta_m = 0.5(1 - P_i)$$

where 0.5 is the probability of transfer of a carried gene and $(1 - P_i)$ is the probability of birth of a daughter.

$$\theta_m = 0.5P_i$$

for the analogous probability of a gene transfer to a son.
In the case of the male individual, probabilities of the transfer of a carried gene will be related to the conditional probabilities of drawing a mother with a given individual strategy:

\[ \theta_{mn} = 0.5 \left( \sum_j f_j P_j \right), \]

\[ \theta_{mf} = 0.5 \left( \sum_j f_j (1 - P_j) \right), \]

the probability of the transfer of a carried gene to the son, and the probability of transfer of a carried gene to a daughter. Nevertheless, in this paper, we limit ourselves to frequency explicit consideration when we extend our model to density dependence. Then a male individual can consider only the mean value that describes the per capita growth rate. Therefore, the number of offspring can be described by the sum of a mean value and the stochastic fluctuation factor (Gaussian noise with mean 0 and appropriate variance). The Gaussian factor describes the effects caused by the finiteness of a population. When we assume a technically infinite population, we can consider only the mean value that describes the per capita number of offspring. The Gaussian fluctuation factor should be explicitly considered when we extend our model to density dependence. Nevertheless, in this paper, we limit ourselves to frequency dependence. Then a male individual \( P_i \) will have

\[ W_{mm} = \theta_{mm} \frac{\lambda_m}{y_i} = 0.5 \left( \sum_j f_j P_j \right) \frac{xk}{y} \text{ sons} \]

(23)

\[ W_{mf} = \theta_{mf} \frac{\lambda_m}{y_i} = 0.5 \left( \sum_j f_j (1 - P_j) \right) \frac{xk}{y} \text{ daughters} \]

(24)

that inherit his strategy (mean value of per capita growth rate). Analogously a female \( P_i \) will have

\[ W_{fm} = \theta_{fm} \frac{\lambda_f}{x_i} = 0.5 P_i k \text{ sons} \]

(25)

\[ W_{ff} = \theta_{ff} \frac{\lambda_f}{x_i} = 0.5 (1 - P_i) k \text{ daughters} \]

(26)

that inherit her strategy. Now, when we equally distribute daughters sired by male carriers of a \( P_i \) gene among females with strategy \( P_j \) and sons of those females among male carriers of a \( P_j \) gene. In that way we can calculate the average per capita growth rates for females and males carrying \( P_i \) gene. In effect, we will obtain payoff functions:

\[ W_m = W_{mm} + \frac{x_i}{y_i} W_{fm} = 0.5 \left( \sum_j f_j P_j \right) \frac{xk}{y} + \frac{x_i}{y_i} 0.5 P_i k \]

\[ = 0.5 k \left( \frac{X_i}{y_i} \sum_j f_j P_j \right) \frac{xk}{y} \]

that describe the mean value of newly produced \( P_i \) males for one adult \( P_i \) male.

\[ W_f = W_{ff} + \frac{y_i}{x_i} W_{mf} = 0.5 (1 - P_i) k \cdot \frac{y_i}{x_i} 0.5 \left( \sum_j f_j (1 - P_j) \right) \frac{xk}{y} \]

\[ = 0.5 k \left( (1 - P_i) + \frac{y_i}{x_i} \sum_j f_j (1 - P_j) \right) \frac{xk}{y} \]

is the analogous value for females. Now, change the coordinates in above formulae to relative frequencies \( m_i, f_i \) and \( P \). Then \( x/y = 1-P/P \) and \( y_i/x_i = (m_i/f_i) (y_i/x_i) = (m_i/f_i) P/(1-P) \). Substitute these conditions into the payoff functions and we will obtain:

\[ W_{mf}(P_i, P, f, m) = k \frac{1-P}{2P} \left( \sum_j f_j P_j + \frac{f_j}{m_j} P \right) \]

−payoff function of males,

\[ W_f(P_i, P, f, m) = k \frac{1}{2} \left( (1 - P) + \frac{m_i}{f_i} \left( 1 - \sum_j f_j P_j \right) \right) \]

−payoff function of females.

The functions given above describe the number of offspring carrying the same gene of a male carrier of \( P_i \) and a female \( P_i \). In the case of continuous replicator dynamics, the fitness function should describe the per capita growth rate. Therefore, we should subtract 1 from the above formulae. However, replicator dynamics are independent of additive transformations of fitness functions, so we can pass over this step (see Argasinski and Kozlowski (2008)).

Appendix D

**Mean fitness functions**

Here we derive the mean fitness functions, which are necessary to formulate replicator equations. Since \( \sum_i m_i = 1 \), in the case of a male subpopulation we obtain:

\[ W_m(P, f, m) = \sum_i m_i W_{m}(P_i, P, f, m) \]

\[ = \sum_i m_i k \frac{1-P}{2P} \left( \sum_j f_j P_j + \frac{f_j}{m_j} P \right) \]

\[ = k \frac{1-P}{2P} \left( \sum_j f_j P_j + \sum_j f_j P_j \right) = k \frac{1-P}{P} \sum_j f_j P_j \]

In the case of a female subpopulation the mean fitness function will be

\[ W_f(P, f, m) = \sum_i f_i W_f(P_i, P, f, m) \]

\[ = \sum_i f_i k \frac{1}{2} \left( (1 - P) + \frac{m_i}{f_i} \left( 1 - \sum_j f_j P_j \right) \right) \]

\[ = k \frac{1}{2} \left( \left( 1 - \sum_i f_j P_j \right) + \sum_i m_i \left( 1 - \sum_j f_j P_j \right) \right) = k \left( 1 - \sum_j f_j P_j \right) \]

The mean fitness function of the whole population has the form:

\[ W(P, f, m) = p W_m(P, f, m) + (1-p) W_f(P, f, m) \]

\[ = pk \frac{1-P}{P} \left( \sum_j f_j P_j + \left( 1-P \right) k \left( 1 - \sum_i f_j P_j \right) \right) \]

\[ = k \left( 1 - P \right) \left( \sum_j f_j P_j + \left( 1 - \sum_i f_i P_i \right) \right) = k \left( 1 - P \right). \]
Appendix E

Replicator dynamics

The form of the replicator equations are
\[ f_i = f_i(W_i(P_i, P, f, m) - W_i(P, f, m)) \quad \text{for} \quad i = (1, \ldots, u-1) \]
\[ m_i = m_i(W_m(P_i, P, f, m) - W_m(P, f, m)) \quad \text{for} \quad i = (1, \ldots, u-1) \]
\[ P = P(W_m(P, f, m) - W(P, f, m)) \]

\[ f_i = f_i \left( k \left( 1 - P_i \right) + \frac{m_i}{f_i} \left( 1 - \sum_j f_j P_j \right) \right) - k \left( 1 - \sum_j f_j P_j \right) \]

\[ = kf_i \left( 1 - P_i \right) + \frac{m_i}{2f_i} \left( 1 - \sum_j f_j P_j \right) \]

\[ = k \left( f_i (1 - P_i) + \frac{m_i}{2f_i} - f_i \right) \left( 1 - \sum_j f_j P_j \right) \]

\[ m_i = m_i \left( 1 - P \left( \sum_j f_j P_j + f_i P_i \right) - k \frac{1 - P}{P} \sum_j f_j P_j \right) \]

\[ = k \left( 1 - P \right) m_i \left( 2 \sum_j f_j P_j + f_i P_i - \sum_j f_j P_j \right) \]

\[ = k \left( 1 - P \right) \frac{m_i}{2} \left( f_i P_i - \sum_j f_j P_j \right) \]

\[ P = P \left( \frac{k}{P} \left( \sum_j f_j P_j - k(1 - P) \right) \right) \]

Thus, we obtain the following set of equations:

\[ f_i = k \left( f_i (1 - P_i) + \frac{m_i}{2f_i} - f_i \right) \left( 1 - \sum_j f_j P_j \right) \]

\[ m_i = k \left( \frac{1}{2} \right) \left( f_i P_i - \sum_j f_j P_j \right) \]

\[ P = k(1 - P) \left( \sum_j f_j P_j - P \right) \]

Appendix F

Proof of Lemma 1. If we substitute the MSE condition (12) into Eq. (8), then we obtain:

\[ f_i = k \left( f_i (1 - P_i) + \left( f_i \frac{P_i}{2 \sum_j f_j P_j} - f_i \right) \left( 1 - \sum_j f_j P_j \right) \right) \]

\[ = kf_i \left( 1 - P_i \right) + \left( \frac{P_i}{2 \sum_j f_j P_j} - 1 \right) \left( 1 - \sum_j f_j P_j \right) \]

\[ = kf_i \left( \frac{P_i}{2 \sum_j f_j P_j} - 1 \right) \left( 1 - \sum_j f_j P_j \right) \]

Thus, \( f_i \) increases when \( 0.5 - \sum_j f_j P_j \left( \frac{P_i}{2 \sum_j f_j P_j} - 1 \right) > 0 \) that is \( \sum_j f_j P_j < 0.5 \) and \( \sum_j f_j P_j < P_i \). If \( \sum_j f_j P_j > 0.5 \) and \( \sum_j f_j P_j > P_i \),

Analogously, \( f_i \) decreases when \( P_i < \sum_j f_j P_j < 0.5 \) or \( P_i > \sum_j f_j P_j > 0.5 \). Therefore, the first part of the thesis is proven.

Now consider stationary points of the dynamics.

The right side of Eq. (8) reaches zero when \( f_i = 0 \), or \( \sum_j f_j P_j = P_i \)

and also when \( \sum_j f_j P_j = 0.5 \). In the latter case the derivative of \( f_i \) is zero for all \( i \), so this is a characterization of the stationary state of the entire female subpopulation. This constitutes the end of the proof.

Appendix G

Proof of Lemma 2. In the stationary state, payoffs of all strategies are equal, so mean payoffs also will have the same value. Let us find the stationary states of replicator equations. For an equation on \( P \), the stationary states are \( P = 1 \) (no females-trivial state), which is a repeller and \( \sum f_j P_j = P \) which is an attractor. Therefore, the equality of the primary and secondary sex ratios is proven. The stable state of male subpopulation dynamics should satisfy the following formula obtained from the MSE condition (12):

\[ m_i = \frac{P_i}{\sum_j f_j P_j} f_i \]

As we know from Lemma 1, in the case when the state of the male subpopulation is sufficiently close to the equilibrium value (MSE), the dynamics of the female subpopulation (8) will have two types of stationary states:

if \( \sum_j f_j P_j = 0.5 \), then \( m_i = 2P f_i \), states that satisfy this condition are elements of an evolutionarily stable set;

if \( \sum_j f_j P_j = P_i \), then \( m_i = f_i = 1 \), because condition \( \sum_j f_j P_j = P_i \) can be satisfied only for one strategy \( P_i \).

Which completes the proof.

References


