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Evolutionarily Stable Allele Distributions

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For the one-locus *m*-allele case we give a definition of an Evolutionarily Stable Allele Distribution (ESAD) for sexual populations, such that the associated game dynamics is a modified Fisher selection equation. For the ESAD we prove some basic statements which are parallel to those known in classical ESS theory. For an illustration, considering a two-allele dominant inheritance, we show that, if there is only a game-theoretical conflict within the population (and no Fisher type selection) then the ESS of the asexual population and the ESAD of the sexual one provide the same phenotype distribution. We also give an example of a two-allele non-dominant inheritance where the phenotype distributions corresponding to ESS and ESAD differ, the mean fitnesses of the two populations at their evolutionarily stable states, however, are equal.

1. Introduction

Fisher's selection model describes a biological situation in which the fitness of an individual (or that of a genotype) does not depend on the actual relative phenotype frequencies (Fisher, 1930). The approach of this model is allele oriented in the sense that the state of the population is described in terms of allele distributions. Although the selection takes place at zygote level, the distribution of phenotypes is uniquely determined by the allele distribution.

Maynard Smith & Price (1973) were the first to define ESS in evolutionary biology, considering an asexual population where each individual can have a unique behavioural phenotype, and the fitness of an individual (or that of a phenotype) depends on the actual relative phenotype frequencies. The approach of this model is phenotype oriented in the sense that the state of the population is described in terms of phenotype distributions.

In the present paper we shall deal with the following biological case: in a one-locus *m*-allele Mendelian population let each individual have a unique phenotype. A game-theoretical conflict is supposed which depends on the actual phenotype frequencies of the population and satisfies the well-known conditions of the evolutionary matrix game (the individuals have pairwise, symmetric random conflicts), with the modification that the population is panmictic and sexual. Our aim is to give an allele oriented evolutionary stability concept where, in analogy with Fisher's model, allele distributions are considered as states of the population. In this case evolutionary stability must consist in stability against perturbations resulting from allele mutations.

Maynard Smith (1973, 1982) defines ESS (evolutionarily stable strategy) as a state of the population in which no rare "mutant" can propagate via natural selection. The formal definition is the following:

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a phenotype frequency vector $y^* \in S_n$ is called an ESS, if the two following conditions are satisfied:

(a) equilibrium condition:

$$y^*\mathbf{A}y^* \geqslant y\mathbf{A}y^* \quad (y \in S_n)$$

(b) stability condition:

$$y \in S_m \backslash \{y^*\} \quad \text{and} \quad y\mathbf{A}y^* = y^*\mathbf{A}y^*$$

imply

$$y\mathbf{A}y < y^*\mathbf{A}y$$

where $\mathbf{A}$ is the pay-off matrix and S_n denotes the simplex of the space R^n.

The following basic statements are important reformulations of the above definition [see Hofbauer & Sigmund (1988); Van Damme (1991)]:

(i) $y^* \in S_n$ is an ESS if and only if for all $z \in S_n \backslash \{y^*\}$ in a neighbourhood of y^* we have

$$y^*\mathbf{A}z > z\mathbf{A}z.$$

(ii) An equilibrium $y^* \in \text{int } S_n$ is an ESS if and only if

$$\xi\mathbf{A}\xi < 0 \text{ for all } \xi \in R^n \backslash \{0\} \text{ with } \sum_i \xi_i = 0.$$

The original definition of ESS and the above reformulations are static in the sense that they do not say anything on how, with what kind of dynamics, a population can arrive at ESS.

Taylor & Jonker (1978) proposed a game dynamics and proved that if an ESS is regular in a certain sense then it is an asymptotically stable equilibrium point of this dynamics. Later Zeeman (1979, 1980) and Hofbauer *et al*. (1979) proved this basic theorem for a general ESS.

In the present paper we shall prove statements analogous to the above basic theorems, concerning a new definition of evolutionary stability proposed for diploid Mendelian populations. We also show that the continuous time selection dynamics obtained in a way similar to the derivation of Fisher's model, is a game dynamics corresponding to this evolutionary stability introduced for Mendelian populations. We shall follow the set up of the excellent book by Hofbauer & Sigmund (1988), proving theorems analogous to those of Chaps 15, 16 of this monograph. [See also Zeeman (1980) and Hofbauer *et al*. (1979)]. Vicker & Cannings (1987), later Bomze & Pötsher (1989) pointed out that for more general investigations it is appropriate to distinguish an uninvadable state from an ESS. Weissing (1991) also considered the concept of uninvadability primary [see also Hoekstra *et al*. (1991)]. In the present paper we shall also start out from the requirement of uninvadability.

Now we give a short survey on the investigations to which the present paper can be related.

Pohley & Thomas (1983) considered the case of a nonlinear individual fitness and in their definition of a *local* ESS the stability condition is required locally. (This definition of local ESS is not based on the notion of uninvadability.) The authors also showed that a local ESS is an asymptotically stable equilibrium point of the corresponding game dynamics.

Thomas (1985a, b) applied the definition of local ESS to Mendelian populations and distinguished a Genetical ESS (where both phenotypes and alleles have equal fitnesses) from the Genic ESS (where only alleles have the same fitnesses). He gave examples in which the two ESSs coincide and differ, respectively. In accordance with the set up of our paper we shall also give such examples.

Cressman (1988a, b) also studied game-theoretical conflicts in sexual populations. Similarly to his papers, we also start out from the selection dynamics. We take, however, a different approach, directly based on the principle of uninvadability.

It is no surprise that Fisher's selection equation turns out to be a game dynamics, since it is known that the equilibrium point of Fisher's equation is an evolutionarily stable state (Hofbauer & Sigmund, 1988). We also mention that Hofbauer *et al*. (1982) already proposed the selection equation as a game dynamics for such two-allele diploid Mendelian populations where each individual has one behavioural phenotype. The authors built the Hardy–Weinberg law into the replicator dynamics; they, however, did not give a definition of evolutionary stability for Mendelian populations.

In the following we also recall some papers dealing with sexual population which influenced our investigations.

Medelian populations have been studied in order to investigate, whether a sexual population can evolve to an ESS. In this approach the main question was *reachability*, i.e. whether a Mendelian population is able to realise an ESS characteristic to an asexual population. The answer in general is negative [Lloyd (1977); Maynard Smith (1981, 1989) and Cressman *et al*. (1996)].

Finally we also mention some authors who investigated evolutionary games in Mendelian populations when the individuals have mixed strategies (their strategies are distributions of pure strategies), which means that, in principle, an individual may have any of the possible behavioural phenotypes.

Lessard (1984) and Hofbauer & Sigmund (1988) posed two conditions to a solution of a game-theoretical conflict: *strategic* equilibrium condition (where the pay-offs of the strategies are equal) and the *genetic* equilibrium condition (which means that the Mendelian population is in a Hardy–Weinberg equilibrium). Cressman (1992) followed a similar idea for frequency dependent fitness, although he also considered Mendelian populations with density dependent fitness.

Hines & Bishop (1983, 1984), later Cressman and Hines (1984) pointed out that the equilibrium of a Mendelian population is not necessarily an ESS characteristic to an asexual population.

We notice that our treatment results in the same dynamics as the replicator dynamics considered by Cressman *et al*. (1996). There is, however, an important difference: we not only want to investigate the dynamics of the selection in a Mendelian population, but also give an appropriate definition of uninvadability and its equivalent operational reformulations. Our approach will be strictly based on allele distributions, an important contribution to this approach, in a different way was given by Weissing (1996).

In the present paper we start out from the following problem: as we have seen, the papers dealing with sexual populations usually require the fitnesses of the alleles to be equal at the equilibrium. Besides, some authors also require that the phenotypes have the same pay-off at equilibrium. In the classical selection model the fitnesses of the zygote individuals of different phenotypes, in general, are not equal (they do not depend on the state of the population). What are equal at a polymorphic equilibrium are the *marginal* (*potential*) fitnesses of the alleles. Therefore, we shall formulate the concept of *uninvadability* for an allele population as a definition of evolutionary stability for sexual populations, with the biologically natural requirement that the marginal fitnesses of the alleles should be equal at equilibrium. Another motivation to introduce an uninvadability concept for allele populations is the following. There are many well-known examples, when the genetic system can not reach the ESS, the natural selection, however, works in these cases, too. For this reason, on defining evolutionary stability for the Mendelian case we shall suppose that the hereditary system and the phenotypic selection together determine the evolutionary process. (This approach goes back to Fisher.) Therefore it is natural to ask that, if the phenotypes of individuals are uniquely determined by their alleles, how to define an appropriate concept of evolutionary stability for allele populations.

2. The Dynamics of the Allele Distribution

Consider a panmictic diploid Mendelian population where the genotype of an individual is determined by autosomal alleles $A_1, A_2, \ldots, A_m$. Suppose furthermore that each genotype determines a unique behavioural phenotype and there is a frequency dependent game-theoretical conflict within the population. Let us introduce the matrix $\mathbf{G} = (a_{ij,kl})$ where $a_{ij,kl}$ is the pay-off of an individual of genotype A_iA_j resulting from its conflict with an individual of genotype A_kA_l. Here we have applied the assumption that each individual can have only one behavioural phenotype. Since $\mathbf{G}$ contains the pay-offs of genotypes, it will be called the *genotypic pay-off matrix*. Suppose that there is no maternal inheritance. Then the *ij* and *ji* rows, as well as the *kl* and *lk* columns of $\mathbf{G}$ are the same, nevertheless, $\mathbf{G}$ is not symmetric. Notice that these partial symmetry properties of the genotypic pay-off matrix depend on the genotype–phenotype correspondence (similar to the symmetry of the Fisher selection matrix). Of course, it does not cause any difficulty if the genotypes have a fixed mixed strategy, instead of a pure one. The important assumption is that individuals are not able to change this strategy. Considering now the case of m alleles and n phenotypes, let $s_{ij} \in S_n$ be the mixed strategy of genotype A_iA_j and $\mathbf{A}$ the phenotypic pay-off matrix (see Introduction). Then the mean pay-off to A_iA_j from its conflict with A_kA_l is

$$a_{ij,kl} = s_{ij}\mathbf{A}s_{kl}$$

which defines the genotypic pay-off matrix. The above formalism is necessary for two reasons. On the one hand, the process is only determined by the allele distributions. In the above way all the model parameters are concentrated in the genotypic pay-off matrix and only the allele distributions are the state variables. On the other hand, we wanted our model to resemble Fisher's model both in notations and model building.

Denote by p_i the relative frequency of the allele A_i and suppose that the increment of the number of individuals of genotype A_iA_j during a time interval Δt is

$$\Delta t\left[x_{ij}\sum_{k,l} a_{ij,kl}p_kp_l\right]$$

where x_{ij} is the actual number of individuals of genotype A_iA_j.

Now, by a train of thought similar to the mentioned derivation of Fisher's selection model we obtain the Fisher type selection equation

$$\dot{p}_i = p_i[W_i(p) - \bar{W}(p)] \quad (i \in \overline{1,m}).$$

where $W_i(p)$, is the marginal fitness of allele A_i, and $\bar{W}(p)$ denotes the mean fitness, being the populationin state p. Formally

$$W_i(p) = \sum_j p_j \sum_{k,l} a_{ij,kl} p_k p_l, \quad \bar{W}(p) = \sum_{i,j} p_i p_i \sum_{k,l} a_{ij,kl} p_k p_l.$$

So the detailed version of the selection equation reads

$$\dot{p}_i = p_i \left[\sum_j p_j \sum_{k,l} a_{ij,kl} p_k p_l - \sum_{i,j} p_i p_i \sum_{k,l} a_{ij,kl} p_k p_l \right] \qquad (1)$$
$$(i = 1, 2, \ldots, m).$$

We remark that in the recalled derivation the population is supposed to be in a Hardy–Weinberg equilibrium all the time. This assumption is the same as that of the Fisher selection model. It is easy to see that dynamics (1) is identical with the replicator dynamics considered by Cressman *et al*. (1996).

3. Dyadic Product of Distributions

Consider an autosomal locus with m allele. Let us find the genotype distribution of the zygotes provided that in the random mating one allele is taken from a population with allele distribution y, and one from another population with allele distribution z. Then, under the usual Hardy–Weinberg type conditions, the required genotype distribution is a generalized Hardy–Weinberg distribution

$$(y_1 z_1, \ldots, y_1 z_m, y_z z_1, \ldots, y_2 z_m, \ldots, y_m z_1, \ldots, y_m z_m)^{\mathrm{T}}$$

which is nothing else than a column vector formed from the rows of the dyadic product of y and z. In the following the above distribution will be called the *dyadic product of distributions* y and z, and denoted by $\underline{yz}$.

In the following we shall use the dyadic product of distributions in a particular situation: let us suppose that in the population of Section 2, being in state p^*, an allele subpopulation of distribution p appears. Denote by x the new, *perturbed* distribution of the whole allele population. If with some $0 < \varepsilon < 1$ the ε part of the whole population is in state p then clearly

$$x = (1 - \varepsilon)p^* + \varepsilon p.$$

For a formal definition of ESAD we shall need the genotypic distribution of a particular zygote subpopulation: take an allele at random from the perturbant subpopulation of distribution p and then, in order to form a diploid zygote, choose another allele at random from the whole perturbed population of distribution x.

Problem

Find the distribution of the zygotes containing at least one allele from the perturbant subpopulation.

The solution to this problem for the *two-allele case* is the following. According to the definition of x as a mixture, choose the second allele with probability ε from the perturbant subpopulation, and with probability $(1 - \varepsilon)$ from the "wild" one. The genotype distribution of the zygotes obtained will be

$$(p_1^2, p_1(1 - p_1), (1 - p_1)p_1, (1 - p_1)^2),$$

if the second allele of the zygote is chosen from the distribution p, and

$$(p_1 p_1^*, p_1(1 - p_1^*), (1 - p_1)p_1^*, (1 - p_1)(1 - p_1^*)),$$

if the second allele is taken from the distribution p^*. (In the first case there are two alleles coming from the perturbant subpopulation, while in the second one only one.)

The mixture of the above distributions formed with respective weights ε and $(1 - \varepsilon)$ provides the distribution of gametes containing at least one allele from the perturbant subpopulation. Notice that the resulting distribution is

$$\underline{px} = \varepsilon \underline{pp} + (1 - \varepsilon)\underline{pp^*}.$$

In the above calculations we, of course, supposed that the panmictic population is *large* and the allele frequencies for both sexes are the same.

Obviously, the dyadic product of distributions can be generalized to any two vectors of the same dimension. It is easy to see that this operation is not commutative. It is, however, bilinear which implies the following equalities

$$\underline{xx} = \varepsilon \underline{px} + (1 - \varepsilon)\underline{p^* x} = \varepsilon^2 \underline{pp} + \varepsilon(1 - \varepsilon)\underline{pp^*} + (1 - \varepsilon)\varepsilon \underline{p^* p} + (1 - \varepsilon)^2 \underline{p^* p^*}.$$

In the following for the matrix $\mathbf{G} \in R^{m^2 \times m^2}$ we shall use the equalities

$$\underline{uv}\mathbf{G}\underline{wz} = \sum_{i,j,k,l} u_i v_j a_{ij,kl} w_k z_l \quad (u, v, w, z \in R^m),$$

$$\bar{W}(x) = \underline{xx}\mathbf{G}\underline{xx} \quad (x \in S_m)$$

and

$$W_i(x) = \underline{e^i x}\mathbf{G}\underline{xx} \quad (x \in S_m)$$

where e^i is the i-th basis vector.

Now system (1) can be written in the form

$$\dot{p}_i = p_i[\underline{e^i p}\mathbf{G}\underline{pp} - \underline{pp}\mathbf{G}\underline{pp}].$$

4. Evolutionarity Stable Allele Distribution (ESAD)

Now we give the definition of uninvadability for the following case: consider a sufficiently large panmictic population in which m autosomal alleles mate at random. The allele distributions for both sexes are supposed to be the same, and the phenotype of a zygote is uniquely determined by its genotype. We shall say that an allele distribution p^* is an ESAD if there is not any small perturbant allele subpopulation which can propagate via natural selection. This means that the mean fitness of an arbitrary perturbant allele subpopulation is less than that of the allele subpopulation at p^*

$$\sum_i p_i W_i(x) < \sum_i p_i^* W_i(x).$$

In this sense the state p^* is uninvadable. Now we give the formal

Definition.

We shall say that an allele distribution p^* is **uninvadable** for the diploid sexual population if for all $p \in S_m \backslash \{p^*\}$ there exists an $\varepsilon_p > 0$ such that

$$\underline{px}\mathbf{G}\underline{xx} < \underline{p^*x}\mathbf{G}\underline{xx}, \quad \text{for all} \quad 0 < \varepsilon < \varepsilon_p \qquad (2)$$

where x (the allele distribution of the whole population) is a mixture of distributions p and p^* taken with respective weights ε and $(1-\varepsilon)$. An uninvadable allele distribution p^* will also be called an **Evolutionarily Stable Allele Distribution**, briefly **ESAD**.

As it is seen, the logical structure of our definition is the same as that of the uninvadability, equivalent to an ESS.

Remark

Now we give a phenotypic interpretation of ESAD. To this end consider the matrix $\mathbf{S} = (s_{11} \ldots s_{1m} \ldots\ldots\ldots s_m \ldots s_{mm})$ with columns $s_{ij} \in S_n$ and the product

$$\mathbf{S}\underline{yz} = \sum_{i,j} y_i z_j s_{ij} \in S_n$$

where y and z are arbitrary allele distributions and, as before, s_{ij} is the genetically determined mixed behavioural phenotype of genotype $\mathrm{A}_i\mathrm{A}_j$. The vector $\mathbf{S}\underline{yz}$, as a mixture of distributions s_{ij} with weights $y_i z_j$, provides the mean phenotypic distribution of zygotes with one allele from distribution y and one from distribution z [see also Cressman *et al*. (1996)]. So matrix $\mathbf{S}$ describes the hereditary system (genotype–phenotype correspondence). With the pay-off matrix $\mathbf{A}$ of pure phenotypes, inequality (2) reads

$$(\mathbf{S}\underline{px})\mathbf{A}(\mathbf{S}\underline{xx}) < (\mathbf{S}\underline{p^*x})\mathbf{A}(\mathbf{S}\underline{xx}).$$

This condition, at zygote level, has the following interpretation: a subpopulation of zygotes having at least one allele from the perturbing allele distribution, at average produces less offspring than a subpopulation of zygotes having at least one allele from the ESAD distribution, provided the perturbation is small (see the meaning of $\underline{px}$ and $\underline{p^*x}$, Section 3). In our formulation two perturbed zygote subpopulations are compared, unlike the ESS, where the resident ESS is compared with the strategy of the "mutants". The above two statements also justify the use of dyadic product of distributions introduced in Section 3.

Remark

Notice that W_i, the marginal fitness of allele A_i, is a cubic function of the state of the whole allele population (containing both perturbant and "wild" subpopulations). Disregarding the dependence of x on p, the fitnesses of the subpopulations depend "linearly" on their strategy choices. This offers the formal interpretation that a subpopulation plays against the whole population and not against "itself".

Since the population is Mendelian (implying that the phenotypes of $\mathrm{A}_i\mathrm{A}_j$ and $\mathrm{A}_j\mathrm{A}_i$ are the same), the second and the third rows of the genotype pay-off matrix $\mathbf{G}$ coincide. Thus $\mathbf{G}\underline{xx}$ can be written in the form

$$\mathbf{G}\underline{xx} = \mathbf{G}[\varepsilon^2\underline{pp} + 2(1-\varepsilon)\varepsilon\underline{pp^*} + (1-\varepsilon)^2\underline{p^*p^*}].$$

Hence, for (2) we obtain an alternative form:

$$\begin{aligned}
&\underline{(p^*-p)p^*}\mathbf{G}\underline{p^*p^*} + \varepsilon\{2\underline{(p^*-p)p^*}\mathbf{G}\underline{p^*(p-p^*)} \\
&\quad + \underline{(p^*-p)(p-p^*)}\mathbf{G}\underline{p^*p^*}\} \\
&\quad + \varepsilon^2\{\underline{(p^*-p)p^*}\mathbf{G}\underline{(p-p^*)(p-p^*)} \\
&\quad + 2\underline{(p^*-p)(p-p^*)}\mathbf{G}\underline{p^*(p-p^*)}\} \\
&\quad + \varepsilon^3\{\underline{(p^*-p)(p-p^*)}\mathbf{G}\underline{(p-p^*)(p-p^*)}\} > 0.
\end{aligned} \qquad (3)$$

Based on the above observation we can prove the following

Theorem 1

An allele distribution $p^* \in S_m$ is an ESAD if and only if the following conditions are satisfied:

1. *equilibrium condition*:

$$\sum_i p_i^* W_i(p^*) \geqslant \sum_i p_i W_i(p^*),$$

or equivalently,

$$\underline{p^*p^*}\mathbf{G}\underline{p^*p^*} \geqslant \underline{pp^*}\mathbf{G}\underline{p^*p^*} \text{ for all } p \in S_m \quad (4)$$

2. *stability condition*:If for some $p \in S_m \backslash \{p^*\}$ equality holds in (4) then

$$\underline{2(p^* - p)p^*}\mathbf{G}\underline{p^*(p - p^*)} + \underline{(p^* - p)(p - p^*)}\mathbf{G}\underline{p^*p^*} \geqslant 0. \quad (5)$$

If for some $p \in S_m \backslash \{p^*\}$ equality holds in both (4) and (5) then

$$\underline{(p^* - p)p^*}\mathbf{G}\underline{(p - p)(p - p^*)} + 2\underline{((p^* - p)(p - p^*)}\mathbf{G}\underline{p^*(p - p^*)} \geqslant 0. \quad (6)$$

If for some $p \in S_m \backslash \{p^*\}$ equality holds in both (4),(5) and (6) then

$$\underline{(p^* - p)(p - p^*)}\mathbf{G}\underline{(p - p^*)(p - p^*)} > 0.$$

Proof

Indeed, if p^* is an ESAD then passing to the limit in (3) for $\varepsilon \to 0$ we obtain (4). Putting equality in (4), by substitution into (3) we get a new inequality. Passing to the limit again we get (5) and so forth.

Conversely, suppose that conditions 1 and 2 hold. In particular, suppose that in (4) strict inequality holds. Then, since (4) is obtained from (3) by passing to the limit, there is an $\varepsilon_p > 0$ such that (3) holds for all $0 < \varepsilon < \varepsilon_p$. The other cases can be treated similarly.

We remark that the above system of conditions is analogous to the requirements (a) and (b) of the Introduction. Furthermore, in (3) we used the condition that the individuals of genotypes $\mathbf{A}_i\mathbf{A}_j$ and $\mathbf{A}_j\mathbf{A}_i$ have the same phenotypes. Nevertheless, this particular condition affects only the form of the stability condition and not that of the equilibrium condition.

The equilibrium condition formulates an optimality requirement for diploid Mendelian populations, which in our case can be interpreted in the following way: consider two subpopulations with respective allele distributions p^* and p. If the mating of the alleles is carried out within a subpopulation of state p^* then the mean fitness of the zygote subpopulation obtained is greater than or equal to the mean fitness of the zygote population obtained from the ''mixed'' mating of allele populations of respective states p and p^*.

Furthermore, in the usual formulation, the equilibrium can be interpreted by saying that p^* *is a best reply against itself*. In this sense the equilibrium condition 1 is closely related to the concept of the Nash equilibrium.

The stability condition 2 is rather sophisticated because the dependence of the marginal fitnesses of alleles on the allele frequencies of the whole population is not linear.

Now we prove a theorem analogous to statement (i) of the Introduction:

Theorem 2

$p^* \in S_m$ is an ESAD if and only if for all states $x \neq p^*$ in a neighbourhood of p^* we have

$$\sum_i p_i^* W_i(x) > \sum_i x_i W_i(x),$$

or equivalently,

$$\underline{p^*x}\mathbf{G}\underline{xx} > \underline{xx}\mathbf{G}\underline{xx}. \quad (7)$$

Proof

Suppose that p^* is an ESAD. Notice that all x from a neighbourhood of p^* can be written in the form $x = (1 - \varepsilon)p^* + \varepsilon p$ where p runs over the set

$$C := \{p \in S_m : p_i = 0 \quad \text{for some} \quad i \in \operatorname{supp}(p^*)\}.$$

Since p^* is an ESAD, any p from C also satisfies inequality (3). For all $p \in S_m$, and $\varepsilon \in]0,1]$ let $f(p, \varepsilon)$ be the l.h.s. of (3). Then with appropriate functions φ_i we have

$$f(p, \varepsilon) = \varphi_1(p) + \varphi_2(p)\varepsilon + \varphi_3(p)\varepsilon^2 + \varphi_4(p)\varepsilon^3 \geqslant 0.$$

Now for a given $p \in C$ either (a) $f(p, \varepsilon) > 0$ for all $\varepsilon \in]0,1]$, or (b) the cubic equation $f(p, \varepsilon) = 0$ has a solution $\varepsilon \in]0,1]$. In case (b) denote by ε_0 the minimal solution in $]0,1]$. Let $\bar{\varepsilon}(p)$ equal 1 in case (a) and $\min\{1, \varepsilon_0\}$ in case (b). Since functions φ_i are continuous, and the zeros of a polynomial depend continuously on their coefficients, function $\bar{\varepsilon}$ is also continuous on the compact set C, and clearly positive. Therefore, by the Weierstrass theorem, $\bar{\varepsilon}$ attains a positive minimum ε_*. Hence, for all $p \in S_m \backslash \{p^*\}$from the ε_*-neighbourhood of p^* we have

$$\underline{px}\mathbf{G}\underline{xx} < \underline{p^*x}\mathbf{G}\underline{xx}, \quad \text{for all} \quad 0 < \varepsilon < \varepsilon_*.$$

Now multiplying (2) by ε and adding $(1 - \varepsilon)\underline{p^*x}\mathbf{G}\underline{xx}$ to both sides we get (7). The proof of the opposite implication is straightforward.

In terms of the marginal fitnesses the equilibrium condition reads

$$\sum_i p_i^* W_i(p^*) \geqslant \sum_i p_i W_i(p^*).$$

Hence, by a reasoning known in the asexual theory of ESS [see Hofbauer & Sigmund (1988)], we obtain that at an interior ESAD p^* the marginal fitnesses of all alleles must be equal. Similarly, at an ESAD

belonging to any subsimplex, the corresponding marginal fitnesses are equal. Hence we have the following

Proposition

If $p^* \in S_m$ satisfies the equilibrium condition then it is an equilibrium point of dynamics (1).

Now we obtain a theorem analogous to statement (ii) of the Introduction. By Theorem 2, $p^* \in S_m$ is an ESAD if and only if

$$\sum_i p_i^* W_i(x) > \sum_i x_i W_i(x)$$

for all $x \neq p^*$ in the ε_*-neighbourhood of p^*, where the functions W_i are cubic polynomials. Introduce the notation $\xi = x - p^*$. Then, using the coefficients of the corresponding Taylor polynomial, $W_i(x)$ can be written as a cubic polynomial of ξ. Denote by $\operatorname{grad} W(p^*)$ the matrix the i-th row of which is $\operatorname{grad} W_i(p^*)$ $(i = 1, \ldots, m)$. Making use of the fact that $W_i(p^*) = W_j(p^*)$ we get the following

Theorem 3

A state $p^* \in \operatorname{int} S_m$ is an ESAD if and only if for all

$$\xi \in R^m \backslash \{0\} \text{ with } \sum_i \xi_i = 0, \text{ we have } \xi(\operatorname{grad} W(p^*))\xi \leqslant 0;$$

if in the above inequality equality holds for some ξ, then

$$\sum_i \xi_i \left(\frac{1}{2} \sum_i \frac{\partial^2 W_i(p^*)}{\partial x_i^2} \xi_i^2 + \sum_{i \neq j} \frac{\partial^2 W_i(p^*)}{\partial x_i \partial x_j} \xi_i \xi_j \right) \leqslant 0;$$

if in both the above inequalities equality holds for some ξ, then

$$\sum_i \xi_i \left(\frac{1}{6} \sum_i \frac{\partial^3 W_i(p^*)}{\partial x_i^3} \xi_i^3 + \frac{1}{2} \sum_{i \neq j} \frac{\partial^3 W_i(p^*)}{\partial x_i^2 \partial x_j} \xi_i^2 \xi_j \right) < 0.$$

Now we show that (1) is a game dynamics for the above ESAD.

Theorem 4

If $p^* \in \operatorname{int} S_m$ is an ESAD then it is an asymptotically stable equilibrium point of the selection equation (1).

Proof Suppose that $p^* \in \operatorname{int} S_m$ is an ESAD and define

$$V(p) = \sum_i p_i^* \left(\log \frac{p_i}{p_i^*} - \frac{p_i}{p_i^*} + 1 \right), \qquad p_i > 0,\ i = 1, \ldots, m.$$

We show that v is a local Lyapunov function for the equilibrium p^*. Indeed, by the concavity of the function log, for all $z > 0$, $z \neq 1$ we have

$$\log z < z - 1.$$

Hence it follows that $V(p) < 0$ for $p \in S_m \backslash \{p^*\}$ and $V(p^*) = 0$. Its derivative with respect to system (1) is

$$\sum_i \left(\frac{p_i^*}{p_i} - 1 \right) p_i [W_i(p) - \bar{W}(p)] = \underline{p^* p} \mathbf{G} \underline{pp} - \underline{pp} \mathbf{G} \underline{pp}.$$

From Theorem 2 we know that the latter is positive in some neighbourhood of p^* in S_m, except p^* where it is zero.

Remark

Unlike the classical case [see e.g. Hofbauer & Sigmund (1988) 16.4], for an interior equilibrium p^*, in general, the asymptotic stability is only local, because by the nonlinearity there may exist another (locally) asymptotically stable equilibrium as well.

Remark

It is not hard to see that in the two-allele case a (locally) asymptotically stable equilibrium point of dynamics (1) is an ESAD, too. This is analogous with a result of the asexual ESS theory recalled in the Introduction.

Remark

Mutation implies a perturbation of the allele distribution, the magnitude of which depends on the mutation rate. It may happen that an allele population state cannot be invaded by a mutant allele subpopulation if the mutation rate is small, for a greater mutation rate, however, it can.

Now we show that, following the train of thought of Sections 3 and 4, by solving the analogon of **Problem** of Section 3 for a certain asexual population, the classical ESS (uninvadability) is obtained. In Section 3 we have seen that in a diploid Mendelian population the genotype distribution of all zygotes having at least one allele from the perturbant subpopulation, in terms of the notation used there, is $\underline{px}$.

Suppose that the asexual population is *haploid*. Then in the two-allele case the genotypic pay-off matrix is

$$\mathbf{G} = \begin{pmatrix} a_{1,1} & a_{1,2} \\ a_{2,1} & a_{2,2} \end{pmatrix}.$$

Denote by p the allele distribution of the perturbant allele subpopulation, and let p^* be that of the "wild" allele subpopulation. Assume that an "ε part" of the population is perturbant. The solution of the corresponding **Problem**, i.e. the haploid genome type distribution of all individuals having an allele from the perturbant subpopulation is p. Therefore, in the Definition of Section 4, in the haploid case, $\underline{px}$ is to be changed for p. Similarly, $\underline{p^* x}$ and $\underline{xx}$ is replaced by p^* and x, respectively. If we include these assumptions into our Definition, we obtain the

basic theorems and game dynamics of the classical theory of ESS of asexual populations.

If in a two-allele diploid asexual population, to different genotypes there correspond different behavioural phenotypes then proceeding similarly to the haploid case, we get back the basics of asexual ESS theory. Notice that our model reduces to Fisher's selection model, if all components of each row of the genotypic pay-off matrix **G** are the same.

5. Examples

In this section we shall show in concrete examples, what asymptotic phenotype distributions are obtained as a result of evolutionary game conflicts in sexual and asexual populations. In our investigations the game dynamics for sexual and asexual populations will be compared.

Example 1

Let us consider a two-allele Mendelian population in which allele A_1 is dominant to allele A_2. Suppose that within the population there is a game conflict. Denote by δ and ρ the dominant and the recessive phenotypes, respectively. Let the corresponding phenotypic pay-offs be as follows:

	δ	ρ
δ	a	b
ρ	c	d

Then for the genotype pay-off matrix **G** we have

$$G = \begin{pmatrix} a & a & a & b \\ a & a & a & b \\ a & a & a & b \\ c & c & c & d \end{pmatrix}$$

Now for a formal simplification it is more convenient to consider the selection equation for the *recessive* allele.

$$\dot{p}_2 = p_2^2(1 - p_2)[c - a + (d - c - b + a)p_2^2].$$

The non-trivial equilibrium point of this dynamics is

$$p_2^* = \left(\frac{c - a}{c - a + b - d}\right)^{1/2},$$

provided that the denominator is not zero and has the same sign as the numerator. Also suppose that $p_2^* \in]0,1[$. Then it is not hard to see that p_2^* is asymptotically stable if and only if $d - c - b + a < 0$. By Theorem 2 it is easy to see that p^* is an ESAD if and only if for all $p_1 \in [0,1]$, $p_1 \neq p_1^*$ we have

$$p_1[(b - a + c - d)x_2^2 + a - c] < p_1^*[(b - a + c - d)x_2^2 + a - c] \quad (8)$$

for all x_2, sufficiently close to p_2^*.

Consider now the pure game conflict for the asexual population. Then the game dynamics for the relative frequency of the phenotype ρ is the following

$$\dot{y}_2 = y_2(1 - y_2)[c - a + (d - c - b + a)y_2].$$

Suppose that

$$y_2^* := \frac{c - a}{c - a + b - d} \in]0, 1[.$$

Then, again y_2^* is asymptotically stable if and only if $d - c - b + a < 0$. By statement (i) of the Introduction, it is clear that y^* is an ESS if and only if for all $y_1 \in [0,1]$, $y_1 \neq y_1^*$ we have

$$y_1[(b - a + c - d)y_2 + a - c < y_1^*[(b - a + c - d)y_2 + a - c] \quad (9)$$

for all y_2 sufficiently close to y_2^*.

Now we compare the sexual and the asexual cases. It is easily seen that for the coordinates of the equilibrium p^* we have

$$p_1^{*2} + 2p_1^*p_2^* = \frac{d - b}{a - c - b + d},$$

$$p_2^{*2} = \frac{a - c}{a - c - b + d}.$$

Notice that the equilibrium relative frequency of the diploid individuals of the sexual population, containing at least one dominant allele A_1 equals the equilibrium relative frequency of the dominant phenotype in the asexual population. Hence the phenotype distribution of the populations coincide at equilibrium. Notice moreover that (8) and (9) are essentially equivalent by the correspondence $y_2 = x_2^2$. The latter equality means that the respective phenotype distributions of the sexual and asexual populations are the same at their evolutionarily stable states.

So we have proved that, for a given game-theoretical conflict an asexual population has a mixed ESS if and only if the sexual population with dominant inheritance has a mixed ESAD. In this case the phenotype frequencies are the same in both populations.

Example 2

Now we consider a numerical example for the two-allele case when in the population there are three conflicting behavioural phenotypes involved which belong to different genotypes.

First we investigate the diploid *asexual* population with a pay-off matrix

$$\mathbf{A} = \begin{pmatrix} 0 & 16 & -35 \\ -15 & 0 & 12 \\ 55 & -11 & 0 \end{pmatrix}$$

where the subsequent rows of **A** contain the pay-offs of the phenotypes corresponding to the genotypes A_1A_1, A_1A_2 and A_2A_2, respectively. Then the gamedynamics of the asexual population is the following:

$$\dot{y}_1 = y_1(16y_2 - 35y_3 - y_1y_2 - 20y_1y_3 - y_2y_3)$$
$$\dot{y}_2 = y_2(12y_3 - 15y_1 - y_1y_2 - 20y_1y_3 - y_2y_3)$$
$$\dot{y}_3 = y_3(55y_1 - 11y_2 - y_1y_2 - 20y_1y_3 - y_2y_3).$$

Its equilibrium point is

$$y^* = \left(\frac{709}{5104}, \frac{3110}{5104}, \frac{1285}{5104}\right).$$

The eigenvalues of the matrix of the system linearised around y^*, for two decimal digits are $\lambda_{1,2} = -2{,}53 \pm i21{,}35$. Therefore y^* is asymptotically stable. [We remark that y^* is also an ESS again, see Hofbauer & Sigmund (1988) 16.5, Exercise 5.]

The mean fitness of the asexual population at equilibrium is $y^*\mathbf{A}y^* = 15/16$.

Consider now the diploid *sexual* population. Then, according to the above, the genotype pay-off matrix is

$$\mathbf{G} = \begin{pmatrix} 0 & 16 & 16 & -35 \\ -15 & -0 & 0 & 12 \\ -15 & 0 & 0 & 12 \\ 55 & -11 & -11 & 0 \end{pmatrix}.$$

The selection equation for the relative frequency ofthe allele A_1 is of the form

$$\dot{p}^*_1 = p_1(1 - p_1)[W_1 - W_2],$$

which in our case provides

$$\dot{p}_1 = p_1(1 - p_1)\left[p_1^3 + 3p_1^2 - \frac{61}{16}p_1 + \frac{3}{4}\right].$$

The non-trivial equilibrium points are the following 1/4, 3/4, -4. It is easy to check that 1/4 and 1 are asymptotically stable, while 3/4 is unstable. So there is only one non-trivial asymptotically stable equilibrium point and here the phenotype distribution is (1/16, 6/16, 9/16). The mean fitness of the population at this equilibrium is 15/16, the same as for the asexual case.

Let us compare now the behaviours of the asexual and the sexual populations in our example. The asexual population has a greater degree of freedom, since at a phenotypic equilibrium the genotypes are not necessarily in a Hardy–Weinberg equilibrium. Nevertheless, the sexual population has an asymptotically stable equilibrium point at which the mean fitness for the sexual population is the same as that obtained for the asexual population at its asymptotically stable equilibrium point. The conclusion is that the phenotype distributions are different for the two populations. Hence it follows that although the pay-offs of the alleles are equal at the asymptotically stable equilibrium point of the sexual population, the pay-offs of the behavioural phenotypes (pure strategies or individuals) are not, unlike the asexual population where the pay-offs of the phenotypes (individuals) are equal.

6. Conclusions

Our definition of ESAD is based on the concept of uninvadability which, in our case, means the following: *an allele distribution is called uninvadable, if there is no small perturbant allele subpopulation which could invade the population via natural selection*. The concept of an ESAD proposed by us turned out to be operational, since concerning this notion we managed to prove statements analogous to the theorems of classical ESS theory recalled in the Introduction.

In Example 1 we have shown that if one of the alleles is dominant and the same game-theoretical conflict takes place in the sexual and asexual populations then ESS and ESAD are equivalent.

In Example 2 we have seen that for the same game-theoretical conflict, asexuality and sexuality of the population may generate different phenotype distributions at equilibrium, the mean fitnesses of the two populations, however, are equal. The main reason for the appearance of different equilibria in sexual and asexual populations is that in sexual populations an equilibrium must be a Hardy–Weinberg genetic equilibrium as well.

It seems that all this stands or falls on what is considered a player. In ESS theory for asexual populations there are two different ideas concerning this problem. One is that *individuals* are players [Vincent & Brown (1988)]. According to another approach *subpopulations* are the "players". In the biological model considered by us, only the *allele subpopulations* are considered as "players", and not the *zygote individuals*. Indeed, on the one hand, according to the Mendelian laws, the allele distribution determines the phenotype distribution in a

unique way. On the other hand, in meiosis two allele subpopulations are formed: the subpopulation of individuals containing at least one allele from the perturbant allele subpopulation and that having only ones from the "wild" allele subpopulation. In terms of mathematical game theory these subpopulations as "players", playing against the whole population, can "choose" a mixed strategy (an allele distribution) and the subpopulation having greater fitness will propagate. Notice that in both allele subpopulations the mean fitnesses of the same allele types are equal. For the whole population, of course, the fitnesses of different alleles are not necessarily equal, and the allele with greater fitness will propagate in the population. In spite of this fact neither an allele nor the whole population can be considered as a "player" in terms of mathematical game theory. Indeed, the following case may occur: a state (i.e. an allele distribution) of the whole population cannot be invaded by a perturbant subpopulation containing only one allele type, however, it may be invaded by a mixed perturbant subpopulation. [See Hofbauer & Sigmund (1988) 16.5, Exercise 9 on the asexual case.]

In evolutionary game theory the crucial concept seems to be the criterion of uninvadability, rather than the Nash equilibrium. Uninvadability is equivalent to the Darwinian principle saying that a subpopulation with greater fitness will propagate. Therefore, it should be taken into consideration that also in other application fields of mathematical game theory (such as economics), a new solution concept based on the concept of uninvadability may be useful, which means a stronger requirement than the Nash solution.

Our approach seems to have a natural generalisation to the multi-locus and multi-allele case. The principle of uninvadability will remain valid, for a description of the more complicated hereditary system, however, a generalisation of the dyadic product of distributions will be necessary. The problem is a lot more difficult when the hereditary system does not uniquely determine the behaviour of an individual, making it possible for the individual to choose a strategy from a genetically limited subset of the set of all possible phenotypes.

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