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Random and non-random mating populations: Evolutionary dynamics in meiotic drive

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ABSTRACT

Game theoretic tools are utilized to analyze a one-locus continuous selection model of sex-specific meiotic drive by considering nonequivalence of the viabilities of reciprocal heterozygotes that might be noticed at an imprinted locus. The model draws attention to the role of viability selections of different types to examine the stable nature of polymorphic equilibrium. A bridge between population genetics and evolutionary game theory has been built up by applying the concept of the Fundamental Theorem of Natural Selection. In addition to pointing out the influences of male and female segregation ratios on selection, configuration structure reveals some noted results, e.g., Hardy–Weinberg frequencies hold in replicator dynamics, occurrence of faster evolution at the maximized variance fitness, existence of mixed Evolutionarily Stable Strategy (*ESS*) in asymmetric games, the tending evolution to follow not only a 1:1 sex ratio but also a 1:1 different alleles ratio at particular gene locus. Through construction of replicator dynamics in the group selection framework, our selection model introduces a redefining bases of game theory to incorporate non-random mating where a mating parameter associated with population structure is dependent on the social structure. Also, the model exposes the fact that the number of polymorphic equilibria will depend on the algebraic expression of population structure.

1. Introduction

Mendel demonstrates that a hybrid between two different varieties possesses both types of parental factors in the gametes (the principle of segregation) and the two individual genes in a particular gene pair (2 alleles) are equally represented in its gametes. Segregation distorters, however, violate this rule by biasing segregation in their favor. Generally, Segregation distortion refers to *any distortion of meiosis or gametogenesis such that one of a pair chromosomes in a heterozygote is recovered in greater than half of the progeny* [1]. The associated gene is said to "drive" and the phenomenon of meiotic drive is the subset of segregation distortion. In an extended sense, a segregation distorter/meiotic drive gene is nothing but the paradigmatic category of selfish genetic elements because such elements are either neutral or detrimental to the organism's fitness [2]. The genes found in an inversion on chromosome 2 of Drosophila melanogaster and *t*-complex in mice are tailored to explain the segregation distortion mechanism.

As segregation distorters occur in many species and are likely to be very common [1,3], a fascinating mathematical theory has been developed through mathematical models to answer the questions concerning the stability and evolution of Mendelian segregation (see Feldman and Otto [4], Haig and Grafen [5], Weissing and van Boven [6], Úbeda and Haig [7] and references therein). Úbeda and Haig [7] were the first population geneticists to formulate a one-locus sex-specific segregation distortion model by including a genomic imprinting concept. They provided a numerical analysis of the equilibria, finding examples of parameter sets with three polymorphic equilibria in which at most two of them were stable. It was also noted that, in general, population mean fitness is not maximized at polymorphic equilibria.

It is seen that since the main stream of studies of evolutionary game dynamics basically concentrates on the evolution of strategies in animal conflicts, the evolutionary dynamics of population genetic mechanisms is often neglected in the context of evolutionary game theory. In the majority of cases, neglecting the underlying genetic architecture in detail, the models of evolutionary structure are developed in the mentioned research field where it has been tried to set up a relation between population genetics and evolutionary game theory through the perception of evolutionary stable strategies and quantitative genetics [8–10]. Most of the evolutionary game theorists believe that, besides the evolution of phenotypes, game dynamics is only directly applicable to study of a single locus haploid genetic model; consequently, genetics related focus has mainly been confined in haploid structure [11–16]. However, it is frequently argued

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that it can equally be applied to study of the more complex genetic evolution than asexual. In the recent years, the concepts of stochastic evolutionary dynamics [17–19] and coalescence theory [20–22] have built a stable foundation between evolutionary game theory and population genetics.

A bridge between population genetics and game theory is constructed by a simple relabeling of terms. Any single-locus, diploid model in population genetics can be interpreted as a game in which individuals are the players and distinct alleles are the strategies [23–25] where the frequencies of genes act as meanfitness optimizer [26]. However, based on different framework structure, game theory for diploid population has also been studied in many literatures [27–30]. Recently, in the context of meiotic derive Traulsen and Reed [25] have formulated a model of diploid population by considering an interaction between alleles in a diploid genome as a two player game. The dynamics is studied through the well-known replicator equation and meiotic derive has been explained as a social dilemma such as the prisoner's dilemma or the snowdrift game. Cyclic dominance found in the rock-paper-scissors game is also embedded in their model.

In general, the evolutionary game theorists use the replicator dynamics to considering game with 2 player or many players in well-mixed large population because its equilibrium concept, an evolutionarily stable strategy (*ESS*) – refinement of Nash equilibrium, *ESS* is an asymptotically stable state of the evolutionary dynamics – positively describes evolutionary outcomes in environments. However, there are many population games with non-random matching, concerning to games of group selection [31–33] where formation shows some assortment. At this point, we cannot deny that the group selection is not the correct expression for what is just non-random matching because the non-random can be coped with in the usual evolutionary framework, in the way in which the concept of non-random matching merely needs to be built into the definition of the game (see Taylor and Nowak [34]). In a particular sense, matching is typically assortative meaning that individuals have a higher probability of being matched with other like-natured individuals than with different-natured individuals.

Since the population structure plays a crucial role in evolutionary theory, the canonical group selection models [35–37] can also influence the genetic literature. The canonical group selection model is called either the haystack model or the multi-level selection model. In order to construct the relation between group selection replicator dynamics and population structure, van Veelen [38] partitions the whole population into groups of size n, within which n-player game is played where group state representing the group frequencies of the different types of the groups in the population, forms the population structure. In the model, fitness is assigned to individuals (individualist perspective; see Kerr and Godfrey-Smith [39] and references therein) rather than to the groups in such a way that replicator dynamics depends only on average payoffs of the individuals similar to the game selection model. However, such models can formally recast so that groups are fitness bearing. It was Kerr and Godfrey-Smith [39] who provided a detailed analysis of the multi-level selection model in the two strategy case. In the recent work of Jensen and Rigos [40], the thought of matching rule is elaborated to any number of strategies by means of a rigorous formalism. Using the analytical thinking of structural conception of replicator dynamics, the model framework draws a concrete line between game and group selection theories.

In this article, I extend a traditional model for selection at an autosomal imprinted locus in a sex-differentiated population, by considering the sex-specific viability with assigning separate viabilities to reciprocal heterozygotes. Allowing overlapping generations, Úbeda and Haig [7]'s discrete genetic framework has been used to develop continuous governing equations of evolutionary dynamics, namely replicator dynamics where, implementing the transmission ratio distortion rules at an imprinted locus, the concept of segregation distortion is incorporated. The group selection version of replicator dynamics is also introduced to examine the non-random influence. Here, my main intention is to formulate a set of link-results between evolutionary game theory and population genetics following the path constructed by Traulsen and Reed [25].

2. The model of evolutionary dynamics

We pay attention to a specific type of evolutionary dynamics, called replicator dynamics to address the question, whether a polymorphic population with the two-population profiles can also be stable by generating the *ESS*. In the evolutionary game, it is assumed that individuals are programmed to use only pure strategies and passes this behavior to its descendants without modification; because of that the individuals in such selection dynamics are called replicators, existed in several different types [13,15,41,42]. As the success of evolutionary game dynamics is defined by the fitness of individuals, considering a pairwise contest population game with different action sets and different payoff matrices, in the context of well-mixed populations, the evolutionary game dynamics can be written as

$$\begin{aligned}\dot{x}_i &= x_i(\pi_1(e^i, Y) - \pi_1(X, Y)),\\ \dot{y}_i &= y_i(\pi_2(e^i, X) - \pi_2(Y, X))\end{aligned} \tag{2.1}$$

where $\pi_1(X, Y))$ and $\pi_2(Y, X))$ signify the expected fitness payoffs of the two populations when the frequencies of types relating to strategy e^i (unit vector) are x_i and y_i, and corresponding average fitnesses of types are given by $\pi_1(e^i, Y)$ and $\pi_2(e^i, X)$.

Letting the pairwise contest population game being performed among alleles at a single locus, we move to the field of population genetics to establish that game theoretic perspective and population genetic perspective lead to exactly the same dynamics in respect of Eqs. (2.1) which govern allele frequencies. Consider an infinite, panmictic diploid population. The viability of genotypes A_1A_1, A_1A_2, A_2A_1 and A_2A_2 in males are designed as W_{11}, W_{12}, W_{21} and W_{22}, with corresponding values $V_{11}, V_{12}, V_{21}, V_{22}$ in females where in males the autosomal allele written first has a paternal origin while the one written second has a maternal origin whereas paternally inherited alleles are listed second in the females. By explicitly considering perfect transmission of one allele in one sex and the other allele in the opposite sex, let the segregation ration of A_1 be k in male meiosis and κ in female meiosis, while the corresponding rations for A_2 are $(1-k)$ and $(1-\kappa)$ $(0 < k, \kappa < 1)$. That is values of k, κ less than one-half can be interpreted as segregation distortion in favor of A_2 or negative segregation distortion of A_1 in respect of particular sex. The term "drive" is used by Burt and Trivers [2] to denote the greater than Mendelian ("super-Mendelian") transmission of a selfish genetic element, whereas "drag" is the opposite implying less than Mendelian inheritance ("sub-Mendelian"). And hence if we let the frequency of gametes A_1, A_2 be x_t, $1-x_t$ in sperm and y_t, $1-y_t$ in eggs, then in the mating pool for the following generation the frequencies of male allele A_1s and female allele A_1s are [7,43]

$$\begin{aligned}x_{t+\Delta t} &= \frac{W_{11}x_ty_t + k(W_{12}x_t(1-y_t) + W_{21}y_t(1-x_t))}{W_{11}x_ty_t + W_{12}x_t(1-y_t) + W_{21}y_t(1-x_t) + W_{22}(1-x_t)(1-y_t)},\\ y_{t+\Delta t} &= \frac{V_{11}x_ty_t + \kappa(V_{12}x_t(1-y_t) + V_{21}y_t(1-x_t))}{V_{11}x_ty_t + V_{12}x_t(1-y_t) + V_{21}y_t(1-x_t) + V_{22}(1-x_t)(1-y_t)}.\end{aligned} \tag{2.2}$$

Differential expression of genes depending on their parental origin is referred to genomic imprinting that can cause reciprocal heterozygotes to have distinguishable phenotypes and different viabilities. Here, considering W_{12}, V_{21} being constants over time, we introduce the following genomic imprinting relations:

$$W_{21} = W_{12}\frac{x_t(1-y_t)}{y_t(1-x_t)} = W_{12}r_{x_ty_t} \quad \text{and}$$

$$V_{12} = V_{21}\frac{y_t(1-x_t)}{x_t(1-y_t)} = V_{21}r_{y_tx_t}.$$