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# Evolutionary Game-Theoretical Models of Deterministic Fertility-Viability Selection

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**Abstract**

Mathematical population genetics and evolutionary game-theoretical modelling together is a facet of post-genomic mathematical biology. Mathematical corrections necessary to the probabilistic application of population genetics in two dynamical systems from a recent illustrative example in the literature are obtained. In particular, interaction between fertility and viability selection is modelled in a recent review on evolutionary multi-player games (Gokhale and Traulsen, 2014, *Dyn. Games Appl.,* v. 4, pp. 68-88; doi:10.1007/s13235-014-0106-2) within a section on population genetics. In the first case, a counter-intuitive solution in evolutionary dynamics reveals a persistent misconception in evolutionary game theory. The second model correction shows the extent of the error incurred under biologically unrealistic use of population genetics. Corrections describe the payoff matrices and marginal expected allelic fitness. Single locus diploid symmetric viability and asymmetric fertility fitness yield the expected offspring allele fitness, when properly translated into a game-theoretical model. The deterministic trajectory of the di-allelic epi-static interaction between female fertility fitness and a maternal-effect dominant viability hazard is then proven.

**Keywords:** Allele dynamics; discrete-time replicator equations; epi-static interaction; maternal-effect dominant autosomal gene.

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## INTRODUCTION

Epi-static interaction and discrete replicator equations are considered to derive expectations of game-theoretical multi-player payoffs from mating patterns in diploid population genetics (*cf.* Gokhale and Traulsen 2014) [17]. Their review [17] contains interesting discussion of evolutionary dynamics, which they demonstrate impacts on a variety of sciences including theoretical socio-biology (Gokhale and Traulsen 2010). Similar game-theoretical heuristics have also been used to envision hypothetical applications to the agricultural science of genetically designed pest management (Gokhale *et al.* 2014). A new paradigm in evolutionary game theory includes population genetics in the modelling (Antal *et al.* 2009; Nowak 2012; Traulsen and Reed 2012). This resurgence has foundations in the evolution of altruistic behaviour (Karlin and Matessi 1983, Eshel and Cavalli-Sforza 1982; Eshel 1982; Uyenoyama and Feldman 1980, 1982) and population genetics of deterministic fertility and viability selection (Liberman and Feldman 1985; Feldman and Liberman 1985; Feldman *et al.* 1983). Another new paradigm includes mathematically unifying adaptive dynamics for evolutionary trajectories of non-discrete strategies and environments (Allen *et al.* 2013; Allen and Tarnita 2014). In the present article, an apparently counter-intuitive randomization structure clarifies such use of population genetics and this represents a new realism previously confounded in similar models. The corresponding mathematics of an earlier game-theoretical model is corrected to prove the deterministic trajectory of the evolutionary dynamics under the heuristic approach differs substantially from the biologically realistic perspective.

In particular, [17] consider a four-player evolutionary game, with two strategies that accords to a model of diploid fitness between two alleles without mutation at a single genetic locus. The mathematical expediency with which they obtain symmetries in allelic payoff matrices and calculate corresponding marginal expectations is shown in this article to be inconsistent. Ameliorated derivations are shown here in four main themes: (i) the corrected game-theoretical model for symmetric viability selection and the corresponding calculation of its strategic payoffs also corrects the use of a classic result in mathematical population genetics (Wright 1949); (ii) payoff equations of the evolutionary dynamics for asymmetric fertility selection; (iii) exposition of the translation from evolutionary game theory to population genetics; and (iv) verification of results by Wade and Beeman (1994) that motivates a precise game-theoretical interpretation of the mating patterns in this genetic system with epi-static selection.

A marginal payoff function is developed in [17] that is supposed to be for a single gene of type $A$ within offspring genotypes and this represents an ill-posed mathematical problem, since the corresponding viability fitness parameters define relative survival rates of offspring genotypes rather than single genes. Their approach was designed to

be a simplification valid within the idiosyncratic logic of evolutionary game theory. However, disconnecting from the population genetics that underpin the mating systems yields confounded game-theoretical payoffs. The resultant error is borne out more fully when both fertility and viability selection are incorporated to the model.

Replicator equations in discrete time, or with discrete generations, describe mathematically the deterministic trajectory of the relative frequency of strategies in evolutionary games (Bishop and Cannings 1978). Discrete generations occur in the Fisher-Wright model of population genetics and our interest here is the translation to a game-theoretical setting (Brown 1983; Rowe 1987, 1988; Schreiber 2001; Imhof and Nowak 2006). The discrete replicator considered describes the allele dynamics for a population of infinite size with non-overlapping generations, for which reproduction in the life-cycle is amenable to a Binomial probability distribution on the number of $A$-genes that arise within the offspring genotypes. A haploid replicator requires two trials, whereas a diploid replicator requires four trials. A diploid genetic model distinguishes between fertility and viability selection. The mathematical and biological details of the corresponding model are clarified later in this article. For discussion of Mendelian inheritance in population genetics models similar to those studied here see Ewens (2004), in particular Sections 1.4, 2.6, and 3.2.

The contrived system of crosses in [17] used for a short-cut to calculate the offspring allele frequency yields a marginal expected payoff function that resembles a haploid replicator. Only three parent gametes are taken into account in this purported short-cut; the remaining gamete is omitted from participation in the crosses. Another step is then necessary to recover the offspring genotype frequencies. Namely, multiplication of the marginal expected payoff functions $\tilde{\pi}_A$ , $\tilde{\pi}_B$ by the omitted parent generation gamete frequencies, *x, 1-x,* respectively. The relative proportions of these adjusted products, $x\tilde{\pi}_A$ , $(1-x)\tilde{\pi}_B$, then yield the gene frequencies of the offspring generation. The required missing factor is recovered by an interchange of a haploid replicator equation and the simplistic marginal expected payoff function. In this article, such an interchange is shown to be invalid due to the use of diploid fitness parameters, which do not carry over to the haploid replicator.

This mathematical expediency confounds the translation between population genetics and game-theoretical models. Biologically the use of sex-unspecified single gametes and one parent genotype to derive offspring frequencies is error-prone in diploid models. Mathematically an abuse of notation yields erroneous functional forms of the difference replicators for sex specific fertility or viability selection. The present article demonstrates that the equivalence of random mating and random union of gametes, see for example Theorem 1.3.1 of Edwards (2000), will not in general yield equivalent haploid and diploid replicator dynamics. That is, unless there is intermediate

heterozygote advantage without dominance, in which case diploid genic selection is equivalent to haploid selection between two alleles. Disentangling this equivalence theorem from the *ad hoc* calculation of game-theoretical payoffs in [17] is the main source of error that is corrected here, although the implications go beyond that particular review article.

Theoretical biologists continue to explore blends of population genetics and evolutionary game theory (Van Cleve 2015). A research survey primer on population biology and modern game theory is Broom and Rychtář (2013). In general, the accurate use of population genetics with evolutionary game-theoretical models will apprehend lacunae in this rapidly expanding field. In this article, new probabilistic mating patterns facilitate the accurate use of population genetics and correct the mathematical description of the corresponding evolutionary dynamics.

## GAMES OF CROSSES WITH CONSTANT FITNESS PARAMETERS

### A multi-player game of viability selection :

Define a game of diploid crosses that corresponds to a projection of the standard 3×3 matrix for genotype crosses into a 2×4 payoff matrix, which is the two strategy four player game in equation (13) of [17] (*cf.* Broom and Rychtář 2013, pp. 162-5). This projection uses a probability rule to ensure correct accounting without overlap that is explained in the context of the derivations to follow. Consider the formation of the possible offspring genotypes. This requires a two-step randomization process. In the first step, this system forms its parent genotypes. Table 1 contains the allele payoffs from the possible pairings in the second step of the randomization, which corresponds to offspring from the diploid crosses. The payoff to the three possible genotypes are $\alpha$ for $(AA)$, $\beta$ for $(Aa)$ and $\gamma$ for $(aa)$. Clearly, there are two focus alleles in homozygotes but only one in the heterozygote. Thus, the payoff to the $A$-gene from $(AA)$ is $\alpha$ and from $(Aa)$ is $\beta/2$. The payoff to the $a$-gene from $(aa)$ is $\gamma$ and from $(Aa)$ is $\beta/2$. Thus, sexually symmetric viability selection, without fertility selection, is such that the offspring genotypes survive to reproductive maturity with rates, $\alpha$, $\beta$ and $\gamma$. Assume random mating, and that each particular type of cross produces an unbiased sex-ratio among the offspring on average. Equal gene frequencies in both sexes then hold, since otherwise these frequencies are immediately averaged in the offspring generation and remain equalized in subsequent generations. Remarks on the validity of unbiased gene frequencies are given in Section 3.3.

**Table 1.** Payoffs to $A$ and $a$ alleles in offspring corresponding to symmetric viability fitness α, β and γ for genotypes $(AA)$, $(Aa)$ and $(aa)$, respectively.

| | $AAA$ | $AAa$ | $Aaa$ | $aaa$ |
|---|---|---|---|---|
| $A$ | α | α/2 + β/4 | α/6 + β/3 | β/4 |
| $a$ | β/4 | β/3 + γ/6 | β/4 + γ/2 | γ |

**Calculation of viability payoffs :**

The only biologically sensible way to incorporate the viability fitness parameters in the model, namely α, β, γ, that appear in equations (15 and 16) of [17] is to consider the mating patterns. More specifically, viability fitness by definition denotes the relative rates of offspring survival. The proportions of offspring from the possible parent genotypes of each pairing in eqn. (15) are therefore required to calculate the corresponding payoffs. [17] correctly obtain the payoffs in their equation (15) for the pairings $A$, $AAA$ and $a$, $aaa$. All other entries in Table 1 are corrections. For instance, $A$ and $AAa$ yields the cross, $[(AA)\times(Aa)\,]$. This yields the offspring ratio, $\frac{1}{2}(AA):\frac{1}{2}(Aa)$. Thus, the payoff to the $A$-gene is, $\frac{\alpha}{2}+\frac{\beta}{4}$. Note [17] have multiplied this term by $\binom{4}{3}/\binom{3}{2}=\frac{4}{3}$ to obtain the corresponding entry in their payoff matrix, eqn. (15). The factor of ⅓ is an error, since it does not belong in the corresponding binomial coefficient of the marginal expected payoff.

Next, consider $A$ and $Aaa$, the crossing ratio is, $\frac{1}{3}[(AA)\times(aa)\,]:\frac{2}{3}[(Aa)\times(Aa)\,]$. The offspring ratios are then, all $(Aa)$ and $\frac{1}{4}(AA):\frac{1}{2}(Aa):\frac{1}{4}(aa)$. The payoff to the $A$-gene is therefore, $\frac{1}{3}\frac{\beta}{2}+\frac{2}{3}\left(\frac{1}{4}\,\alpha+\frac{1}{2}\frac{\beta}{2}\right)=\frac{1}{6}(\alpha+2\beta)$. Note multiplying this term by $\binom{4}{2}/\binom{3}{1}=2$ as [17] do would introduce another erroneous factor of ⅓. The next entry in the top line of Table 1 corresponds to $A$, $aaa$. This yields the cross, $[(Aa)\times(aa)]$, and the offspring ratio is $\frac{1}{2}(Aa):\frac{1}{2}(aa)$. The payoff to the $A$-gene is therefore, $\beta/4$. Note again multiplying this term by $\binom{4}{1}/\binom{3}{0}=4$ would coincidentally incorporate the correct binomial coefficient. Similar calculations yield the remaining payoffs to the $a$-gene in Table 1. Combinatorial sampling factors of parent genotype frequencies that are drawn from the population are thus correctly separated from payoffs.

Unstated in [17] is that the marginal expected allele payoff proceeds according to the binomial probability distribution with four trials, not three, and success probability $x$, where $x$ denotes the population frequency of the $A$-gene in the parent generation. That is, the five possible combinations of four parent alleles form a partition of unity, since there is a clear analogy with the number of successes in four binomial trials and the

number of focus alleles in each entry of Table 1 for a particular row. Note a zero payoff results once in each row and is omitted. Calculation of the marginal expected payoff for the alleles then yields,

$$\pi_A = x[\alpha x + \beta(1-x)]; \tag{2.2.1}$$

$$\pi_a = (1-x)[\beta x + \gamma(1-x)]. \tag{2.2.2}$$

Equations (2.2.1 - 2) correct equations (16) given by [17] that are linear in *x.* Importantly, although in agreement with the perspective of Section 5.2 from Crow and Kimura (2009), where equation 5.2.8 is due to Sewall Wright (i.e., the first equation on p. 371 in Wright, 1949), this is due to a conversion from relative to absolute marginal expected payoff and confounds the genetic interpretation. Namely, the calculation required to obtain (2.2.1 – 2) yields the payoff of a single allele. Division by the missing factor *x*, or (*1-x*), must proceed as a next step and cannot be obtained directly without violating the underlying population genetics.

**A multi-player game of fertility selection :**

Fertility selection is known to yield more complicated allele dynamics than viability selection. The payoff equations (2.4.1-2) of the evolutionary dynamics for asymmetric fertility selection show that the game-theoretical model is extendable. Consider sexually asymmetric fertility selection without viability selection. The fitness scheme is such that male fertility fitness is $\alpha_m$, $\beta_m$, $\gamma_m$ and female fertility fitness is $\alpha_f$, $\beta_f$, $\gamma_f$ for parent genotypes *(AA), (Aa)* and *(aa),* respectively. Offspring survival is neutral and all genotypes have viability fitness 1. When the crosses in the parent generation are enumerated the probabilistic rule of the formation must also consider the sex of the particular parent genotypes. The derivation is similar to that shown in the next model. The payoff matrix is described in Table 2.

**Table 2.** Payoffs to the $A$ and $a$ alleles in offspring, corresponding to asymmetric fertility selection without viability selection.

| | *AAA* | *AAa* | *Aaa* | *aaa* |
|---|---|---|---|---|
| $A$ | $\alpha_m\alpha_f$ | $\frac{3}{8}(\alpha_m\beta_f + \alpha_f\beta_m)$ | $\frac{1}{12}(\alpha_m\gamma_f + \alpha_f\gamma_m + 4\beta_m\beta_f)$ | $\frac{1}{8}(\beta_m\gamma_f + \beta_f\gamma_m)$ |
| $a$ | $\frac{1}{8}(\alpha_f\beta_m + \alpha_m\beta_f)$ | $\frac{1}{12}(\alpha_m\gamma_f + \alpha_f\gamma_m + 4\beta_m\beta_f)$ | $\frac{3}{8}(\beta_m\gamma_f + \beta_f\gamma_m)$ | $\gamma_m\gamma_f$ |

**Calculation of fertility payoffs :**

Calculation of the marginal expected payoff for the alleles then yields,

$$\pi_A = \frac{1}{2}x^4[2C_1 - 3C_2 + C_3 - C_4] + \frac{1}{2}x^3[3C_2 - 2C_3 + 3C_4] + \frac{1}{2}x^2[C_3 - 3C_4] + \frac{1}{2}xC_4; \tag{2.4.1}$$

$$\pi_a = \frac{1}{2}x^4[-C_2 + C_3 - 3C_4 + 2C_5] + \frac{1}{2}x^3[C_2 - 2C_3 + 9C_4 - 8C_5] + \frac{1}{2}x^2[C_3 - 9C_4 + 12C_5] + \frac{1}{2}x[3C_4 - 8C_5] + C_5, \tag{2.4.2}$$

where $C_1 = \alpha_m\alpha_f$, $C_2 = \alpha_m\beta_f + \alpha_f\beta_m$, $C_3 = \alpha_m\gamma_f + \alpha_f\gamma_m + 4\beta_m\beta_f$, $C_4 = \beta_m\gamma_f + \beta_f\gamma_m$, $C_5 = \gamma_m\gamma_f$.

Thus, the marginal expected payoffs of (2.4.1-2) are quartic in *x* with six parameters; symmetric fertility selection yields a quartic in *x* with three parameters. When symmetric viability fitness is found with asymmetric fertility selection the marginal expected payoffs are quartic in *x*. The coefficients are then sums of certain positive or negative triples of eight out of the nine parameters, each triple has two fertility parameters and one viability parameter. In the next model, payoffs from offspring viability fitness depend on the parent genotypes.

## FERTILITY-VIABILITY SELECTION

**A multi-player game of an epi-static interaction :**

Experimental evolution with the flour beetle *Tribolium castaneum* demonstrates asymmetric sexual selection yields resilience to the mutational load that naturally accumulates under inbreeding, and when unmitigated is eventually catastrophic (Lumley *et al.* 2015). The Medea system (Wade and Beeman 1994), maternal effect dominant embryonic arrest alleles, is a model of interaction between fertility and viability selection applied to this species. Medea alleles were also discovered in mice, *Mus musculus domesticus,* (Weichenhan *et al.* 1996). Stability analysis of the Medea system in a game-theoretical model applied to transgenic constructs for population transformation in genetic pest management is undertaken by Gokhale *et al.* (2014). Their analysis also does however rely upon a similar mathematical expediency that was falsified in Section 2. The correct game-theoretical model that describes the Medea mating system is derived in the present Section.

This sex-chromosome linked effect is inherited as an autosomal gene such that a hazard rate, $0 \leq t \leq 1$, to offspring is only switched on if the female parent is heterozygous. In

this case, the viability fitness of the *(++)* homozygote offspring decreases from 1 to 1-t, whereas the viabilities of both the *(M+)* heterozygote and *(MM)* homozygote offspring remain constant at 1. There is a fertility cost for carrying the Medea gene that affects female parents only. Namely, the fertility fitness of the female parent genotypes $(MM)^{\varphi}$, $(M+)^{\varphi}$ and $(++)^{\varphi}$ are ν, μ and 1, respectively. (Note the parameter μ replaces use of ω in [17].) There is a dominance parameter for female fertility fitness, $0 \leq h \leq 1$, such that $\nu = 1 - s$, and $\mu = 1 - hs$, where $0 < s < 1$ is the female fertility cost parameter. It is possible to consider a negative cost (or benefit) similarly. Male parent genotypes all reproduce at the same rate, so that male fertility fitness is always equal to 1. Thus, in this system there is an evolutionary genetic trade-off against female parents that carry the Medea gene who suffer reduced fertility, and a viability hazard suffered by offspring that do not carry the Medea gene; hence this is also known as a type of selfish gene.

**Calculation of epi-static payoffs :**

Consider the life cycle. First, the parent generation reproduces. Second, their embryonic offspring faces the viability hazard. Thus offspring must survive viability selection to then eventually constitute the next generation of parents. Details for informative pairings are shown here. *M* and *MM+* yields the crossing ratio, $\frac{1}{2}\left[(MM)^{\varphi} \times (M+)^{\sigma}\right]:\frac{1}{2}\left[(MM)^{\sigma} \times (M+)^{\varphi}\right]$. The corresponding offspring ratio is $\frac{1}{2}(MM):\frac{1}{2}(M+)$, in both cases. The payoff to the *M*-gene is therefore, $\frac{1}{2}\left[\nu\left(\frac{1}{2}+\frac{1}{4}\right)\right]+\frac{1}{2}\left[\mu\left(\frac{1}{2}+\frac{1}{4}\right)\right]=\frac{3}{8}(\nu+\mu)$, which corrects that given in eqn. (18) by [17]. The remaining expressions shown in Table 3 are also corrections, except for the pairings *M, MMM* and *+, +++*. For instance, *M* and *M++* yields the crossing ratio, $\frac{1}{3}[\frac{1}{2}\left[(MM)^{\sigma} \times (++)^{\varphi}\right]:\frac{1}{2}\left[(MM)^{\varphi} \times (++)^{\sigma}\right]]:\frac{2}{3}\left[(M+)^{\sigma}\times(M+)^{\varphi}\right]$. The corresponding offspring ratios are, firstly all (*M+*) in both cases and secondly $\frac{1}{4}(MM):\frac{1}{2}(M+):\frac{1}{4}(++)$. The payoff to the *M* gene is therefore, $\frac{1}{3}\left[\frac{1}{4}+\frac{1}{4}\nu\right]+\frac{2}{3}\left[\mu\left(\frac{1}{4}+\frac{1}{4}\right)\right]=\frac{1}{12}(1+\nu+4\mu)$.

The viability hazard is realized in the following two pairings. *+* and *M++* yields the crossing ratio, $\frac{1}{2}\left[(M+)^{\varphi}\times(++)^{\sigma}\right]:\frac{1}{2}[(M+)^{\sigma}\times(++)^{\varphi}]$. The corresponding offspring ratio is $\frac{1}{2}(M+):\frac{1}{2}(++)$, in both cases. In the first case, (++) faces a hazard with rate *t*, since its female parent is a heterozygous carrier of Medea. In the second

case the hazard is switched off since its female parent is homozygous. The payoff to the $+$ gene is therefore, $\frac{1}{2}\left[\mu\left(\frac{1}{4}+\frac{(1-t)}{2}\right)\right]+\frac{1}{2}\left[\frac{1}{4}+\frac{1}{2}\right]=\frac{3}{8}(1+\mu-\frac{2}{3}\mu t)$. The pairing $+$ and $MM+$ yields the same crossing ratio as for $M$ and $M++$, shown in the preceding paragraph. The payoff to the $+$ gene is therefore, $\frac{1}{3}\left[\frac{1}{4}\nu+\frac{1}{4}\right]+\frac{2}{3}\left[\mu\left(\frac{1}{4}+\frac{1}{4}(1-t)\right)\right]=\frac{1}{12}(1+\nu+4\mu-2\mu t)$. Similar calculations, in which the hazard does not appear, give the remaining payoffs of Table 3.

**Table 3.** Payoffs to *M* and $+$ alleles in the Medea system. Fertility fitness is ν, μ and 1 for female parent genotypes (*MM*), (*M*+) and (++), respectively. Male parent genotypes all reproduce with rate 1. Offspring that are homozygous for the non-Medea gene and have a female heterozygous parent face a viability hazard rate *t.*

| | *MMM* | *MM+* | *M++* | *+++* |
|---|---|---|---|---|
| *M* | $\nu$ | $\frac{3}{8}(\nu+\mu)$ | $\frac{1}{12}(1+\nu+4\mu)$ | $\frac{1}{8}(1+\mu)$ |
| $+$ | $\frac{1}{8}(\nu+\mu)$ | $\frac{1}{12}(1+\nu+4\mu-2\mu t)$ | $\frac{3}{8}\left(1+\mu-\frac{2}{3}\mu t\right)$ | 1 |

The marginal expected payoffs are then obtained in the same way as that described for (2.2.1-2), and in terms of genotype fitness,

$$\pi_M=\tfrac{1}{2}x^3(1+\nu-2\mu)-\tfrac{1}{2}x^2(2-\mu-\nu)+\tfrac{1}{2}x(1+\mu); \quad (3.2.1)$$

$$\pi_+=x^3\left[\mu(1-t)-\tfrac{1}{2}(1+\nu)\right]-x^2\left[\mu\left(\tfrac{5}{2}-2t\right)-2-\tfrac{1}{2}\nu\right]+x\left[\mu\left(\tfrac{3}{2}-t\right)-\tfrac{5}{2}\right]+1, \quad (3.2.2)$$

where *x* now denotes the population frequency of the *M*-gene in the parent generation. Equations (3.2.1-2) correct equations (19) of Gokhale and Traulsen (2014). Note also these results can be shown to correct equations (1) and (A.1) of Gokhale *et al.* (2014), primarily seen from the fact that both fertility fitness parameters should be present in the first two cases of their equations.

Writing equations (3.2.1-2) in terms of the genetic parameters,

$$\pi_M=\ x\left[x^2s\left(h-\tfrac{1}{2}\right)-\tfrac{1}{2}xs(h+1)+1-\tfrac{1}{2}hs\right]; \quad (3.2.3)$$

$$\pi_+ = 1 + x\left[x^2\left[s\left(\frac{1}{2} - h(1-t)\right) - t\right] - x\left[s\left(\frac{1}{2} - h\left(\frac{5}{2} - 2t\right)\right) - 2t\right] - 1 - hs\left(\frac{3}{2} - t\right) - t\right].$$

(3.2.4)

The payoffs in (3.2.1-2) and (3.2.3-4) from the constant selection on viability and fertility, rather than frequency-dependent selection, are thus cubic functions of *x.* The gene frequencies of the offspring survivors are obtained as the relative proportions of these payoffs summation, $p'_M = \pi_M/[\pi_M + \pi_+]$. The deterministic trajectory of the resultant gene frequencies from generation to generation can then be quantified using the differences. Recall that $p_M = x$, and thus,

$$\Delta p_M = p_M - p'_M$$

$$= \frac{x[x^3[t(hs-1)] + x^2\left[2t(1-hs) + s\left(h - \frac{1}{2}\right)\right] - x[t(1-hs) + \frac{1}{2}s(3h-1)] + \frac{1}{2}hs]}{1 - x[x^2t(1-hs) - x[s(2h-1) + 2t(1-hs)] + 2hs + t(1-hs)]}$$

(3.2.5)

Equation (3.2.5) is quartic in *x,* and gives the change in frequency of the *M*-gene between the parent generation and the surviving offspring generation.

This game-theoretical model therefore verifies equations (1, 2 and 3) in Wade and Beeman (1994), which describe the offspring genotype frequencies, population fitness and change in allele frequency. Observe the factors of ½ in their equations are necessary due to the population frequency of the heterozygous parent, $D_{M+} = 2p_D(1-p_D)$. Their derivation relies on a table of all possible genotypic crosses.

**Male and female frequency of the M-gene :**

Dioecious populations admit two sexes, in which case the random union of gametes requires modification from autosomal loci compared to sex-linked loci (Ewens 2004, Sections 2.2-3; Eshel and Feldman 1982). An intricacy of the Medea system, equation (3.3.2), is shown here in the context of sex-biased allele frequency. Let $p_m$ and $p_f$ denote the male and female frequency of the *M*-gene in the parent generation. Then calculate a weighted sum of the genotype frequencies taken over all crosses, where the weights are fitness. Note there is a combinatorial factor $\binom{2}{1}$ for each occurrence of the heterozygous parent in the crosses. The proportions of offspring genotypes must also be factored into this weighted sum. Thus, the expected population fitness of the offspring generation is,

$$1 - p_f[p_m(hst - t)(1 - p_f) + (2hs - hst + t)(1 - p_f) + sp_f]. \quad (3.3.1)$$

The corresponding expression for the frequency of the *M*-gene in offspring survivors,

$p'_M = \mathrm{P}'(MM) + \frac{1}{2}\mathrm{P}'(M+)$, as a function of the male and female *M*-gene frequencies in the parent generation, has numerator,

$$\tfrac{1}{2}\left[p_m + p_f - sp_f^2 - sp_m p_f^2 + hsp_f^2 - hsp_f\right] - hsp_m p_f\left(1 - p_f\right), \tag{3.3.2}$$

and denominator (3.3.1). Remember this offspring generation is then equalized on average such that, $p'_m = p'_f = p'_M$. In subsequent generations the expressions (3.3.1) and (3.3.2) continue to hold with the iterative replacement, $p'_M \stackrel{\text{def}}{=} x$; the surviving offspring generation becomes the next parent generation. The corresponding expressions are then the denominator of (3.2.5) and the payoff $\pi_M$ in (3.2.3). Thus the assumption of the same gene frequencies between the sexes among surviving offspring over the generations is valid.

## CONCLUSION

Evolutionary game theory is the study of frequency-dependent selection in a strictly limited sense. The following caveat is required: the marginal strategic payoffs to players (or expected fitness of strategies) are modified from one generation to the next, whereas population geneticists define frequency-dependent selection with the genetic parameters of fitness functions of *x*. Equations 3.2.3-4 show that the genic selection of Medea is frequency-dependent in the former sense, yet defined and parameterized by constant fertility and viability genotypic selection in parent and offspring generations.

The evolutionary multi-player games suggested in [17] create an oversimplified interpretation of the Medea system. The correct derivation given here yields the payoff matrix (Table 3) of the four (parent) players in each round of a game with two strategies. The marginal expected payoffs for the corresponding Medea system (3.2.1-2) are found to involve a non-trivial increase of one degree in *x*. A simpler correction is necessary for the evolutionary dynamics of an allele subject to diploid symmetric viability selection only (Table 1; Equations 2.2.1-2). The game-theoretical model developed is extendable and this is shown for asymmetric fertility selection (Table 2, Equations 2.4.1-2). These results represent a logical thought progression in two separate ways. First, in Section 2, a direct proof is obtained for the probabilistic mating patterns necessary due to avoid violation of population genetics constraints. Second, in Section 3, a proof by contradiction is obtained that an identical heuristic game-theoretical model proposed in [17] under the same assumptions of the model in Section 2 is inconsistent with population genetic constraints. Note also that normalization of the fitness parameters $\alpha.$, $\beta.$ and $\gamma.$ in each of the models described simplifies equivalently to at least one unitary and at most two non-unitary fitness parameter values.

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