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# Kin selection and coefficients of relatedness in family-structured populations with inbreeding $\stackrel{\text{tructured}}{\Rightarrow}$

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#### Abstract

We consider family specific fitnesses that depend on mixed strategies of two basic phenotypes or behaviours. Pairwise interactions are assumed, but they are restricted to occur between sibs. To study the change in frequency of a rare mutant allele, we consider two different forms of weak selection, one applied through small differences in genotypic values determining individual mixed strategies, the other through small differences in viabilities according to the behaviours chosen by interacting sibs. Under these two specific forms of weak selection, we deduce conditions for initial increase in frequency of a rare mutant allele for autosomal genes in the partial selfing model as well as autosomal and sex-linked genes in the partial sib-mating model with selection before mating or selection after mating. With small differences in frequency obtained in additive kin selection models. With particular reference to altruism versus selfishness, we provide explicit ranges of values for the selfing or sib-mating rate based on a fixed cost-benefit ratio and the dominance scheme that allow the spreading of a rare mutant allele into the population. This study confirms that more inbreeding does not necessarily promote the evolution of altruism. Under the hypothesis of small differences in viabilities, the situation is much more intricate unless an additive model is assumed. In general however, conditions for initial increase in frequency of a mutant allele can be obtained in terms of fitness effects that depend on the genotypes of interacting individuals or their mates and generalized conditional coefficients of relatedness according to the inbreeding condition of the interacting individuals. © 2004 Elsevier Inc. All rights reserved.

Keywords: Partial selfing; Partial sib-mating; Kin selection; Relatedness

#### 1. Introduction

This paper is an attempt to explain evolutionary properties in family-structured populations with inbreeding from a kin selection perspective. To this end, inbreeding caused by partial selfing or partial sib-mating and individual fitness based on mixed strategies used in interactions between sibs will be considered. More precisely, the fitness of an individual will depend upon the choice between two possible pure strategies (phenotypes or behaviours), which choice is a probability distribution determined by the genotype of the individual, and by the corresponding choice of one of its sibs chosen at random and interacting with it. Pairwise interactions are classical in ESS theory (Maynard Smith and Price, 1973), but in this paper they will be restricted to occur only between individuals within the same sibship in order to study the effect of inbreeding on kin selection theory. The resulting fitnesses can be interpreted as family specific

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genotypic fitnesses in the sense of Michod (1980), who considered both the partial selfing model and the partial sibmating model at an autosomal locus with two alleles, one of which coding for an altruistic behaviour (see also Michod and Abugov, 1980, for the random mating case), allowing the genotypic fitnesses to vary from one family type to another. Michod (1980) used numerical calculations and simulations to study local stability conditions at both fixation states under the assumption that costs and benefits of altruistic acts combine additively or multiplicatively. Wade and Breden (1981) also studied the partial sib-mating model with additive genotypic fitnesses but from a group selection perspective.

Both Michod (1980) and Wade and Breden (1981) argued that inbreeding should facilitate the evolution of an altruistic allele that enhances the fitness of kin at the expense of its carrier, although the first author gave an example of extreme altruism in the multiplicative model for which the initial increase in frequency of a rare altruistic allele is not made easier by more inbreeding. Actually, it was inferred that inbreeding should promote altruism by increasing the between-family genetic variance while decreasing the within-family genetic variance. However, Uyenoyama (1984) showed that, even in additive models, more inbreeding may apparently increase the within-family variance by producing rare homozygotes at a higher frequency, resulting in more stringent conditions for the initial invasion of an altruistic allele. Besides restricting her study to the additive model for altruism with mating schemes mixing selfing, parthenogenesis or sib-mating with random mating, Uyenoyama (1984) assumed at least one of the following additional conditions in order to avoid hidden non-additive effects: (a) the sexes are indistinguishable with respect to fitness, (b) the relative frequencies of males within families are unaffected by selection, or (c) inbreeding females are inseminated before selection. Conditions for initial increase in frequency and polymorphic equilibrium were reported for all models but it was assumed that the heterozygote did not exhibit overdominance or underdominance in the propensity to perform altruism. In this paper, we will focus on conditions for initial increase and assume either (a) or (c) above but we will not restrict the analysis to additive models for fitness effects or cases of no dominance or complete dominance in genotypic values.

Another issue is the appropriate definition for coefficients of relatedness in inbred populations to measure the genetic relationship of an individual, possibly inbred, to another. The coefficient of relatedness of an individual X to an individual Y, denoted by  $R_{X \to Y}$ , has been generally defined in populations with inbreeding as a covariance ratio, actually the covariance between the frequency of a given allele in Y and a given genotypic value at the same locus in X over the covariance between these two quantities in X (Michod and Hamilton, 1980; see also Uyenoyama et al., 1981, for a definition in a context of multiple alleles at a single locus with the additive genotypic value replacing the frequency of a given allele). In cases of inbreeding caused by partial selfing or partial sib-mating and in the absence of selection, it has been shown that such a coefficient of relatedness reduces to a pedigree index that does not depend on dominance or gene frequency (Lessard, 1990, 1992; see also Uyenoyama and Bengtsson, 1982), actually the coefficient of kinship between X and Y over the coefficient of kinship between X and itself, the coefficient of kinship between X and Y, denoted by  $f_{XY}$ , being defined as the probability that a gene chosen at random in Y be identical by descent (i.b.d.) to a gene chosen at random at the same locus in X (Malécot, 1948). Actually, in this case, this pedigree index gives the expected fraction of genes in Y i.b.d. to one or more genes in X at the same locus and, if X is diploid, this fraction is the same given that X is inbred or given that X is outbred. This pedigree index corresponds also to the coefficient of regression of the frequency of a given allele in Y to the frequency of the same allele in X (Hamilton, 1972). In the symmetric case, with X and Y having the same ploidy at the locus considered, this reduces to a coefficient of correlation, actually Wright's (1922) coefficient of relationship. The role and limitation of such coefficients to predict evolution in populations with inbreeding are addressed in this paper.

In a previous paper (Lessard and Rocheleau, 2003), we studied the change in frequency of a rare mutant allele in partially inbred populations in the case where the fitness of an individual depends only on its own genotype and we ascertained two basic results that we shall use in the present paper. These allow an approximation for the change in frequency of a mutant allele under weak selection, when introduced in small frequency into a structured infinite population, which was previously at a fixation state. The weak selection hypothesis is rendered through a parameter *s*, which measures the intensity of selection and which is assumed to be positive and small (s = 0 corresponding to selective neutrality). Also, it is supposed that the population state can be described by a vector whose entries represent the frequencies of group types (in this paper, genotypes or mating types) carrying the mutant allele. Moreover, the linear approximation for the transformation of this vector near the origin from one generation to the next is given by a non-negative matrix  $\mathbf{M}(s)$ . This matrix  $\mathbf{M}(s)$  is assumed to be smooth enough with respect to *s* and to have at least some power that exhibits only positive entries. Under these hypotheses, the leading eigenvalue of  $\mathbf{M}(s)$ , denoted by  $\lambda(s)$ , will determine the fate of the mutant allele as long as this allele remains rare enough in the population. We reproduce below the two basic results (see, e.g., Taylor, 1985, 1989, for similar statements, and Lessard and Rocheleau, 2003, for formal proofs).

**Result 1.** For s small enough, the leading eigenvalue of  $\mathbf{M}(s)$  is approximated by

$$\lambda(s) \cong 1 + Ls,\tag{1}$$

where

$$L = \frac{\xi(0)^T \,\mathbf{\dot{M}}(0)\eta(0)}{\xi(0)^T \eta(0)} \tag{2}$$

with  $\mathbf{M}(0)$  being the derivative of  $\mathbf{M}(s)$  with respect to s evaluated at s = 0,  $\boldsymbol{\xi}(0)$  and  $\boldsymbol{\eta}(0)$  being respectively left and right (positive) eigenvectors of  $\mathbf{M}(0)$  associated to the eigenvalue 1 and T denoting matrix transposition.

The approximation in Result 1 is valid up to terms of order *s*. The second result, which is more important from a biological point of view, provides an approximation for the change in frequency of a rare mutant allele from one generation to the next.

**Result 2.** Let  $p^{(k)}$  be the frequency of a rare mutant allele at generation k in a population initially at fixation. Under weak selection (s small enough) and for k sufficiently large (but not too large in the case L > 0 to ensure that  $p^{(k)}$  remains small enough), the change in frequency of the mutant allele from generation k to generation k + 1 is approximated by

$$\Delta p^{(k)} \cong L p^{(k)} s. \tag{3}$$

The approximation in Result 2 ignores terms of order  $p^{(k)}s^2$  or  $(p^{(k)})^2s$  and all other smaller terms. Owing to Results 1 and 2, the sign of the derivative of  $\lambda(s)$  evaluated at s = 0, denoted by L, will predict the fate of the mutant allele when introduced in small frequency into the population: L < 0 will entail extinction, while L > 0 will imply protection. The quantity L can be seen as a rate of increase (if positive) or decrease (if negative) for the frequency of a rare mutant allele with respect to the frequency of this allele and the intensity of selection.

In this paper, to apply Results 1 and 2 presented above, two different forms of weak selection will be considered: one applied through small differences in genotypic values determining individual mixed strategies and one through small differences in fitness parameters according to the pure strategies chosen by interacting individuals. Section 2 describes the consequences of these hypotheses on the genotypic fitnesses. In particular, it is shown that the first form of weak selection is formally equivalent to the second form with an additive model for the fitness parameters. However, general effects on genotypic fitnesses including multiplicative effects can be taken into account under the second form of weak selection. Under either form of weak selection, no hypothesis about the propensity for an individual to adopt either of the pure strategies as complete dominance or no dominance is made.

In the next sections, some specific models with regular systems of mating that create inbreeding are considered. Section 3 is devoted to the study of the partial selfing model under the two aforementioned forms of weak selection. In the case of inbreeding caused by partial sib-mating, selection at an autosomal locus is considered first: in Section 4, selection takes place before mating as in a classical viability model while, in Section 5, selection occurs after mating but before reproduction. This latter assumption corresponds to a multiplicative fertility model. When there is inbreeding, this assumption is not expected to lead to the same recurrence equations and results than the assumption of selection before mating (see, e.g., Pollak, 1995; Caballero, 1996), contrary to what occurs in the case of random mating (see, e.g., Karlin, 1968). This assumption is also considered in the case of selection at a sex-linked locus in Section 6. For all models considered, the rate of change L for the frequency of a rare mutant allele is expressed in terms of coefficients of relatedness, whose definition is extended, and interpreted in a kin selection perspective. In the case of altruism that imposes specific inequalities between the fitness parameters and under the assumption of small differences in strategy parameters, conditions for protection of an altruistic allele according to the value of a cost-benefit ratio are given in Section 7. The meaning of the results is discussed in Section 8. All the recurrence equations and technical details are deferred to Appendix A.

#### 2. Family specific fitnesses based on individual strategies

We consider a special type of family specific fitnesses (see, e.g., Michod, 1980, and references therein). Suppose that an individual can choose between two possible phenotypes or behaviours, called pure strategies and represented by 1 and 2. In a kin selection context for instance, it might be an altruistic behaviour versus a selfish behaviour. In a diploid population with two alleles,  $A_1$  and  $A_2$ , segregating at an autosomal locus, an individual of genotype  $A_iA_j$  either chooses strategy 1 with probability  $h_{ij}$  (= $h_{ji}$ ) or strategy 2 with complementary probability  $1 - h_{ij}$ . Then, the genotypic value  $h_{ij}$  corresponds to a mixed strategy. As usual, there is complete dominance if  $h_{12} = h_{11}$  or  $h_{22}$ , underdominance if  $h_{12} < h_{11}$  and  $h_{22}$ , overdominance if  $h_{12} > h_{11}$  and  $h_{22}$ , and no dominance if  $h_{12} = (h_{11} + h_{22})/2$ .

Furthermore, random pairwise interactions between individuals are assumed but they occur only between individuals within the same sibship. As a mere consequence, this implies that the fitness of an individual will depend not only upon its own genotype but also upon the genotype of one of its sibs chosen at random, which in turn depends upon the genotypes of their parents. More explicitly, sibships are assumed to be of infinite size and the fitness of an  $A_iA_j$  individual within a sibship produced by parents of genotypes  $A_iA_k$  and  $A_jA_l$ , denoted by  $f_{ij:ik \times jl}$  (= $f_{ij:jl \times ik}$ ), takes the form

$$f_{ij:ik\times jl} = h_{ij}[h_{ik\times jl}m_{11} + (1 - h_{ik\times jl})m_{12}] + (1 - h_{ij})[h_{ik\times jl}m_{21} + (1 - h_{ik\times jl})m_{22}],$$
(4)

where  $h_{ik\times jl}$  designates the probability that a sib chosen at random adopts strategy 1 and  $(1 - h_{ik\times jl})$  the probability that it chooses strategy 2, while the parameter  $m_{uv} \ge 0$  (u, v=1, 2) denotes the fitness of an individual adopting strategy u when in interaction with a sib adopting strategy v. In general,  $m_{12} \ne m_{21}$  unless stated otherwise. Notice that we have the equalities

$$\begin{aligned} h_{11\times11} &= h_{11}, \quad h_{11\times12} = \frac{1}{2}h_{11} + \frac{1}{2}h_{12}, \\ h_{11\times22} &= h_{12}, \quad h_{12\times12} = \frac{1}{4}h_{11} + \frac{1}{2}h_{12} + \frac{1}{4}h_{22}, \\ h_{12\times22} &= \frac{1}{2}h_{12} + \frac{1}{2}h_{22}, \quad h_{22\times22} = h_{22}. \end{aligned}$$

$$(5)$$

On the other hand, the special relation

$$m_{11} - m_{12} = m_{21} - m_{22},\tag{6}$$

which can also be written as

$$m_{11} - m_{21} = m_{12} - m_{22},\tag{7}$$

defines an *additive model* with respect to the fitness parameters. In such a model, the increments or decrements in fitness following changes in the pure strategies chosen by interacting individuals combine additively. If they would rather combine multiplicatively for instance, so that

$$\frac{m_{11}}{m_{12}} = \frac{m_{21}}{m_{22}} \tag{8}$$

or equivalently

$$\frac{m_{11}}{m_{21}} = \frac{m_{12}}{m_{22}},\tag{9}$$

then we would be in the presence of a *multiplicative model*. Such relations will not be assumed in the following unless stated otherwise.

Fitnesses analogous to (1) can be defined in a context of a sex-linked locus (or equivalently, for a haplo-diploid population). If females are diploid and males haploid at the concerned locus, then the possible genotypes for females are given by  $A_1A_1$ ,  $A_1A_2$ ,  $A_2A_2$ , whereas for males, they are  $A_1$  and  $A_2$ . A female of genotype  $A_iA_j$  either chooses strategy 1 with probability  $h_{ij}$  or strategy 2 with complementary probability  $1-h_{ij}$ , while a male of genotype  $A_i$  either chooses strategy 1 with probability  $h_i$  or strategy 2 with complementary probability  $1-h_i$ . By analogy with Eq. (4), the female and male fitnesses are expressed as

$$f_{ij:ik\times j} = h_{ij}[h_{ik\times j}m_{11} + (1 - h_{ik\times j})m_{12}] + (1 - h_{ij})[h_{ik\times j}m_{21} + (1 - h_{ik\times j})m_{22}]$$
(10)

and

$$f_{i:ik\times j} = h_i [h_{ik\times j}m_{11} + (1 - h_{ik\times j})m_{12}] + (1 - h_i)[h_{ik\times j}m_{21} + (1 - h_{ik\times j})m_{22}],$$
(11)

respectively, where

$$h_{11\times 1} = \frac{1}{2}h_{11} + \frac{1}{2}h_1, \quad h_{12\times 1} = \frac{1}{4}h_{11} + \frac{1}{4}h_{12} + \frac{1}{4}h_1 + \frac{1}{4}h_2, \quad h_{22\times 1} = \frac{1}{2}h_{12} + \frac{1}{2}h_2, \\ h_{11\times 2} = \frac{1}{2}h_{12} + \frac{1}{2}h_1, \quad h_{12\times 2} = \frac{1}{4}h_{12} + \frac{1}{4}h_{22} + \frac{1}{4}h_1 + \frac{1}{4}h_2, \quad h_{22\times 2} = \frac{1}{2}h_{22} + \frac{1}{2}h_2.$$

$$(12)$$

This is assuming a one-to-one sibship sex-ratio and pairwise interactions between sibs irrespective of sex. Other assumptions could easily be incorporated. For the sake of convenience, we let the allele designated by *i* in (10) and (11) be the one transmitted by the mother. A more precise but cumbersome notation might have been used to identify clearly the origin of the alleles, since the female fitness  $f_{ij:ik \times j}$  is not generally equal to  $f_{ij:jk \times i}$  (the same fact prevails for the male fitnesses).

To approximate the change in frequency of a mutant allele when rare in the population, weak selection will be assumed in order to apply the aforementioned Results 1 and 2. A first form of weak selection is obtained in the case of small differences in strategy parameters.

## Assumption A.

$$h_{ij} = h + c_{ij}s \ (h_i = h + c_is, \text{ for males at a sex-linked locus}).$$
(13)

The parameter *s* is positive and small and measures the intensity of selection, while 0 < h < 1 is a reference value. Notice that there are no constraints on the parameters  $c_{ij}$  and  $c_i$  except the symmetry condition  $c_{ij} = c_{ji}$  for all *i*, *j*. When allele  $A_1$  is rare, the differences

$$d_{11} = c_{11} - c_{22}, \quad d_{12} = c_{12} - c_{22} \tag{14}$$

and

$$d_1 = c_1 - c_2 \tag{15}$$

in males at a sex-linked locus, will come into play. These are rates of strategy changes from one genotype to another with respect to an increase in the intensity of selection. Notice that  $d_{11}$  and  $d_{12}$  are of the same sign in the case of no dominance.

Under Assumption A, the following quantities will be considered:

$$K(h) = h^{2}m_{11} + h(1 - h)(m_{12} + m_{21}) + (1 - h)^{2}m_{22},$$
  

$$A(h) = h(m_{11} - m_{21}) + (1 - h)(m_{12} - m_{22}),$$
  

$$B(h) = h(m_{11} - m_{12}) + (1 - h)(m_{21} - m_{22}).$$
(16)

We can interpret K(h) as the mean fitness in the population when all individuals use the same mixed strategy h. In such a population, the rate of increase in the fitness of an individual with respect to an initial increase in the probability for this individual to adopt strategy 1 is given by A(h), while the rate of increase in the fitness of an individual with respect to an initial increase in the probability for a sib interacting with this individual to adopt strategy 1 is given by B(h). Initially increasing the probability of using strategy 1 is beneficial (detrimental) to the individual doing it when A(h) is positive (negative) and beneficial (detrimental) to a sib interacting with this individual if B(h) is positive (negative). We are in a context of altruism, strategy 1 corresponding to an altruistic behaviour and strategy 2 to a selfish one, when A(h) is negative and B(h) positive. Notice that, when the model is additive, we have A(h) = A and B(h) = B, where

$$A = m_{11} - m_{21} = m_{12} - m_{22} \tag{17}$$

and

$$B = m_{11} - m_{12} = m_{21} - m_{22} \tag{18}$$

do not depend on *h*.

A second form of weak selection is obtained in the case of small differences in fitness parameters.

#### Assumption B.

$$m_{ij} = 1 + u_{ij}s.$$
 (19)

Again, the parameter s is positive and small and measures the intensity of selection. In general, we have  $u_{ij} \neq u_{ji}$  when  $i \neq j$ , unless stated otherwise. Under Assumption B, the following quantities will come into play:

$$a(h_{ij}) = h_{ij}(u_{11} - u_{21}) + (1 - h_{ij})(u_{12} - u_{22}),$$
  

$$b(h_{ij}) = h_{ij}(u_{11} - u_{12}) + (1 - h_{ij})(u_{21} - u_{22}).$$
(20)

This time,  $a(h_{ij})$  represents the rate of increase in the fitness of an individual with respect to initial increases in the intensity of selection and the probability for this individual to adopt strategy 1 when the individual with which it interacts uses strategy  $h_{ij}$ , while  $b(h_{ij})$  gives the rate of increase in the fitness of an individual using strategy  $h_{ij}$  with respect to initial increases in the intensity of selection and the probability for a sib interacting with this individual to adopt strategy 1. Similarly, to include the haplo-diploid case, we define

$$a(h_i) = h_i(u_{11} - u_{21}) + (1 - h_i)(u_{12} - u_{22}),$$
  

$$b(h_i) = h_i(u_{11} - u_{12}) + (1 - h_i)(u_{21} - u_{22}).$$
(21)

Under Assumption B, the rates of increase in fitness of interacting sibs depend on their exact genotypic values unless the model is additive, in which case  $a(h_{ij}) = a(h_i) = a$  and  $b(h_{ij}) = b(h_i) = b$  for all *i* and *j*, where

$$a = u_{11} - u_{21} = u_{12} - u_{22} \tag{22}$$

and

b

$$= u_{11} - u_{12} = u_{21} - u_{22} \tag{23}$$

do not depend on the genotypic value. Then, up to terms of order s, the family specific genotypic fitness defined in Eq. (4) is approximated by

$$f_{ii:ik\times il} \cong m_{22} + [h_{ij}a + h_{ik\times jl}b]s, \tag{24}$$

which is in the same form as the corresponding approximation in the case of Assumption A given by

$$f_{ij:ik\times il} \cong K(h) + [c_{ij}A(h) + c_{ik\times jl}B(h)]s, \tag{25}$$

where  $c_{ik \times jl}$  is defined with respect to  $c_{ij}$ ,  $c_{il}$ ,  $c_{kj}$  and  $c_{kl}$  in the same way as  $h_{ik \times jl}$  with respect to  $h_{ij}$ ,  $h_{kl}$ ,  $h_{kj}$  and  $h_{kl}$  in Eq. (5). Similar approximations hold for the fitnesses in Eqs. (10) and (11) in the haplo-diploid case. Therefore, there is a direct correspondence between Assumption B in the additive case and Assumption A. Moreover, if b = 0, the model reduces to the case of no interaction between sibs.

#### 3. Partial selfing: selection at an autosomal locus before mating

Consider a single autosomal locus with two alleles,  $A_1$  and  $A_2$ , in an infinite diploid, hermaphrodite population undergoing discrete, non-overlapping generations. Assume that every individual of the population can reproduce with single insemination, either by selfing with probability  $\alpha$  ( $0 < \alpha < 1$ ), or by random outcrossing with complementary probability  $1 - \alpha$ . Let  $P_{11}$ ,  $P_{12}$  and  $P_{22}$  designate the frequencies of the genotypes  $A_1A_1$ ,  $A_1A_2$  and  $A_2A_2$ , respectively, among the adults of the current generation, after selection but before mating. Following mating, reproduction and selection according to the fitnesses defined in (4), the genotypic frequencies in the population among the adults of the next generation are described by the recurrence equations given in Appendix A.2.

#### 3.1. Small differences in strategy parameters

Suppose that allele  $A_1$  is rare and that weak selection is applied through Assumption A. Using the fitnesses (4) with the mixed strategies  $h_{ij}$  in the form (13) in the recurrence equations for  $P_{11}$ ,  $P_{12}$  near fixation of  $A_2$  ( $P_{11}$ ,  $P_{12} \cong 0$ ), one obtains a matrix of linear approximation whose value and derivative at s = 0 are given in Appendix A.2. Then, applying Result 1 and rearranging terms yield

$$L = \frac{1}{K(h)} \left[ R_{I \to I} A(h) + R_{I \to S} B(h) \right] \left[ F d_{11} + (1 - F) d_{12} \right], \tag{26}$$

where

$$R_{I \to I} = 1, \quad R_{I \to S} = \frac{1+\alpha}{2}, \quad F = \frac{\alpha}{2-\alpha}.$$
 (27)

Here, *F* is the inbreeding coefficient at equilibrium in the partial selfing model when there is no selection, that is, when s = 0 (Wright, 1921). This is in agreement with Nagylaki (1997), who confirmed that the above value of *F* can be used as an approximation in the case of weak viability selection (see also, e.g., Holsinger et al., 1984; Rocheleau and Lessard, 2000, for other studies on viability selection in partial selfing models). Moreover,  $R_{I \to I}$  is the coefficient of relatedness of an individual *I* with itself, whereas  $R_{I \to S}$  is the coefficient of relatedness of an individual *I* with a sib *S*, both calculated in the absence of selection.

Since K(h) in (26) is always positive, the increase in frequency of a rare mutant allele, which occurs when L > 0, depends upon the product of two factors in agreement with studies of kin selection models without inbreeding previously made by Uyenoyama and Feldman (1981) and Uyenoyama et al. (1981): a structural factor and a viability-analogous factor. The structural factor takes into account the changes in fitness produced by the diverse interactions between sibs, while the viability-analogous factor refers to the adaptive topography proposed by Wright (1942) for partially inbred populations under weak selection in classical viability models. In the partial selfing model, the structural factor corresponds to the rate of increase A(h) in the fitness of a randomly chosen individual I with respect to an initial increase in the probability for this individual to use strategy 1, plus the rate of increase B(h) that such a

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change in I induces in the fitness of a sib S with which it interacts, weighted by the coefficient of relatedness of I to S, since this contribution of I to the reproductive success of S can only be inherited through genes transmitted by S. On the other hand, the viability-analogous factor is given by a weighted average of the increases (or decreases) in the probability of using strategy 1 induced by the substitution of a gene of type  $A_1$ , and all genes i.b.d. to this one, for a gene of type  $A_2$  when  $A_1$  is rare in the population. These changes for inbred individuals and outbred individuals are  $d_{11}$ and  $d_{12}$ , respectively, and are weighted by the inbreeding coefficient F and its complementary 1 - F, respectively.

#### 3.2. Small differences in viabilities

This time, we shall use fitnesses (4) with  $m_{uv}$  in the form (19). Substituting these values into the recurrence equations and developing these further near fixation of  $A_2$  give the same matrix of linear approximation at s = 0 as previously but a different derivative of this matrix at s = 0 (see Appendix A.2). Applying Result 1 and rearranging terms yield

$$L = \left[ \Delta_{II}^{(1)} a(h_{22}) + \Delta_{IS}^{(1)} b(h_{11}) + \frac{\Delta_{IS}^{(3)}}{2} b(h_{12}) \right] (h_{11} - h_{22}) + \left[ \Delta_{II}^{(7)} a(h_{22}) + \Delta_{IS}^{(5)} b(h_{11}) + \left( \Delta_{IS}^{(7)} + \frac{\Delta_{IS}^{(8)}}{2} \right) b(h_{12}) \right] (h_{12} - h_{22}),$$
(28)

where

$$\Delta_{II}^{(1)} = \frac{\alpha}{(2-\alpha)}, \qquad \Delta_{II}^{(7)} = \frac{2(1-\alpha)}{(2-\alpha)}, \Delta_{IS}^{(1)} = \frac{\alpha(1+3\alpha)}{4(2-\alpha)}, \qquad \Delta_{IS}^{(3)} = \Delta_{IS}^{(5)} = \frac{\alpha(1-\alpha)}{2(2-\alpha)}, \Delta_{IS}^{(7)} = \frac{(1-\alpha)(2+2\alpha-\alpha^2)}{2(2-\alpha)^2}, \qquad \Delta_{IS}^{(8)} = \frac{2(1-\alpha)^2}{(2-\alpha)^2}.$$
(29)

These are Gillois' (1965) condensed identity coefficients in the partial selfing model (as shown in Rocheleau, 2003). In general, for two individuals X and Y, the coefficient  $\Delta_{XY}^{(1)}$  is the probability that the two genes of X be i.b.d. to the two genes of Y,  $\Delta_{XY}^{(2)}$  the probability that the two genes of X be i.b.d. and the two genes of Y be i.b.d. but the genes of X not i.b.d. to the genes of Y,  $\Delta_{XY}^{(3)}$  the probability that the two genes of X be i.b.d. to one and only one gene of Y,  $\Delta_{XY}^{(4)}$  the probability that the two genes of X be i.b.d. and the two genes of Y not i.b.d. to one another and to any gene of X,  $\Delta_{XY}^{(5)}$ the probability that one and only one gene of X be i.b.d. to the two genes of Y hot i.b.d. to one another and to any gene of X,  $\Delta_{XY}^{(f)}$ the probability that one and only one gene of X be i.b.d. to the two genes of Y,  $\Delta_{XY}^{(6)}$  the probability that the two genes of Y be i.b.d. and the two genes of X not i.b.d. to one another and to any gene of Y,  $\Delta_{XY}^{(f)}$  the probability that each gene of X be i.b.d. to one and only one gene of Y,  $\Delta_{XY}^{(8)}$  the probability that one and only one gene of X be i.b.d. to one and only one gene of Y, and  $\Delta_{XY}^{(9)}$  the probability that none of the genes of X and Y be i.b.d. to another. Introducing the coefficient  $R_{XY\to Z}^{x,y}$  defined as the expected fraction of genes in Z that are i.b.d. to x genes in X and y genes in X given that x genes in Y are i.b.d. (2 if Y is inbred on 1 is Y is outbred), the share measured are the state

genes in Y given that x genes in X are i.b.d. (2 if X is inbred or 1 is X is outbred), the above expression can be written into the form

$$L = \left[ R_{I,I \to I}^{2,2} a(h_{22}) + R_{I,S \to S}^{2,2} b(h_{11}) + R_{I,S \to S}^{2,1} b(h_{12}) \right] F(h_{11} - h_{22}) + \left[ R_{I,I \to I}^{1,1} a(h_{22}) + R_{I,S \to S}^{1,2} b(h_{11}) + R_{I,S \to S}^{1,1} b(h_{12}) \right] (1 - F)(h_{12} - h_{22}).$$
(30)

In the case of small differences in viabilities, the number of genes in the sib that are i.b.d. to one (if the individual is outbred) or two (if the individual is inbred) genes in the individual with which it interacts and its genotypic value come into play. We may wonder why only the genotypic value  $h_{22}$  of the sib comes into play in the effect of a strategy change in the individual on its own fitness while it is the only one that does not in the effect of such a change on the fitness of the sib. Actually, the first effect is obtained by keeping the genotypic value of the sib equal to the reference value  $h_{22}$ and the second by considering changes with respect to this value.

In general, the above coefficients of relatedness given that the individual is inbred or outbred are different. However, the above expression for L takes the form of the previous one for the case of small differences in strategies, that is,

$$L = [R_{I \to I}a + R_{I \to S}b][F(h_{11} - h_{22}) + (1 - F)(h_{12} - h_{22})],$$
(31)

in the case of an additive model with a and b defined in Eqs. (22) and (23), since

$$R_{I,I \to I}^{2,2} = R_{I,I \to I}^{1,1} = 1$$
(32)

and

$$R_{I,S\to S}^{2,2} + R_{I,S\to S}^{2,1} = R_{I,S\to S}^{1,2} + R_{I,S\to S}^{1,1} = R_{I\to S}$$
(33)

as shown in Lessard (1992) using a different notation. Notice that a similar form is obtained if allele A<sub>1</sub> is dominant, with *a* replaced by  $a(h_{22})$  and *b* by  $b(h_{11})$ , since then  $b(h_{12}) = b(h_{11})$ .

#### 4. Partial sib-mating: selection at an autosomal locus before mating

Again, consider a single autosomal locus with alleles  $A_1$  and  $A_2$  in an infinite diploid population undergoing discrete, non-overlapping generations. This time, every individual mates and reproduces with a sib chosen at random with probability  $\beta$  or with an individual chosen at random in the whole population with complementary probability  $1-\beta$  $(0 < \beta < 1)$ . The genotypic frequencies after selection but before mating,  $P_{11}$ ,  $P_{12}$ ,  $P_{22}$ , are defined as in the partial selfing model. Nonetheless, the genotypic frequencies are no longer sufficient to describe the complete dynamics of the model from one generation to the next.

Let  $x_1$ ,  $x_2$ ,  $x_3$ ,  $x_4$ ,  $x_5$ ,  $x_6$  designate the frequencies of the mating types  $A_1A_1 \times A_1A_1$ ,  $A_1A_1 \times A_1A_2$ ,  $A_1A_1 \times A_2A_2$ ,  $A_1A_2 \times A_1A_2$ ,  $A_1A_2 \times A_2A_2$ ,  $A_2A_2 \times A_2A_2$ , respectively, in the previous generation. Then, the genotypic frequencies among the adults of the current generation after selection according to the fitnesses in (4) can be expressed with respect to these frequencies and the frequencies of the mating types in the current generation can be described by recurrence equations given in Appendix A.3.

Notice that, with selection taking place before mating, the contribution of a mated couple in mated sibs in the next generation is proportional to the mean fitness of its female offspring, assuming that all females are fertilized. If the mated couple is of type  $A_1A_2 \times A_1A_2$ , for instance, the mean fitness of the offspring, male or female, is  $(1/4)f_{11:12\times12} + (1/2)f_{12:12\times12} + (1/4)f_{22:12\times12}$ , represented by  $\bar{f}_4$ , and the contribution in mated sibs of genotype  $A_1A_2$ , for instance, will be proportional to  $f_4$  times the square of  $(1/2)f_{12:12\times12}/\bar{f}_4$ , which gives  $(1/4)f_{12:12\times12}/\bar{f}_4$ . On the other hand, the contribution in randomly mated individuals, male or female, is proportional to the mean fitness of the offspring.

Developing the recurrence equations near fixation of allele  $A_2(x_1, x_2, ..., x_5 \cong 0)$ , the matrix of linear approximation and its derivative at s = 0 can easily be obtained under the Assumption A or B (see Appendix A.3). We get the same results as previously but with the coefficients

$$R_{I \to I} = 1, \quad R_{I \to S} = \frac{1}{2 - \beta}, \quad F = \frac{\beta}{4 - 3\beta},$$
 (34)

in the case of Assumption A, and the coefficients

$$\Delta_{II}^{(1)} = \frac{\beta}{(4-3\beta)},$$

$$\Delta_{II}^{(7)} = \frac{4(1-\beta)}{(4-3\beta)},$$

$$\Delta_{IS}^{(1)} = \frac{\beta(2+\beta)}{(2-\beta)(4-\beta)(4-3\beta)},$$

$$\Delta_{IS}^{(3)} = \Delta_{IS}^{(5)} = \frac{4\beta(1-\beta)}{(2-\beta)(4-\beta)(4-3\beta)},$$

$$\Delta_{IS}^{(7)} = \frac{8(1-\beta)(32-24\beta+2\beta^2-\beta^3)}{(2-\beta)(4-\beta)(4-3\beta)^2(8+\beta)},$$

$$\Delta_{IS}^{(8)} = \frac{64(1-\beta)^2(8-4\beta-\beta^2)}{(2-\beta)(4-\beta)(4-3\beta)^2(8+\beta)},$$
(35)

in the case of Assumption B. The coefficient F is the inbreeding coefficient at equilibrium in the partial sib-mating model without selection (see, e.g., Karlin, 1968), and similarly all the other coefficients have the same meanings as previously but in the context of inbreeding caused par partial sib-mating (as checked in Rocheleau, 2003).

#### 5. Partial sib-mating: selection at an autosomal locus after mating

In the case of partial sib-mating with selection after mating but before reproduction, we have the recurrence equations given in Appendix A.4. This time, the contribution of a mated couple in mated sibs in the next generation is proportional to the square of the mean fitness of its offspring. If the mated couple is of type  $A_1A_2 \times A_1A_2$ , for instance, its contribution in mated sibs of genotype  $A_1A_2$  will be proportional to  $\overline{f}_4^2$  times the square of  $(1/2)f_{12:12\times 12}/\overline{f}_4$ , which gives  $(1/4)f_{12:12\times 12}^2$ . On the other hand, its contribution in randomly mated individuals is proportional to the mean fitness of its offspring as in the case of selection before mating.

#### 5.1. Small differences in strategy parameters

Under Assumption A, we get

$$L = \frac{1}{K(h)} [(R_{I \to I} + R_{I \to IM})A(h) + (R_{I \to S} + R_{I \to SM})B(h)][Fd_{11} + (1 - F)d_{12}],$$
(36)

where F,  $R_{I \rightarrow I}$ ,  $R_{I \rightarrow S}$  are the same as previously and

$$R_{I \to IM} = R_{I \to SM} = \frac{\beta}{2 - \beta}.$$
(37)

All these coefficients represent coefficients of relatedness of an individual I to an individual interacting with I; subscript IM stands for its mate, S for one of its sibs and SM for the mate of one of its sibs.

The difference between the structural factors in the case of selection after mating versus those in the case of selection before mating stems from the fact that an individual does not reproduce in the former case not only when it does not survive but also when its mate does not survive and vice versa; then, selection will affect not only the reproductive success of each individual separately but also the reproductive success of its mate, which mate can bear some genetical relationship with the individual, possibly being one of its sibs. In addition, an individual will influence not only the reproductive success of the sib with which it interacts and to which it is related, but also the reproductive success of the mate of that sib, which mate can be genetically related to the individual, possibly being another of its sibs.

Besides the coefficients of relatedness to individuals alone weighting the effects of selection before mating, the effects of selection after mating are weighted by coefficients of relatedness to their mates. Actually, the coefficient of A(h) corresponds to twice the fraction of genes in an individual and its mate that are i.b.d. to genes in the individual, which can be represented by  $R_{I \rightarrow I,IM}$ , and similarly, the coefficient of B(h) is twice the fraction of genes in a sib and its mate that are i.b.d. to genes in the individual, represented by  $R_{I \rightarrow S,SM}$ . These can be viewed as coefficients of relatedness of an individual to mated couples.

#### 5.2. Small differences in viabilities

Under Assumption B, we have

$$L = \begin{bmatrix} \left(\frac{2}{2-\beta}\right)a(h_{22}) + \left(\frac{2(1+\beta+\beta^2)}{(2-\beta)(4-\beta)}\right)b(h_{11}) \\ + \left(\frac{(1-\beta)(2+\beta)^2}{2(2-\beta)(4-\beta)}\right)b(h_{12}) + \left(\frac{\beta(1-\beta)}{2(4-\beta)}\right)b(h_{22}) \end{bmatrix} F(h_{11} - h_{22}) \\ + \begin{bmatrix} \left(\frac{2}{2-\beta}\right)a(h_{22}) + \left(\frac{\beta(8+4\beta+\beta^2)}{8(2-\beta)(4-\beta)}\right)b(h_{11}) \\ + \left(\frac{(8+\beta^2-\beta^3)}{2(2-\beta)(4-\beta)}\right)b(h_{12}) + \left(\frac{\beta(4-3\beta)}{8(2-\beta)}\right)b(h_{22}) \end{bmatrix} \\ \times (1-F)(h_{12} - h_{22}).$$
(38)

This takes the form

$$L = \begin{bmatrix} \left(R_{I,I \to I}^{2,2} + R_{I,I \to IM}^{2,2}\right) a(h_{22}) + \left(R_{I,S \to S}^{2,2} + R_{I,S \to SM}^{2,2}\right) b(h_{11}) \\ + \left(R_{I,S \to S}^{2,1} + R_{I,S \to SM}^{2,1}\right) b(h_{12}) + \left(R_{I,S \to SM}^{2,0}\right) b(h_{22}) \end{bmatrix} F(h_{11} - h_{22})$$

$$+ \begin{bmatrix} \left(R_{I,I \to I}^{1,1} + R_{I,I \to IM}^{1,1}\right)a(h_{22}) + \left(R_{I,S \to S}^{1,2} + R_{I,S \to SM}^{1,2}\right)b(h_{11}) \\ + \left(R_{I,S \to S}^{1,1} + R_{I,S \to SM}^{1,1}\right)b(h_{12}) + \left(R_{I,S \to SM}^{1,0}\right)b(h_{22}) \end{bmatrix} (1 - F)(h_{12} - h_{22}),$$
(39)

where

$$\begin{split} R_{I,I \to IM}^{2,2} &= \beta \left( \Delta_{IS}^{(1)} + \frac{1}{2} \Delta_{IS}^{(3)} \right), \\ R_{I,S \to SM}^{2,2} &= \beta^2 \left( \Delta_{IS}^{(1)} + \frac{3}{16} \Delta_{IS}^{(3)} + \frac{3}{16} \Delta_{IS}^{(5)} + \frac{1}{16} \Delta_{IS}^{(7)} + \frac{1}{32} \Delta_{IS}^{(8)} \right), \\ R_{I,S \to SM}^{2,1} &= \beta^2 \left( \frac{3}{16} \Delta_{IS}^{(3)} + \frac{3}{16} \Delta_{IS}^{(5)} + \frac{1}{8} \Delta_{IS}^{(7)} + \frac{1}{16} \Delta_{IS}^{(8)} \right), \\ R_{I,S \to SM}^{2,0} &= \beta^2 \left( \frac{1}{16} \Delta_{IS}^{(7)} + \frac{1}{32} \Delta_{IS}^{(8)} \right), \\ R_{I,I \to IM}^{1,1} &= \beta \left( \Delta_{IS}^{(5)} + \Delta_{IS}^{(7)} + \frac{1}{2} \Delta_{IS}^{(8)} \right) + (1 - F), \\ R_{I,S \to SM}^{1,2} &= \beta^2 \left( \frac{3}{16} \Delta_{IS}^{(3)} + \frac{3}{16} \Delta_{IS}^{(5)} + \frac{1}{8} \Delta_{IS}^{(7)} + \frac{1}{16} \Delta_{IS}^{(8)} \right), \\ R_{I,S \to SM}^{1,1} &= \beta^2 \left( \Delta_{IS}^{(2)} + \frac{1}{4} \Delta_{IS}^{(3)} + \frac{5}{8} \Delta_{IS}^{(4)} + \frac{1}{4} \Delta_{IS}^{(5)} + \frac{5}{8} \Delta_{IS}^{(6)} \right) \\ &+ \frac{1}{4} \Delta_{IS}^{(7)} + \frac{1}{4} \Delta_{IS}^{(8)} + \frac{1}{4} \Delta_{IS}^{(9)} \right) \\ &+ \beta(1 - \beta) \left( F^2 + \frac{5}{4} F(1 - F) + \frac{1}{4}(1 - F)^2 \right), \\ R_{I,S \to SM}^{1,0} &= \beta^2 \left( \frac{1}{16} \Delta_{IS}^{(3)} + \frac{1}{8} \Delta_{IS}^{(7)} + \frac{3}{16} \Delta_{IS}^{(8)} + \frac{1}{4} \Delta_{IS}^{(9)} \right) \\ &+ \beta(1 - \beta) \left( \frac{1}{4} F(1 - F) + \frac{1}{4} (1 - F)^2 \right) \end{split}$$

using the coefficients in Eq. (35) and

$$\Delta_{IS}^{(2)} = \frac{\beta(1-\beta)(32-24\beta+2\beta^2-\beta^3)}{(2-\beta)(4-\beta)(4-3\beta)^2(8+\beta)},$$
  

$$\Delta_{IS}^{(4)} = \Delta_{IS}^{(6)} = \frac{4\beta(1-\beta)^2(8-4\beta-\beta^2)}{(2-\beta)(4-\beta)(4-3\beta)^2(8+\beta)},$$
  

$$\Delta_{IS}^{(9)} = \frac{16(1-\beta)^3}{(4-\beta)(4-3\beta)^2}.$$
(41)

(40)

The above expressions are obtained by conditioning on the event that the mate of S (or I) is a sib (probability  $\beta$ ) and its parents are sibs (probability  $\beta$ ) or not (probability  $1 - \beta$ ). To the coefficients of relatedness in Eq. (30) must be added the corresponding coefficients for mates when selection occurs after mating. This explain the presence of terms  $b(h_{22})$  with non-null coefficients in Eq. (39).

When the model is additive, Eq. (39) reduces to

$$L = [(R_{I \to I} + R_{I \to IM})a + (R_{I \to S} + R_{I \to SM})b] \times [F(h_{11} - h_{22}) + (1 - F)(h_{12} - h_{22})],$$
(42)

using Eqs. (22) and (23), which is in the same form as Eq. (36).

#### 6. Partial sib-mating: selection at a sex-linked locus after mating

Now, consider a sex-linked locus (or equivalently, a haplo-diploid population) for which females are diploid while males are haploid. The frequencies of genotypes  $A_1A_1$ ,  $A_1A_2$  and  $A_2A_2$  among females in the current generation after selection are written  $Q_{11}$ ,  $Q_{12}$ ,  $Q_{22}$ , respectively, and the corresponding frequencies for genotypes  $A_1$  and  $A_2$  among males  $P_1$  and  $P_2$ , respectively. Let  $x_1$ ,  $x_2$ ,  $x_3$ ,  $x_4$ ,  $x_5$ ,  $x_6$  be the frequencies of the mating types  $A_1A_1 \times A_1$ ,  $A_1A_2 \times A_1$ ,  $A_2A_2 \times A_1$ ,  $A_1A_1 \times A_2$ ,  $A_1A_2 \times A_2$ ,  $A_2A_2 \times A_2$ , respectively, in the prevous generation. Using the fitnesses given in Eqs. (10) and (11), the genotypic frequencies among females and among males, respectively, in the next generation after selection are given by the recurrence equations in Appendix A.5.

## 6.1. Small differences in strategy parameters

Under Assumption A, we get the result

$$L = \frac{1}{K(h)} \left\{ \frac{R_{I \to I,IM} A(h)}{+(\frac{1}{2}R_{I \to S,SM} + \frac{1}{2}R_{I \to B,BM})B(h)} \right\} [Fd_{11} + (1 - F)d_{12}] + \frac{1}{K(h)} \left\{ \frac{R_{J \to J,JM} A(h)}{+(\frac{1}{2}R_{J \to S,SM} + \frac{1}{2}R_{J \to B,BM})B(h)} \right\} d_1,$$
(43)

where

$$R_{X \to Y,YM} = \frac{\pi(Y)}{3} R_{X \to Y} + \frac{\pi(YM)}{3} R_{X \to YM}$$

$$\tag{44}$$

with  $\pi(Y)$  and  $\pi(YM)$  denoting the ploidy of Y and YM, respectively (1 if haploid, 2 if diploid) and

$$R_{I \to I} = R_{J \to J} = 1, \quad R_{I \to IM} = \frac{\beta}{2-\beta}, \quad R_{J \to JM} = \frac{\beta}{4-3\beta}, \quad F = \frac{\beta}{4-3\beta},$$

$$R_{I \to S} = \frac{3-\beta}{2(2-\beta)}, \quad R_{J \to S} = \frac{1}{4-3\beta}, \quad R_{I \to SM} = \frac{\beta}{2-\beta}, \quad R_{J \to SM} = \frac{\beta(2-\beta)}{4-3\beta},$$

$$R_{I \to B} = \frac{1}{2-\beta}, \quad R_{J \to B} = \frac{2-\beta}{4-3\beta}, \quad R_{I \to BM} = \frac{\beta(3-\beta)}{2(2-\beta)}, \quad R_{J \to BM} = \frac{\beta}{4-3\beta}.$$
(45)

Here *I* stands for a female, *J* for a male, *S* for a sister, *B* for a brother and *YM* for the mate of *Y*. The interpretation of the coefficients involved in the sex-linked case is analogous to the one in the autosomal case:  $R_{X \to Y,YM}$  represents the fraction of genes in an individual *Y* and its mate *YM* that are i.b.d. to genes in an individual *X*. Of course, at a sex-linked locus with diploid females and haploid males, this is 2/3 the fraction of genes in the female of the couple that are i.b.d. to genes in *X* plus 1/3 the corresponding fraction in the male of the couple. The fraction 1/2 which appears in the *B*(*h*) terms simply reflects the implicit hypothesis of equal numbers in females and males at each generation, so that half of the interactions are produced by females and the other half by males.

Surprisingly, Eq. (43) can be written into the simpler form

$$L = \frac{(4-\beta)}{(2-\beta)K(h)} \left[ A(h) + \left(\frac{1+\beta}{2}\right) B(h) \right] \\ \times \left[ F(d_{11}+d_1) + (1-F) \left( d_{12} + \frac{d_1}{2} \right) \right],$$
(46)

which is reminiscent of Eq. (26).

#### 6.2. Small differences in viabilities

Under Assumption B, we have

$$L = \begin{bmatrix} R_{I,I \to I,IM}^{2,2}(a(h_{22}) + a(h_{2})) + R_{I,S \to S,SM}^{2,2}b(h_{11}) + R_{I,S \to S,SM}^{2,1}b(h_{12}) \\ + R_{I,B \to B,BM}^{2,1}b(h_{1}) + R_{I,B \to B,BM}^{2,0}b(h_{2}) \end{bmatrix} \frac{F(h_{11} - h_{22})}{2} \\ + \begin{bmatrix} R_{I,I \to I,IM}^{1,1}(a(h_{22}) + a(h_{2})) + R_{I,S \to S,SM}^{1,2}b(h_{11}) + R_{I,S \to S,SM}^{1,1}b(h_{12}) \\ + R_{I,S \to S,SM}^{1,0}b(h_{22}) + R_{I,B \to B,BM}^{1,1}b(h_{1}) + R_{I,B \to B,BM}^{1,0}b(h_{2}) \end{bmatrix} \frac{(1 - F)(h_{12} - h_{22})}{2} \\ + \begin{bmatrix} R_{I,J \to J,JM}^{1,1}(a(h_{22}) + a(h_{2})) + R_{J,S \to S,SM}^{1,2}b(h_{1}) + R_{J,S \to S,SM}^{1,0}b(h_{2}) \\ + R_{J,S \to S,SM}^{1,0}b(h_{22}) + R_{J,B \to B,BM}^{1,2}b(h_{1}) + R_{J,S \to S,SM}^{1,0}b(h_{2}) \end{bmatrix} \frac{(h_{1} - h_{2})}{2},$$

$$(47)$$

where F is the same as previously and

$$\begin{aligned} R_{I,I \to I,IM}^{2,2} &= R_{I,I \to I,IM}^{1,1} = \frac{4-\beta}{3(2-\beta)}, \quad R_{I,S \to S,SM}^{2,2} = \frac{4+\beta+\beta^{2}}{6(2-\beta)}, \\ R_{I,S \to S,SM}^{2,1} &= \frac{(1-\beta)(2+\beta)}{6(2-\beta)}, \quad R_{I,B \to B,BM}^{2,1} = \frac{(1+\beta)(2+\beta)}{6(2-\beta)}, \\ R_{I,B \to B,BM}^{2,0} &= \frac{\beta(1-\beta)}{2(2-\beta)}, \quad R_{I,S \to S,SM}^{1,2} = \frac{\beta(4+\beta)}{24(2-\beta)}, \\ R_{I,B \to B,BM}^{1,1} &= \frac{12-4\beta+\beta^{2}}{12(2-\beta)}, \quad R_{I,S \to S,SM}^{1,0} = \frac{\beta(4-3\beta)}{24(2-\beta)}, \\ R_{I,B \to B,BM}^{1,1} &= \frac{2+\beta+\beta^{2}}{6(2-\beta)}, \quad R_{I,B \to B,BM}^{1,0} = \frac{\beta(5-3\beta)}{6(2-\beta)}, \\ R_{I,J \to J,JM}^{1,1} &= \frac{4-\beta}{3(4-3\beta)}, \quad R_{I,S \to S,SM}^{1,2} = \frac{\beta(4+\beta+\beta^{2})}{6(2-\beta)(4-3\beta)}, \\ R_{J,S \to S,SM}^{1,1} &= \frac{(1-\beta)(4+2\beta+\beta^{2})}{3(2-\beta)(4-3\beta)}, \quad R_{J,S \to S,SM}^{1,0} = \frac{\beta(1-\beta)}{6(2-\beta)}, \\ R_{J,B \to B,BM}^{1,1} &= \frac{4-2\beta+\beta^{2}}{3(2-\beta)(4-3\beta)}, \quad R_{J,B \to B,BM}^{1,0} = \frac{2\beta(1-\beta)}{3(2-\beta)(4-3\beta)}. \end{aligned}$$

Here,  $R_{X,Y \to Z,W}^{x,y}$  represents the fraction of genes in Z and W that are i.b.d. to x genes in X and y genes in Y given that x genes in X are i.b.d. (2 if X is diploid and inbred or 1 if X is diploid and outbred or haploid). If the model is additive, then Eq. (47) is analogous in structure to Eq. (43) and takes the simple form

$$L = \frac{(4-\beta)}{(2-\beta)} \left[ a + \left(\frac{1+\beta}{2}\right) b \right] \\ \times \left[ F(h_{11} - h_{22} + h_1 - h_2) + (1-F) \left( h_{12} - h_{22} + \frac{h_1 - h_2}{2} \right) \right],$$
(49)

where a and b are defined in Eqs. (22) and (23).

#### 7. Models for altruism

Suppose that strategy 1 corresponds to an altruistic behaviour in a standard context of a kin selection model. Then it seems natural to assume the following inequalities:

$$m_{11} < m_{21}, \quad m_{12} < m_{22}$$
 (50)

and

$$m_{11} > m_{12}, \quad m_{21} > m_{22}.$$
 (51)

These inequalities clearly imply that A(h) < 0 and B(h) > 0. Thus, following, e.g., Karlin and Matessi (1983), -A(h) can be viewed as the cost for an individual who increases its probability of performing an altruistic act (donor) and B(h) as the benefit for an individual who profits from such an increase in a sib interacting with it (recipient). Then, under Assumption A, it is possible to restate our results in this particular context. Table 1 schematically gives conditions on the proportion  $\gamma$  of inbred matings (the probability of selfing or sib-mating each generation) to have protection (L>0) of a rare mutant allele  $A_1$  in all the partial inbreeding models considered. These are classified according to ranges of values for the cost–benefit ratio

$$r = -\frac{A(h)}{B(h)} \tag{52}$$

and the signs of strategy differences  $D_{11}$  and  $D_{12}$  as defined below.

For the partial selfing model at an autosomal locus,

$$\gamma = \alpha, D_{11} = d_{11}, \quad D_{12} = d_{12},$$
  
 $\gamma_0 = 2r - 1 \text{ and } \gamma_1 = \frac{2D_{12}}{2D_{12} - D_{11}}.$ 
(53)

For the partial sib-mating model with selection at an autosomal locus before selection,

$$\gamma = \beta, \quad D_{11} = d_{11}, \quad D_{12} = d_{12},$$
  
 $\gamma_0 = \frac{2r - 1}{r} \text{ and } \gamma_1 = \frac{4D_{12}}{4D_{12} - D_{11}}.$ 
(54)

Table 1

Conditions for protection of a rare mutant allele  $A_1$  in the partial inbreeding models under Assumption A (see text for definitions of  $D_{11}$ ,  $D_{12}$ ,  $\gamma_0$  and  $\gamma_1$ )

Signs of strategy differences	Cost-benefit ratio		
	$r \leq 1/2$	1/2 <r<1< th=""><th><math>r \ge 1</math></th></r<1<>	$r \ge 1$
$\overline{D_{11} > 0, D_{12} > 0}$	All y	$\gamma > \gamma_0$	Νο γ
$D_{11} < 0, D_{12} < 0$	Νογ	$\gamma < \gamma_0$	All γ
$D_{11} < 0, D_{12} > 0$	$\gamma < \gamma_1$	$\min(\gamma_0, \gamma_1) < \gamma < \max(\gamma_0, \gamma_1)$	$\gamma > \gamma_1$
$D_{11} > 0, D_{12} < 0$	$\gamma > \gamma_1$	$\gamma < \min(\gamma_0, \gamma_1) \text{ or } \gamma > \max(\gamma_0, \gamma_1)$	$\gamma < \gamma_1$

For the partial sib-mating model with selection at an autosomal locus after mating,

$$\gamma = \beta, \quad D_{11} = d_{11}, \quad D_{12} = d_{12},$$
  
 $\gamma_0 = 2r - 1 \text{ and } \gamma_1 = \frac{4D_{12}}{4D_{12} - D_{11}}.$ 
(55)

For the partial sib-mating model with selection at a sex-linked locus after mating,

$$\gamma = \beta, \quad D_{11} = \frac{1}{2} d_{11} + \frac{1}{2} d_1,$$
  

$$D_{12} = \frac{2}{3} d_{12} + \frac{1}{3} d_1, \quad \gamma_0 = 2r - 1$$
  
and  $\gamma_1 = \frac{3D_{12}}{3D_{12} - D_{11}}.$ 
(56)

Notice that the threshold level  $\gamma_0$  for the proportion of inbred matings increases as the cost–benefit ratio r increases, taking the value 0 when  $r = \frac{1}{2}$  and 1 when r = 1, while the threshold level  $\gamma_1$  increases as the ratio  $-D_{12}/D_{11}$  increases, from 0 to 1 as this ratio goes from 0 to infinity.

The conditions in Table 1 ensure that the structural and viability-analogous factors in the expression of L are of the same sign. As  $\gamma$  increases, increasing the value of the inbreeding coefficient F and the weight of the B(h) term compared to that of the A(h) term, the sign of the structural factor passes from the sign of A(h) (negative) to the sign of B(h) (positive), while the sign of the viability-analogous factor passes from the sign of  $D_{12}$  to the sign of  $D_{11}$ , these signs being positive if allele  $A_1$  codes for more altruism in the heterozygote  $A_1A_2$  and the homozygote  $A_1A_1$ , respectively, with respect to the homozygote  $A_2A_2$ , and negative otherwise. From Table 1, we conclude that more inbreeding favours the evolution of altruism when  $D_{12}$  and  $D_{11}$  are of the same sign, which is the case if there is dominance of one of the alleles or no dominance at all, but not necessarily when  $D_{12}$  and  $D_{11}$  are of opposite signs, in which case the protection of  $A_1$  requires an intermediate value between 0 and 1 for the level of inbred matings if there is overdominance.

Analogous results can be obtained under Assumption B in the case of an additive model. Then, the cost-benefit ratio becomes r = -a/b with a and b defined in Eqs. (22) and (23) while the parameters  $D_{ij}$  are now defined using the differences  $h_{ij}-h_{22}$  and  $h_i-h_2$  instead of  $d_{ij}$  and  $d_i$  for all i and j.

Moreover, to get conditions for the protection of allele  $A_2$ , it suffices to permute alleles  $A_1$  and  $A_2$ . Of course, the protection of both alleles means a protected polymorphism.

#### 8. Discussion

We have studied family specific fitnesses based on mixed strategies following Michod (1980). Here, a mixed strategy refers to a probabilistic choice among a set of pure strategies or behaviours adopted by an individual when interacting with another individual. In this paper, we have restricted ourselves to two possible pure strategies and to pairwise interactions between individuals within the same sibship. Despite the fact that the fitness of an individual is determined both by the genotype of the individual and the genotype of one of its sibs chosen at random, the whole distribution of such pairs of genotypes is not necessary to obtain an expression for such a fitness: it suffices to condition on the mating type of the parents (see, e.g., Michod, 1982, for an introduction to kin selection theory). Detailed expressions in the diploid case as well as their analogues in the haplo-diploid case are provided in Section 2.

Two hypotheses of weak selection, bearing either on the strategy parameters or the fitness parameters, have been considered, in order to apply a formula ascertained in Lessard and Rocheleau (2003) for the change in frequency of a rare mutant allele. In that paper, inbreeding models with partial selfing in diploid populations or partial sib-mating in diploid or haplo-diploid populations, all with constant selective values, were studied and the results interpreted from a kin selection perspective, although there were no fitness interactions as such between kin. In the present paper, we have considered pairwise interactions between kin affecting fitness, either before mating or after mating, to study in details the factors and coefficients that determine the initial fate of a mutant allele as reported in Sections 3–6.

Under the hypothesis of small differences in strategy parameters, or small differences in fitness parameters but then assuming an additive model, it has been shown that the rate of change in the frequency of a mutant allele, denoted by L, can be expressed as a product of two factors: a structural factor and a viability-analogous factor (named so following studies for models with random mating by Uyenoyama and Feldman (1981), and Uyenoyama et al., 1981). This is in agreement with Uyenoyama (1984) at least in the case of the additive model with partial sib-mating. As we have seen, this particular form of L in this case is a result of special relationships between identity coefficients given in Eqs. (32) and (33). Such relationships hold also in models with female assemblies as those considered in Uyenoyama (1984). This could explained why Michod and Hamilton's (1980) covariance ratio to measure relatedness may depend in this case on gene frequency, selfing proportion and the dominance relationships among genotypes. This could also explain why a higher level of selfing may not necessarily favour in such a case the evolution of a mutant allele coding for altruism even in the absence of overdominance or underdominance.

Under Assumption A of small differences in genotypic values around some mixed strategy h, the genotypic fitnesses within families are tantamount to fitnesses in additive kin selection models with additive increments or decrements A(h)and B(h) as defined in Eq. (16). In the particular context of altruism, the coefficient -A(h) can be viewed as the cost of performing an altruistic act incurred by the donor and the coefficient B(h) as the benefit bestowed to the recipient of such an altruistic act. In this context, we have deduced explicit restrictions on the proportion of inbred matings,  $\gamma$ , for protection of a mutant allele  $A_1$  segregating with a resident allele  $A_2$ , which can be written in terms of the strategy differences  $D_{11}$  and  $D_{12}$  for the homozygote  $A_1A_1$  and the heterozygote  $A_1A_2$ , respectively, with respect to the homozygote  $A_2A_2$ , which are present in the viability-analogous factor, and the cost-benefit ratio r = -A(h)/B(h), which comes from the structural factor.

In view of Table 1, we can assess that a small cost-benefit ratio (actually,  $r \le 1/2$ ) tends to favour the evolution of altruism while a large cost-benefit ratio ( $r \ge 1$ ) tends to disfavour such an evolution, although this rule is not of general validity. For instance, in completely random mating populations ( $\gamma = 0$ ), a small cost-benefit ratio will lead to an increase in the frequency of a rare mutant allele if and only if the mutant heterozygote performs more altruism than the resident homozygote ( $D_{12} > 0$ ). However, the condition  $D_{12} > 0$  is no longer sufficient (nor necessary) to maintain a rare allele  $A_1$  when there is inbreeding. In fact, if the mutant homozygote performs less altruism than the resident homozygote ( $D_{11} < 0$ ), the rare mutant allele cannot be maintained unless the proportion of inbred matings in the population is lower than a given threshold that depends on the model considered ( $\gamma < \gamma_1$ ), and this threshold diminishes as  $D_{12}$  gets smaller compared to  $D_{11}$  in absolute value. On the other hand, when  $D_{12} < 0$ , a mutant homozygote enhancing altruism compared to the resident homozygote ( $D_{11} > 0$ ) can guarantee the protection of the mutant allele  $A_1$  as long as the proportion of inbred matings is sufficiently high ( $\gamma > \gamma_1$ ).

Here, as in classical viability models, inbreeding acts as an evolutionary force which can overcome the effects of differential change in strategy by producing more or less homozygotes, depending upon the proportion of inbred matings in the population. In order to overcome the negative effect induced by the mutant homozygote performing less altruism than the resident homozygote, inbreeding paradoxically might act as if it was favouring the mutant heterozygote exhibiting overdominance by producing just enough homozygous individuals. This is achieved by keeping the proportion of inbred matings relatively small enough. In the case of underdominance, on the contrary, the proportion of inbred matings must be kept high enough.

A more interesting case is provided by intermediate values for the cost-benefit ratio (1/2 < r < 1), since then restrictions on the proportion of inbred matings  $\gamma$  to have protection involve not only a threshold  $\gamma_1$  depending on strategy differences, but also a threshold  $\gamma_0$  depending on r, both depending on the model considered. These restrictions can lead to various different conditions under which a rare mutant allele  $A_1$  can be protected. To illustrate this, consider the case of underdominance where the mutant homozygote has a slight advantage over the resident homozygote ( $D_{11}$  positive, but near 0, and  $D_{12} < 0$ ). Then, with a cost-benefit ratio barely higher than  $\frac{1}{2}$ , we have min( $\gamma_0$ ,  $\gamma_1$ ) =  $\gamma_1$  close to 0 and max( $\gamma_0$ ,  $\gamma_1$ ) =  $\gamma_0$  close to 1, and the mutant allele is protected for either very small values of  $\gamma$  or very large values of  $\gamma$ . In the case of overdominance, it is exactly the contrary.

As expected, the coefficients of A(h) and B(h) in the structural factors for the partial inbreeding models considered with selection before mating are coefficients of relatedness between individuals as defined in Michod and Hamilton (1980). Under weak selection, the coefficient of relatedness of an individual X to an individual Y reduces to a pedigree index given by the expected fraction of genes in Y i.b.d. to genes in X. With selection after mating, the unit of selection becomes the mated couple and the coefficients that come into play measure relatedness between individuals and mated couples, such a coefficient for an individual X to a couple made of an individual Y and its mate YM being defined as the expected fraction of genes in Y and YM i.b.d. to genes in X. This corresponds to a weighted combination of the coefficients of relatedness of X to Y and X to YM, the weights being the relative fractions of genes carried by Y and YM, respectively.

In a previous paper (Lessard and Rocheleau, 2003), we studied partially inbred populations evolving under weak selection but without interaction between kin. In that paper, we proposed an approximate adaptive topography to predict the change in frequency of a mutant allele, which was quantitatively different from the one proposed by Wright (1942) in the case of partial sib-mating, and even qualitatively different in the case of selection at a sex-linked locus. Pollak (1995) had already noticed such a discrepancy with Wright's formula in the partial sib-mating model at an autosomal locus and provided an explanation based on the positive correlation between the frequencies of the mutant allele in two mates under this type of mating. We confirmed this finding and showed that the disagreement with Wright's formula was even more important in the partial sib-mating model at a sex-linked locus (or equivalently, haplo-diploid model). Because of the asymmetry induced by a sex-linked model, two regression coefficients for the frequency of the mutant allele in one mate on the corresponding frequency in the other mate had to be used instead of a correlation coefficient and the effect on the rate of change in the frequency of the mutant allele could not be reduced to a positive multiplicative factor. But actually, the models of partial sib-mating that were considered both in Pollak (1995) and Lessard and Rocheleau (2003) are in the class of models with selection occurring after mating as pointed out by Caballero (1996). Therefore they do not fall in the range of application of Wright's formula. Although interactions between kin are not apparent, they do occur in mated couples whose reproduction depends on the viabilities of both mates. We are in a context of kin selection (Hamilton, 1964) and the results can be interpreted in terms of transfers of fitness changes from actors to beneficiaries weighted by coefficients of relatedness. Of course, this is also the case when the viabilities themselves depend on sib interactions as in the present paper.

Under Assumption B of small differences in viabilities according to the pure strategies chosen by interacting sibs, the rate of change L in the frequency of a mutant allele can be expressed as a sum of products of structural and viabilityanalogous factors according to the number of genes i.b.d. in a given individual at the locus considered, and the number of genes itself at a sex-linked locus, 1 or 2. Such a sum does not generally reduce to a single product unless the viabilities follow the additive model. In each product, the viability-analogous factor depends only on strategy changes following the substitution of one mutant gene, and all genes i.b.d. to this one, for a gene of the resident type, but it is weighted by the inbreeding coefficient F, if the individual is diploid and inbred, or its complementary 1-F, if the individual is diploid and outbred, compared to 1 if the individual is haploid. On the other hand, the structural factor is a weighted sum of rates of increase in fitness in the individual and in the sib with which it interacts with respect to an initial positive change in the strategy of the individual. These rates depend on the genotype of the sib and their weights can be viewed as coefficients of relatedness conditional on the number of genes i.b.d. in the individual that extend coefficients introduced in Lessard (1992). In the case of selection before mating, these are expected fractions of genes in the sib (or the individual) that are i.b.d. to given numbers of genes in the individual and the sib (or the individual), while in the case of selection after mating it is the expected fractions of genes in the sib (or the individual) and its mate. These definitions may seem tautological, but we have to remind ourselves that the genes in the sib are not necessarily i.b.d.

## Appendix A

#### A.1. Notation

Under Assumption A:

$$a_{22} = a_2 = \frac{A(h)}{K(h)}, \quad b_{ij} = b_i = \frac{B(h)}{K(h)}, \quad \delta_{ij} = d_{ij}, \quad \delta_i = d_i.$$

Under Assumption B:

$$a_{22} = a(h_{22}), \quad b_{ij} = b(h_{ij}), \quad \delta_{ij} = h_{ij} - h_{22},$$

$$a_2 = a(h_2), \quad b_i = b(h_i), \quad \delta_i = h_i - h_2.$$

# A.2. Partial selfing: selection at an autosomal locus before mating

$$\begin{split} TP'_{11} &= \alpha \big[ f_{11:11\times 11} P_{11} + \frac{1}{4} f_{11:12\times 12} P_{12} \big] \\ &\quad + (1-\alpha) \big[ f_{11:11\times 11} P_{11}^2 + f_{11:11\times 12} P_{11} P_{12} + \frac{1}{4} f_{11:12\times 12} P_{12}^2 \big], \\ TP'_{12} &= \alpha \big[ \frac{1}{2} f_{12:12\times 12} P_{12} \big] \\ &\quad + (1-\alpha) \bigg[ \frac{f_{12:11\times 12} P_{11} P_{12} + \frac{1}{2} f_{12:12\times 12} P_{12}^2 \big] \\ &\quad + 2 f_{12:11\times 22} P_{11} P_{22} + f_{12:12\times 22} P_{12} P_{22} \big], \\ TP'_{22} &= \alpha \big[ f_{22:22\times 22} P_{22} + \frac{1}{4} f_{22:12\times 12} P_{12} \big] \\ &\quad + (1-\alpha) \big[ f_{22:22\times 22} P_{22}^2 + f_{22:12\times 22} P_{12} P_{22} + \frac{1}{4} f_{22:12\times 12} P_{12}^2 \big], \end{split}$$

$$\mathbf{M}(0) = \begin{bmatrix} \alpha & \alpha/4 \\ 2(1-\alpha) & 1-\alpha/2 \end{bmatrix},$$
  

$$\xi(0)^{T} = (1, \frac{1}{2}),$$
  

$$\eta(0)^{T} = (\alpha, 4(1-\alpha)),$$
  

$$\mathbf{\dot{M}}(0) = \begin{bmatrix} \alpha \delta_{11}(a_{22} + b_{11}) & \frac{\alpha}{16}(4\delta_{11}a_{22} + (\delta_{11} + 2\delta_{12})b_{11}) \\ 2(1-\alpha)\delta_{12}(a_{22} + b_{12}) & \frac{\alpha}{8}(4\delta_{12}a_{22} + (\delta_{11} + 2\delta_{12})b_{12}) + \frac{(1-\alpha)}{2}\delta_{12}(2a_{22} + b_{12}) \end{bmatrix}.$$

# A.3. Partial sib-mating: selection at an autosomal locus before mating

$$\begin{split} x_1' &= (1-\beta)P_{11}^2 + \frac{\beta}{T_{\rm R}} \left[ f_{11:11\times11}^2 \frac{x_1}{\bar{f}_1} + \frac{1}{4} f_{11:11\times12}^2 \frac{x_2}{\bar{f}_2} + \frac{1}{16} f_{11:12\times12}^2 \frac{x_4}{\bar{f}_4} \right], \\ x_2' &= 2(1-\beta)P_{11}P_{12} + \frac{\beta}{T_{\rm R}} \left[ \frac{1}{2} f_{11:11\times12} f_{12:11\times12} \frac{x_2}{\bar{f}_2} + \frac{1}{4} f_{11:12\times12} f_{12:12\times12} \frac{x_4}{\bar{f}_4} \right], \\ x_3' &= 2(1-\beta)P_{11}P_{22} + \frac{\beta}{T_{\rm R}} \left[ \frac{1}{8} f_{11:12\times12} f_{22:12\times12} \frac{x_4}{\bar{f}_4} \right], \\ x_4' &= (1-\beta)P_{12}^2 + \frac{\beta}{T_{\rm R}} \left[ \frac{1}{4} f_{12:11\times12}^2 \frac{x_2}{\bar{f}_2} + f_{12:11\times22}^2 \frac{x_3}{\bar{f}_3} + \frac{1}{4} f_{12:12\times12}^2 \frac{x_4}{\bar{f}_4} + \frac{1}{4} f_{12:12\times22}^2 \frac{x_5}{\bar{f}_5} \right], \\ x_5' &= 2(1-\beta)P_{12}P_{22} + \frac{\beta}{T_{\rm R}} \left[ \frac{1}{4} f_{12:12\times12} f_{22:12\times12} \frac{x_4}{\bar{f}_4} + \frac{1}{4} f_{12:12\times22}^2 f_{22:12\times22} \frac{x_5}{\bar{f}_5} \right], \\ x_6' &= (1-\beta)P_{22}^2 + \frac{\beta}{T_{\rm R}} \left[ \frac{1}{16} f_{22:12\times12}^2 \frac{x_4}{\bar{f}_4} + \frac{1}{4} f_{22:12\times22}^2 \frac{x_5}{\bar{f}_5} + f_{22:22\times22}^2 \frac{x_6}{\bar{f}_6} \right], \end{split}$$

$$\begin{split} T_{\mathrm{R}} P_{11} &= f_{11:11\times11} x_1 + \frac{1}{2} f_{11:11\times12} x_2 + \frac{1}{4} f_{11:12\times12} x_4, \\ T_{\mathrm{R}} P_{12} &= \frac{1}{2} f_{12:11\times12} x_2 + f_{12:11\times22} x_3 + \frac{1}{2} f_{12:12\times12} x_4 \\ &\quad + \frac{1}{2} f_{12:12\times22} x_5, \\ T_{\mathrm{R}} P_{22} &= \frac{1}{4} f_{22:12\times12} x_4 + \frac{1}{2} f_{22:12\times22} x_5 + f_{22:22\times22} x_6, \\ \bar{f}_1 &= f_{11:11\times11}, \bar{f}_2 &= \frac{1}{2} f_{11:11\times12} + \frac{1}{2} f_{12:11\times12}, \bar{f}_3 = f_{12:11\times22}, \\ \bar{f}_4 &= \frac{1}{4} f_{11:12\times12} + \frac{1}{2} f_{12:12\times12} + \frac{1}{4} f_{22:12\times12}, \bar{f}_5 &= \frac{1}{2} f_{12:12\times22} + \frac{1}{2} f_{22:12\times22}, \\ \bar{f}_6 &= f_{22:22\times22}, \end{split}$$

$$\mathbf{M}(0) = \begin{bmatrix} \beta & \beta/4 & 0 & \beta/16 & 0\\ 0 & \beta/2 & 0 & \beta/4 & 0\\ 2(1-\beta) & (1-\beta) & 0 & (4-3\beta)/8 & 0\\ 0 & \beta/4 & \beta & \beta/4 & \beta/4\\ 0 & (1-\beta) & 2(1-\beta) & (4-3\beta)/4 & (2-\beta)/2 \end{bmatrix},$$
  
$$\xi(0)^{T} = (1, \frac{3}{4}, \frac{1}{2}, \frac{1}{2}, \frac{1}{4}),$$

$$\eta(0)^{T} = \left(\frac{\beta(2+\beta)}{16(2-\beta)(1-\beta)}, \frac{\beta}{2(2-\beta)}, \frac{1}{2}, 1, \frac{5\beta^{2}-20\beta+16}{2\beta(2-\beta)}\right),$$

 $\mathbf{M}_{11}(0) = \beta \delta_{11}(a_{22} + b_{11}),$  $\mathbf{\dot{M}}_{12}(0) = \frac{\beta}{16} [2(3\delta_{11} - \delta_{12})a_{22} + (\delta_{11} + \delta_{12})(3b_{11} - b_{12})],$  $\mathbf{\dot{M}_{14}(0)} = \frac{\beta}{256} [4(7\delta_{11} - 2\delta_{12})a_{22} + (\delta_{11} + 2\delta_{12})(7b_{11} - 2b_{12} - b_{22})],$  $\mathbf{\dot{M}}_{22}(0) = \frac{\beta}{2}(\delta_{11} + \delta_{12})(2a_{22} + b_{11} + b_{12}),$  $\mathbf{\dot{M}_{24}(0)} = \frac{\beta}{64} [4(3\delta_{11} + 2\delta_{12})a_{22} + (\delta_{11} + 2\delta_{12})(3b_{11} + 2b_{12} - b_{22})],$  $\mathbf{\dot{M}}_{31}(0) = 2(1-\beta)\delta_{11}(a_{22}+b_{11}),$  $\mathbf{\dot{M}}_{32}(0) = \frac{(1-\beta)}{2} [2\delta_{11}a_{22} + (\delta_{11} + \delta_{12})b_{11}],$  $\mathbf{M}_{34}^{\bullet}(0) = \frac{(1-\beta)}{2} [4\delta_{11}a_{22} + (\delta_{11} + 2\delta_{12})b_{11}]$ +  $\frac{\beta}{128}[4(3\delta_{11}-2\delta_{12})a_{22}+(\delta_{11}+2\delta_{12})(3b_{11}-2b_{12}+3b_{22})],$  $\mathbf{\dot{M}}_{42}(0) = \frac{\beta}{16} [2(3\delta_{12} - \delta_{11})a_{22} + (\delta_{11} + \delta_{12})(3b_{12} - b_{11})],$  $\mathbf{\dot{M}}_{43}(0) = \beta \delta_{12}(a_{22} + b_{12}),$  $\mathbf{\dot{M}}_{44}(0) = \frac{\beta}{64} [4(6\delta_{12} - \delta_{11})a_{22} + (\delta_{11} + 2\delta_{12})(6b_{12} - b_{11} - b_{22})],$  $\mathbf{\dot{M}}_{45}(0) = \frac{\beta}{16} \,\delta_{12}(6a_{22} + 3b_{12} - b_{22}),$  $\mathbf{M}_{52}^{\bullet}(0) = \frac{(1-\beta)}{2} [2\delta_{12}a_{22} + (\delta_{11} + \delta_{12})b_{12}],$  $\mathbf{\dot{M}}_{53}(0) = 2(1-\beta)\delta_{12}(a_{22}+b_{12}),$  $\mathbf{\dot{M}_{54}(0)} = \frac{(1-\beta)}{4} [4\delta_{12}a_{22} + (\delta_{11} + 2\delta_{12})b_{12}]$  $+\frac{\beta}{64}[4(2\delta_{12}-\delta_{11})a_{22}+(\delta_{11}+2\delta_{12})(2b_{12}+3b_{22}-b_{11})],$ 

$$\mathbf{M}_{55}(0) = \frac{(1-\beta)}{2} \,\delta_{12}(2a_{22}+b_{12}) + \frac{\beta}{8} \,\delta_{12}(2a_{22}+b_{12}+b_{22}).$$

## A.4. Partial sib-mating: selection at an autosomal locus after mating

$$\begin{split} Tx'_1 &= (1-\beta)P_{11}^2 T_{\rm R}^2 + \beta \big[ f_{11:11\times 11}^2 x_1 + \frac{1}{4} f_{11:11\times 12}^2 x_2 + \frac{1}{16} f_{11:12\times 12}^2 x_4 \big], \\ Tx'_2 &= 2(1-\beta)P_{11}P_{12}T_{\rm R}^2 + \beta \big[ \frac{1}{2} f_{11:11\times 12} f_{12:11\times 12} x_2 + \frac{1}{4} f_{11:12\times 12} f_{12:12\times 12} x_4 \big], \\ Tx'_3 &= 2(1-\beta)P_{11}P_{22}T_{\rm R}^2 + \beta \big[ \frac{1}{8} f_{11:12\times 12} f_{22:12\times 12} x_4 \big], \\ Tx'_4 &= (1-\beta)P_{12}^2 T_{\rm R}^2 + \beta \big[ \frac{1}{4} f_{12:11\times 12}^2 x_2 + f_{12:11\times 22}^2 x_3 + \frac{1}{4} f_{12:12\times 12}^2 x_4 + \frac{1}{4} f_{12:12\times 22}^2 x_5 \big], \\ Tx'_5 &= 2(1-\beta)P_{12}P_{22}T_{\rm R}^2 + \beta \big[ \frac{1}{4} f_{12:12\times 12} f_{22:12\times 12} x_4 + \frac{1}{2} f_{12:12\times 22} f_{22:12\times 22} x_5 \big], \\ Tx'_6 &= (1-\beta)P_{22}^2 T_{\rm R}^2 + \beta \big[ \frac{1}{16} f_{22:12\times 12}^2 x_4 + \frac{1}{4} f_{22:12\times 22}^2 x_5 + f_{22:22\times 22}^2 x_6 \big]. \end{split}$$

Same M(0),  $\xi(0)$  and  $\eta(0)$  as previously.

$$\begin{split} \dot{\mathbf{M}_{11}}(0) &= 2\beta\delta_{11}(a_{22} + b_{11}), \\ \dot{\mathbf{M}_{12}}(0) &= \frac{\beta}{4}[2\delta_{11}a_{22} + (\delta_{11} + \delta_{12})b_{11}], \\ \dot{\mathbf{M}_{14}}(0) &= \frac{\beta}{32}[4\delta_{11}a_{22} + (\delta_{11} + 2\delta_{12})b_{11}], \\ \dot{\mathbf{M}_{22}}(0) &= \frac{\beta}{4}[(\delta_{11} + \delta_{12})(2a_{22} + b_{11} + b_{12})], \\ \dot{\mathbf{M}_{24}}(0) &= \frac{\beta}{16}[4(\delta_{11} + \delta_{12})a_{22} + (\delta_{11} + 2\delta_{12})(b_{11} + b_{12})], \\ \dot{\mathbf{M}_{31}}(0) &= 2(1 - \beta)\delta_{11}(a_{22} + b_{11}), \\ \dot{\mathbf{M}_{32}}(0) &= \frac{(1 - \beta)}{2}[2\delta_{11}a_{22} + (\delta_{11} + 2\delta_{12})b_{11}], \\ \dot{\mathbf{M}_{34}}(0) &= \frac{(1 - \beta)}{8}[4\delta_{11}a_{22} + (\delta_{11} + 2\delta_{12})b_{11}] + \frac{\beta}{32}[4\delta_{11}a_{22} + (\delta_{11} + 2\delta_{12})(b_{11} + b_{22})], \\ \dot{\mathbf{M}_{42}}(0) &= \frac{\beta}{4}[2\delta_{12}a_{22} + (\delta_{11} + \delta_{12})b_{12}], \\ \dot{\mathbf{M}_{43}}(0) &= 2\beta\delta_{12}(a_{22} + b_{12}), \\ \dot{\mathbf{M}_{44}}(0) &= \frac{\beta}{8}[4\delta_{12}a_{22} + (\delta_{11} + 2\delta_{12})b_{12}], \\ \dot{\mathbf{M}_{45}}(0) &= \frac{\beta}{4}[2\delta_{12}a_{22} + \delta_{12}b_{12}], \\ \dot{\mathbf{M}_{53}}(0) &= 2(1 - \beta)\delta_{12}(a_{22} + b_{12}), \\ \dot{\mathbf{M}_{53}}(0) &= 2(1 - \beta)\delta_{12}(a_{22} + b_{12}) \delta_{12}] + \frac{\beta}{16}[4\delta_{12}a_{22} + (\delta_{11} + 2\delta_{12})(b_{12} + b_{22})], \\ \dot{\mathbf{M}_{53}}(0) &= 2(1 - \beta)\delta_{12}(a_{22} + b_{12}) \delta_{12} + \frac{\beta}{16}[4\delta_{12}a_{22} + (\delta_{11} + 2\delta_{12})(b_{12}$$

$$\mathbf{M}_{55}(0) = \frac{(1-\beta)}{2} \,\delta_{12}(2a_{22}+b_{12}) + \frac{\beta}{4} \,\delta_{12}(2a_{22}+b_{12}+b_{22}).$$

# A.5. Partial sib-mating: selection at a sex-linked locus after mating

$$\begin{split} & Tx_1' = (1 - \beta)Q_{11}P_1T_FT_M + \beta \Big[ f_{1:1:1:1:1}T_{1:1:1:1} + \frac{1}{4}f_{1:1:2:1}f_{1:1:2:1}T_{2:1}^{1} + \frac{1}{4}f_{1:2:2:2}f_{1:1:2:2}T_{2:1}^{1} + \frac{1}{4}f_{1:1:2:2:1}T_{2:1}^{1} + \frac{1}{4}f_{1:1:2:2:1}T_{2:1}^{1} + \frac{1}{4}f_{1:2:2:2}f_{1:1:2:2}T_{2:1}^{1} + \frac{1}{4}f_{1:2:1:2:2}f_{1:1:2:2}T_{2:1}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{1:1:2:2}T_{2:1:2:1}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{1:1:2:2}T_{2:1:2:1}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{1:1:2:2}T_{2:1:2:1}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{2:1:2:2}T_{2:1:2:1}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{2:1:2:2}T_{2:1:2:1}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{2:1:2:2}T_{2:1:2:1}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{2:1:2:2}T_{2:1:2:2}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{2:1:2:2}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{2:1:2:2}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{2:1:2:2}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{2:1:2:2}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{2:1:2:2}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{2:1:2:2}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{2:2:2}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{2:1:2:2}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{2:1:2:2}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{2:1:2:2}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{2:2:2}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{2:1:2:2}^{1} + \frac{1}{4}f_{1:2:1:2:2}T_{2:2:2}^{1} + \frac{1}{4}f_{1:1:2:2}^{1} + \frac{1}$$

$$\begin{split} \dot{\mathbf{M}}_{34}(0) &= \frac{(1-\beta)}{2} [\delta_1(a_{22}+a_2) + (\delta_{12}+\delta_1)b_1], \\ \dot{\mathbf{M}}_{35}(0) &= \frac{(1-\beta)}{8} [2\delta_1(a_{22}+a_2) + (\delta_{12}+\delta_1)b_1] + \frac{\beta}{16} [2\delta_1(a_{22}+a_2) + (\delta_{12}+\delta_1)(b_{22}+b_1)], \\ \dot{\mathbf{M}}_{41}(0) &= \frac{(1-\beta)}{2} [\delta_{11}(a_{22}+a_2) + (\delta_{11}+\delta_1)b_1], \\ \dot{\mathbf{M}}_{42}(0) &= \frac{(1-\beta)}{8} [2\delta_{11}(a_{22}+a_2) + (\delta_{11}+\delta_{12}+\delta_1)b_{11}] + \frac{\beta}{16} [2\delta_{11}(a_{22}+a_2) + (\delta_{11}+\delta_{12}+\delta_1)(b_{11}+b_2)] \\ \dot{\mathbf{M}}_{53}(0) &= \frac{(1-\beta)}{2} [\delta_{12}(a_{22}+a_2+b_{12})] + \frac{\beta}{2} [\delta_{12}(a_{22}+a_2+b_{12}+b_2)], \\ \dot{\mathbf{M}}_{54}(0) &= \frac{(1-\beta)}{2} [\delta_{12}(a_{22}+a_2) + (\delta_{12}+\delta_1)b_{12}], \\ \dot{\mathbf{M}}_{55}(0) &= \frac{(1-\beta)}{8} [2\delta_{12}(a_{22}+a_2) + (\delta_{12}+\delta_1)b_{12}] + \frac{\beta}{16} [2\delta_{12}(a_{22}+a_2) + (\delta_{12}+\delta_1)(b_{12}+b_2)]. \end{split}$$

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