Age-Dependent Sexually Asymmetric Selection: 
The Use of Intrinsic Values

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ABSTRACT
To study the evolutionary role played by differential male and female fertility (sexual asymmetry) both between individuals and over the life span within single individuals, the terms "intrinsic male fertility" and "intrinsic female fertility" are introduced. With the help of these terms, the concept of sexual asymmetry can be made precise and its effect on the establishment and maintenance of genetic polymorphisms can be analyzed. The main conclusions are: (1) any mutant causing a modification of the male fertility parameters which result in an increased intrinsic male fertility becomes established; (2) a corollary of this is that age-specific sexual asymmetry, as results from alternating degrees of female and male flowering in successive reproduction cycles, for example, has only secondary effects on the initial growth rate; (3) under the biologically reasonable premise that modifications of life histories result from reallocation of fixed net reproduction resources (defined as constant total female and male net reproduction output), a shift of net reproduction (whether female, male, or both in arbitrary proportions) to earlier ages is evolutionarily successful in growing but not in declining populations; shifts of net reproduction to later ages have opposite consequences.

Sexual asymmetry usually refers to the situation where the male-to-female ratio of a given trait varies between individuals or types of individuals. With respect to selection parameters, for example, this concept of sexual asymmetry applies to the average number of male and female gametes of genotypes in a bisexual (hermaphroditic or monoecious) population. In contrast, in dioecious populations sexual asymmetry can be meaningfully defined only for non-sex-specific traits such as those coded for by autosomal gene loci, since this allows consideration of both sex functions in the same "type" of individual.

Variation in male-to-female ratios of viability between genotypes is the most frequently considered form of sexual asymmetry in dioecious animal populations. Apparently it is this form to which the earliest analytical population genetic studies of sexual asymmetry were devoted (Owen 1953; Shaw and Mohler 1955; Mérat 1969). The first to consider asymmetric variability in fertility were probably Bodmer (1965) and Purser (1966). In its most extreme form sexual asymmetry arises in mixed sexual systems (such as gyno-, andro-, and trioecy), where bisexual and unisexual individuals occur simultaneously. Male-to-female fertility ratios may then vary from zero to infinity. While these quite conspicuous types of asymmetry have been well known at least since Darwin's 1877 book, the more subtle differences between male and female flowering in bisexual plant populations have attracted experimental attention only more recently [see the paper of Devlin and Stephenson (1987) and the literature cited therein and the review of Ross (1990)].

Among iteroparous organisms a second type of sexual asymmetry can be observed, which is characterized by differential investment into male and female functions at different ages of an individual or type of individual. Protogyny and protandry together with the extreme form of consecutive or sequential bisexuality (hermaphroditism) are well known examples of this kind of asymmetry. This age-specific asymmetry is within individuals or types of individuals and is analogously characterized by non-constancy of the male-to-female ratio over age. These ratios may again vary between individuals or types of individuals. However, variable sex functions among age classes make it difficult to unambiguously characterize sexual asymmetry between individuals or types, since this would require a single male-to-female ratio to be specified across all age classes. It will be shown in the present paper that for studies of the evolution of life histories such ratios can be defined meaningfully on the basis of a generalized concept of intrinsic value.

The significance of the intrinsic multiplication rate (intrinsic growth rate) in the analysis of dynamical models of selection in overlapping generations has been recognized almost from the beginning of such studies [cf. Norton (1928) and Charlesworth (1980) for a summary]. Yet, apparently no attempt has been made so far to generalize the concept underlying the intrinsic multiplication rate to include in the analyses aspects other than those characteristic of symmetry between the sexes and random fusion. In

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that, due to the excessive pollen production, pollen compete for eggs but not vice versa. On the other hand, particularly in studies of sexual asymmetry, the assumption of complete fertilization may become critical as individuals producing comparatively few pollen but abundant ovules become frequent. An extreme example is provided by gynodioecious populations, where, however, irrespective of the mode of inheritance the fraction of male sterile (female) plants is quite limited due to insufficient pollination of their ovules by the hermaphrodites. If, however, because of special modes of inheritance involving segregation distortion, for example, a relative female excess should persist, population extinction could be the consequence. Yet, since establishment of new life histories rather than stability of polymorphisms or replacement is the main topic of the present treatise, the assumption of equal chances for all female gametes to be fertilized at the locus considered need not be further restricted.

The basic life history parameters to be used in the analysis are specified in Table 2, where the absence of effective temporal and spatial environmental variation allows us to consider these parameters as constant.

Recall that the intrinsic rate of increase implied by a given life history simply equals the growth rate of a population at age class equilibrium for this life history. This suggests generalization of the definition of intrinsic values to parameters other than the growth rate, provided these parameters can be analogously expressed as characteristics of populations in age class equilibrium. Hence the

**Definition:** The intrinsic value of a population parameter (including selection and mating system parameters) is defined as the value this parameter assumes at the age class equilibrium determined by the underlying life history.

In the discrete time version applied here, the age class equilibrium can be conceived to project all effects of iteroparity on population growth into a single reproduction period. Thus, the multiplication rate at age class equilibrium corresponds directly to this rate in populations reproducing in separated generations, where one generation coincides with one reproduction period. By analogy, female and male fertilities, which are defined as averages and vary with age in iteroparous organisms, can each be specified as a

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**TABLE 1**

<table>
<thead>
<tr>
<th>Model assumptions</th>
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<tbody>
<tr>
<td>(i) In each reproduction cycle the female and male gametes produced in the population fuse at random with respect to the gene locus considered;</td>
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<tr>
<td>(ii) Population size is effectively infinite;</td>
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<tr>
<td>(iii) For the gene locus considered, all female gametes have the same chance to be fertilized; whether or not this chance is high enough to guarantee fertilization of all female gametes is irrelevant for many purposes</td>
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**TABLE 2**

<table>
<thead>
<tr>
<th>Survival and fertility parameters</th>
</tr>
</thead>
<tbody>
<tr>
<td>$I_k$ := probability of survival of genotype $AA$, from birth to age $k$ ($k = 1, 2, \ldots$, and $I_1(1) = 1$)</td>
</tr>
<tr>
<td>$f_k$ := average number of female gametes of genotype $AA$, at age $k$</td>
</tr>
<tr>
<td>$m_k$ := average number of male gametes of genotype $AA$, at age $k$</td>
</tr>
<tr>
<td>$F_k$ := $I_k f_k$ or “net female fertility” at age $k$</td>
</tr>
<tr>
<td>$M_k$ := $I_k m_k$ or “net male fertility” at age $k$</td>
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SPECIFICATION OF THE MODEL AND THE INTRINSIC VALUES

Consider an iteroparous plant population for which the model assumptions listed in Table 1 hold with respect to a diploid gene locus with two alleles, $A_1, A_2$ and genotypes $A_i A_j$ ($i, j = 1, 2$). In many plant populations assumption (iii) is closely realized even to the degree that all ovules are fertilized, which suggests that, due to the excessive pollen production, pollen compete for eggs but not vice versa. On the other
single value by taking the average across all ages at age class equilibrium. This principle guides the definitions specified in Table 3.

THE CONDITION FOR ESTABLISHMENT

The establishment or protectedness of a newly arising gene is decided by its dynamics at very low frequencies. Since, due to the low frequency of the new gene, neither the population density nor the frequencies of resident genotypes are noticeably affected, the density and frequency feedback on these dynamics is negligible. Therefore, the results of the analysis of the infinitesimal dynamics apply irrespective of such interactions among frequent types. The most basic characterization of these dynamics is in terms of initial increase or decrease and is measured by the parameter

\[ \theta_i := \text{initial multiplication rate of the relative frequency (initial growth rate, for shortness) of the allele } A_i. \]

According to this definition, \( A_i \) becomes established if \( \theta_i > 1 \), and it does not become established if \( \theta_i < 1 \). The case \( \theta_i = 1 \) requires additional characterization of the initial dynamics. However, since this case implies a considerable effect of random drift events on the dynamics, it will not be further pursued here.

For the present model with two alleles it follows from the general analysis in GREGORIUS (1987) that the initial growth rate of \( A_i \) is given by

\[ \theta_i = \frac{\rho_{ij}}{\rho_{jj}} \]

where \( i, j = 1, 2 \), \( i \neq j \) and \( \rho_{ij} \) is the positive solution for \( \rho \) in

\[ \sum_i l_i(k) \cdot \frac{1}{2} \left[ f_{12}(k) + m_{12}(k) \cdot \frac{\varphi}{\mu_j} \right] \cdot \rho^4 = 1. \]  

As was mentioned above, this growth rate results from consideration of the ultimate dynamics of the allele \( A_i \) at very low frequencies. It is therefore not based on any assumptions concerning the stage of the age class distribution among the carriers of the rare allele (see GREGORIUS 1987). Even though intrinsic values are used for the rare heterozygote, this should not be misinterpreted to imply that the analysis rests on the (biologically meaningless) assumption of age class equilibrium for this rare genotype.

In the Equation 2 the term \( \frac{1}{2} (m_{12}(k) \cdot \varphi/\mu_j) \) is half the average number of successful (female plus male) gametes produced by the rare heterozygote genotype \( A_1A_2 \) at age \( k \) in a population of \( A_1A_1 \) genotypes at age class equilibrium. The effect of the resident genotype \( A_1A_1 \) on this average number is summarized in this genotype's female-to-male ratio of intrinsic fertilities, and the number of successful gametes forms the specific fertility parameter in a life table with survival parameter \( l_{12}(k) \). (Note that this result differs fundamentally from that in CHARNOV's (1982) book on p. 134ff.)

Consequently, \( \rho_{12}^{(i)} \) again is an intrinsic multiplication rate, which now refers to a population with a life table specified by these survival and fertility parameters. To distinguish it from \( \rho_{12} \), which is independent of any effects of other genotypes, \( \rho_{12}^{(i)} \) will be called the incipient intrinsic multiplication rate of the heterozygote. One thus arrives at the intuitively appealing statement that the initial growth rate of an allele equals the quotient formed of the incipient intrinsic multiplication rate of the heterozygote and the intrinsic multiplication rate of the resident homozygote. Based on this terminology, the classical finding for random mating populations is confirmed, in that a newly arising allele may be established or not if its heterozygote is superior or inferior, respectively, to the resident homozygote.

EFFECTS OF SEXUAL ASYMMETRY

There are various indices for the measurement of sexual asymmetry [see ROSS (1990) for a review] all of which, however, share the idea that there ought to be a male-to-female or female-to-male reference setting the standard for comparison of other sex proportions. For example, if the gametic output of two her-
maphroditic plants is to be evaluated with respect to the gametic sex proportions, both individuals would be considered equivalent if their numbers of male gametes per female gamete were identical. If the numbers differ, sexual asymmetry exists by definition. However, this asymmetry refers to a relative rather than to an absolute characteristic in the sense that the degree of sexual asymmetry of a given individual depends on which other individuals it is compared with. Hence, any of the two numbers can be chosen as the standard defining sexual symmetry. Other symmetry standards, such as the ratio of the sum of male and the sum of female gametes each taken over both individuals, can be chosen equally well.

The essence of the measurement of sexual asymmetry thus consists in setting a standard for the number of male gametes per female gamete or vice versa, and measuring the other cases against this standard. To account for unisexuality, a bounded measure is preferable that ranges symmetrically from pure maleness to pure femaleness as the extremes of sexual asymmetry. Given $h$ as the reference number of female gametes per male gamete (defining sexual symmetry), say, then the $m$ male gametes of an individual or class of individuals correspond to $m \cdot h$ female gametes. If $f$ is the number of female gametes, such an individual or class of individuals has an "effective" number $f + m \cdot h$ of gametes, among which a fraction

$$ S := \frac{f}{f + m \cdot h} $$

consists of female gametes. Thus, while increasing from 0 to 1, $S$ represents a continuum of sexuality reaching from pure maleness to pure femaleness and marks sexual asymmetry at $S = \frac{1}{2}$ (where $f/m = h$).

In fact, all of the measures discussed by Ross (1990) in his review are simple transformations of $S$ and apply to various references $h$. In particular, if only successful gametes (appearing in zygotes) are counted, then $h = 1$ for all such gametes in a population and $S$ equals the "gender" of Lloyd (1980) and the "functional sex" of Ross and Gregorius (1983). For the present purpose, two measures of sexual asymmetry are relevant, one, $S^G$, applying to the asymmetry between genotypes, and the other, $S^A$, applying to the age-specific asymmetry (asymmetry between age classes) of the same genotype. The measurement of $S^G$ is based on the intrinsic female and male fertilities with the reference $h$ specified by the resident homogynote $A_A$. Hence, $h = \varphi_{ij}/\mu_{ij}$, and the measure $S^G_{ij}$ of sexual asymmetry of the heterozygote becomes

$$ S^G_{ij} = \frac{\varphi_{ij}}{\varphi_{ij} + \mu_{ij} \cdot \varphi_{ij}/\mu_{ij}} $$

while, because of the choice of $h$, $S^G_{ii} = \frac{1}{2}$.

Concerning age-specific asymmetry of a genotype, the reference $h$ defining the state of sexual asymmetry is again derived from the gametic sex ratio at the age class equilibrium specified by the genotype's life history. For genotype $A_A$, $h$ is thus given by $\varphi_{ij}/\mu_{ij}$, and the measure of sexual asymmetry at age $k$ is specified by

$$ S^A_{ij}(k) = \frac{f_{ij}(k)}{f_{ij}(k) + \mu_{ij}(k) \cdot \varphi_{ij}/\mu_{ij}} $$

Note that across all ages either all $S^A$-values are identically $\frac{1}{2}$ (marking the case of complete age-specific sexual symmetry), or some are greater and others less than $\frac{1}{2}$. This follows from the fact that $\varphi_{ij}$ and $\mu_{ij}$ are averages of the $f_{ij}(k)$'s and the $m_{ij}(k)$'s, respectively, taken over the same age class frequencies.

**Sexual asymmetry between genotypes**: It follows from the definition of $\rho_{12}$ and the intrinsic fertilities of the heterozygote (see Table 3), that

$$ \sum_k \frac{1}{2} \left[ F_{12}(k) + M_{12}(k) \cdot \frac{\varphi_{ij}}{\mu_{ij}} \right] \cdot \rho_{12}^A $$

$$ = \frac{1}{2} \left[ 1 + \frac{\mu_{12} \cdot \varphi_{ij}}{\varphi_{12} \cdot \mu_{ij}} \right] = \frac{1}{2} S^G_{12}^{(2)}, $$

where the right side of this equation is verified by a simple rearrangement. As is seen from this expression in connection with Equation 2, the relations between $\rho_{12}$ and $\rho_{ij}^{(2)}$ are directly determined by the amount of sexual asymmetry $S^G_{ij}$ between the heterozygote and the resident homogynote. Together with the fact that functions of the type specified by the left side of Equation 2 are strictly increasing with increasing $\rho$, this yields the basic

**Result 1**: Under the present model assumptions, sexual asymmetry has the following effects on the establishment of mutant life histories:

(i) The resident genotype affects the initial growth rate of a mutant allele only via its intrinsic multiplication rate and its ratio of the intrinsic female to the intrinsic male fertility. Age-specific sexual asymmetry of the resident genotype thus has no effect.

(ii) If and only if sexual symmetry exists between the rare heterozygote and the resident homogynote ($S^G_{12} = \frac{1}{2}$) and thus $\mu_{12}/\varphi_{12} = \mu_{ij}/\varphi_{ij}$, the intrinsic and the incipient intrinsic multiplication rate of the rare heterozygote are identical ($\varphi_{ij}/\mu_{ij}^2 = \rho_{12}$). Thus, sexual asymmetry between genotypes affects the initial growth rate of the mutant even if all other parameters, including the intrinsic multiplication rate, are unaffected by the mutation.

(iii) The intrinsic multiplication rates being equal, sexual asymmetry between the resident homogynote and the heterozygote has the effect of increasing or decreasing the initial growth rate of the mutant allele according to whether $S^G_{12} < \frac{1}{2}$ (i.e., $\mu_{12}/\varphi_{12} > \mu_{ij}/\varphi_{ij}$) or $S^G_{12} > \frac{1}{2}$ (i.e., $\mu_{12}/\varphi_{12} < \mu_{ij}/\varphi_{ij}$), respectively.

Any modification of a life history that affects only
the male fertilities does not change the intrinsic multiplication rate as long as the numbers of fertilized ovules remain the same. Hence, the restriction introducing statement (iii) does not limit its applicability to an insignificant number of biologically relevant cases. However, even if the modification affects the (effective) female fertilities or the survival probabilities, statement (iii) is still meaningful, when the modification is conceived of as involving two steps: the first step results in a new intrinsic multiplication rate, and the second step specifies the deviation of this rate from the original rate as a consequence of the sexual asymmetry in the modification. It can therefore happen that a reduction in intrinsic multiplication rate is compensated by sexual asymmetry in favor of the male functions.

**Age-specific sexual asymmetry:** By part (i) of Result 1, the age-specific status of the resident homozygote does not affect the conditions for establishment of a mutant life history. Hence, the effect of the heterozygote cannot be explained by any type of modification in age-specific asymmetry in the homozygote. Moreover, by part (ii) of Result 1 sexual symmetry between the two genotypes rules out any effect of age-specific asymmetry in the heterozygote (of course only if it does not _per se _change the intrinsic multiplication rate). One therefore expects such effects only if the heterozygote results from a sexually asymmetric modification of the resident homozygote’s life history. To see this, consider the following decomposition of Equation 2:

\[
\frac{1}{2} \left[ 1 + \frac{\mu_{12}}{\varphi_{12}} \cdot \sum_k F_{12}(k) \cdot \rho^{-k} + \frac{\mu_{12}}{\varphi_{12}} \cdot \frac{\varphi_{12}}{\mu_{12}} \cdot M_{12}(k) \cdot \rho^{-k} \right] - \frac{1}{2} \sum_k M_{12}(k) \cdot \rho^{-k} = 1.
\]

Substitution by the age-specific asymmetry measures \(S_{12}^A(k)\) and by \(S_{12}^E\) in this equation yields

\[
\frac{1}{2} \left[ \frac{S_{12}^E}{S_{12}^A} \right]^{-1} \cdot \sum_k F_{12}(k) \cdot \rho^{-k} + \frac{1}{2} \cdot \frac{S_{12}^E}{S_{12}^A} \cdot \sum_k F_{12}(k) \cdot \rho^{-k} = 1,
\]

where the second summand disappears for sexual symmetry across all age classes. To generalize this observation let \(\rho_{12}^{\text{asym}}\) designate the value of \(\rho\) for which the left summand in Equation 4 equals 1, so that the case of age-specific sexual symmetry across all age classes (i.e., \(S_{12}^A(k) = \frac{1}{2}\) for all \(k\)) implies \(\rho_{12}^{\text{asym}} = \rho_{12}^{\text{o}}\). Considering again the monotonicity of the function specified by the right side of Equation 4, one therefore arrives at the

**Result 2:** For age-specific sexual symmetry across all age classes within the heterozygote, the incipient intrinsic multiplication rate is identical to the intrinsic multiplication rate of a life history derived from the original one by multiplying the female fertilities by a constant factor equating half the reciprocal of the amount of sexual asymmetry between the heterozygote and the resident homozygote (i.e., \(\frac{1}{2} \cdot \frac{S_{12}^{E}}{S_{12}^{A}}\)).

(i) Let \(\rho_{12}^{\text{asym}}\) be the positive solution for \(\rho\) in

\[
\frac{1}{2} \cdot \frac{S_{12}^{E}}{S_{12}^{A}} \cdot \sum_k F_{12}(k) \cdot \rho^{-k} = 1.
\]

Then age-specific sexual asymmetry within the heterozygote has the effect of increasing, not affecting, or decreasing the initial growth rate of the mutant allele \(A\), according to whether the term

\[
\sum_k F_{12}(k) \cdot \frac{1}{2} \cdot \frac{S_{12}^{E}}{S_{12}^{A}} \cdot \rho_{12}^{\text{asym}}^{-k}
\]

is positive, zero, or negative, respectively.

(ii) Sexual symmetry between the resident homozygote and the heterozygote cancels all age-specific effects of sexual asymmetry.

(iii) Age-specific asymmetry cannot reverse a tendency to increase or decrease the initial growth rate set by asymmetry between the genotypes.

The introductory statement of Result 2 follows from setting \(S_{12}^A(k) = \frac{1}{2}\) for all \(k\), so that equation (4) reduces to \(\sum_k [\frac{1}{2} \cdot \frac{S_{12}^{E}}{S_{12}^{A}} \cdot F_{12}(k)] \cdot \rho^{-k} = 1\). Part (iii) follows directly from part (iii) of Result 1.

A life history modification that illustrates Result 2 is obtained by consideration of a mutation that increases or decreases the male fertilities across all age classes by the same factor without affecting any other life history parameter. This change involves sexual asymmetry between the resident and the mutant type, but it leaves unaffected the intrinsic multiplication rate and the age-specific asymmetries. Hence, the age-specific asymmetry which has no effect in the resident homozygote becomes effective in the heterozygote and may there amplify or weaken the tendency set by the asymmetry between the genotypes.

**LIFE HISTORY SHIFTS**

The previous considerations of effects of sexual asymmetry on the initial growth rate did not explicitly take into account the implications of the distribution of an individual’s reproduction capacity over its life span. This will be the theme of the present chapter. To relate it to the intrinsic multiplication rate, the idea of distribution of reproduction over age will be based on the net fertilities \(F(k)\) at age \(k\) (see Table 2). The fraction of the total reproduction output of a cohort (which is proportional to the net reproduction rate \(\sum F(k)\)) that is attributable to individuals of age \(k\) is therefore

\[
D_F(k) = \frac{F(k)}{\sum_i F(i)}.
\]
and the set of these values will be referred to as the net reproduction distribution $D_F$ of a cohort with net fertilities $F$. The associated cumulative net reproduction distribution $C_F$ is then defined by the quantities

$$C_F(k) = \sum_{i=1}^{k} D_F(i).$$

This definition suggests to conceive of the intrinsic multiplication rate $p_F$ produced by the net fertilities $F$ as a product of the net reproduction distribution and the net reproduction rate $\sum_i F(k)$. One expects in particular that for fixed net reproduction distribution the intrinsic multiplication rate increases with increasing net reproduction rate. That this is true can be seen directly from the following rearrangement of the equation defining $p_F$:

$$\sum_k D_F(k) \cdot \rho^{-k} = \left[ \sum_k F(k) \right]^{-1}.$$ 

Indeed, in order to fulfill this equation, $\rho$ must strictly increase with increasing $\sum F(k)$. Moreover, this representation of the intrinsic multiplication rate reveals that it is completely determined by the net reproduction distribution and the net reproduction rate. Consequently, it remains to characterize the effects of changes in the net reproduction distribution on the intrinsic multiplication rate given that the net reproduction rate remains constant.

Among the various aspects for which two net reproduction distributions can be compared, timing of reproduction is among the most important in demographic theory, and within this scope changes resulting in earlier or later reproduction are of prime interest. The formulation of this concept is obvious in simple cases involving two reproduction cycles only: as the net fertility in the first cycle (age class) increases at the expense of the second cycle, reproduction output is moved to the earlier cycle. However, the concept requires a more sophisticated specification for arbitrary numbers of cycles and reproduction distributions. The following definition does this.

**Definition:** Consider two life histories with net reproduction distributions $D_F$ and $D_F'$ and with associated cumulative net reproduction distributions $C_F$ and $C_F'$. Then $D_F$ is said to derive from $D_F'$ by a shift of reproduction to earlier ages if $C_F(k) \geq C_F'(k)$ for all $k$, and $C_F(k) > C_F'(k)$ for at least one $k$.

The effect of such shifts on the intrinsic multiplication rate can be easily derived from the following mathematical identity, which holds for an arbitrary pair $D_F$ and $D_F'$ of net reproduction distributions and all nonzero values for $\rho$:

$$\sum_k [D_F(k) - D_F'(k)] \cdot \rho^{-k} = \rho \cdot \sum_k [C_F(k) - C_F'(k)] \cdot \rho^{-k}. \quad (5)$$

Thus, if $D_F$ can be derived from $D_F'$ by a shift in reproduction to earlier ages, then the above identity implies that $\sum_k (D_F(k) - D_F'(k)) \cdot \rho^{-k}$ is positive or negative according to whether $\rho$ is greater or less than 1, respectively. Recalling that between the intrinsic multiplication rate and the net reproduction rate one of the relations $\sum_i F(k) = 1 = p_F$, $1 < p_F < \sum_i F(k)$, or $\sum_i F(k) < p_F < 1$ is always realized (for more detailed relations see GREGORIUS 1979), this provides us with the desired

**Result 3:** The intrinsic multiplication rate of a life history is completely determined by its net reproduction distribution and net reproduction rate. For fixed net reproduction distribution, the intrinsic multiplication rate is a strictly increasing function of the net reproduction rate.

Consider two life tables with net fertilities $F$ and $F'$ exhibiting identical net reproduction rates, i.e., $\sum_i F(k) = \sum_i F'(k)$, and having intrinsic multiplication rates $p_F$ and $p_F'$, respectively. Furthermore, assume that the net reproduction distribution $D_F$ derives from $D_F'$ by a shift of reproduction to earlier ages. Then

(i) for a net reproduction rate greater than 1 (such as in growing populations) the shift increases the intrinsic multiplication rate (i.e., $p_F > p_F'$);

(ii) for a net reproduction rate less than 1 (such as in declining populations) the shift decreases the intrinsic multiplication rate (i.e., $p_F < p_F'$);

(iii) for a net reproduction rate equal to 1 (such as in stationary populations) no change in the net reproduction distribution whatever has an effect on the intrinsic multiplication rate (i.e., $p_F = p_F'$).

When applied to problems of the establishment of a modified life history, Result 3 reveals an interesting ecological population genetic aspect of iteroparity. At least for sexual symmetry between genotypes, a mutation causing a shift in reproduction to earlier ages (without changing the net reproduction rate) will not become established as long as the population lives at its carrying capacity. An environmental change increasing the carrying capacity without affecting the net reproduction distributions and without causing differential net reproduction rates among the genotypes then allows establishment of the mutant during the following phase of population growth. The same effect is of course produced by a temporarily acting environmental stress accompanied by a population decline. During the following recuperation phase the conditions for establishment are realized. Besides, this phenomenon helps to explain the observation that in many cases even within the same species there seems to be no consistent advantage to annual, biennial, or perennial modes of reproduction.

**Sexual asymmetry:** The question now is whether sexual asymmetry can invalidate the above principle for establishment. Since in this case male and female function may change largely independently of each
other, it is useful to explicitly distinguish female from male net reproduction distributions, where each is defined according to the above concept. Changes in survival probabilities will not be considered in the following, since, at least in bisexual organisms, they affect both sexes equally. Furthermore, as was already done above, all changes will be assumed to be restricted to the net reproduction distribution, so that no changes will be assumed to occur in the (female and male) net reproduction rates. The latter may be conceived of as a model for the allocation of female and male reproduction resources which is based on equal net reproduction outputs. This model avoids, for example, the conceptual problems arising with the evaluation of differential "costs" to be paid for the various states of allocation of resources.

First consider the male gametic sex. Any change in the male fertilities can be represented as in Equation 5, which includes as a special value for \( p \) the intrinsic multiplication rate \( p_F \), say, determined by the net female fertilities. Recalling the definition of the intrinsic male fertility \( \mu \) (see Table 3), it therefore follows that \( \mu \) increases, decreases, or is unaffected by a shift of male reproduction to earlier ages according to whether \( p_F > 1 \), \( p_F < 1 \), or \( p_F = 1 \), respectively. In terms of the amount of sexual asymmetry \( S_{12} \) between the modified (mutant) and the original (resident) life history, this implies \( S_{12} < \frac{1}{2}, \ S_{12} > \frac{1}{2}, \) or \( S_{12} = \frac{1}{2} \), respectively. Hence, by part (iii) of Result 1, a shift in the male gametic sex alone has the same effect as a sexually symmetric shift has on the initial growth rate of the modified life history.

To analyze the problem for a shift in the female gametic sex, recall that (see Table 3), \( \mu_{M/\varphi} = \sum \varphi_j \mu_j - \sum \varphi_j \mu_j \). Consequently, a shift of female reproduction affects \( S_{12} \) only via \( \mu_{12} \) such that the former is a strictly increasing function of the latter. Thus, a shift of female reproduction to earlier ages triggers effects of the intrinsic multiplication rate and the amount of sexual asymmetry on the initial growth rate, which act in opposite directions. Fortunately this indeterminacy can be easily solved by direct application of Equation 2. For constant net female and male reproduction rates, each shift in female reproduction induces an equivalent shift in the age-specific (successful male plus female) net fertilities \( \frac{1}{2} \left[ \sum \left( F_{12}(k) + M_{12}(k) \right) \varphi_{\mu/\mu_0} \right] \), and for both types of shift Equation 5 yields the same expression.

Hence, the opposing effect of sexual asymmetry cannot reverse the effect of the intrinsic multiplication rate on the initial growth rate, and thus shifts in female reproduction again show the same consequences for establishment as sexually symmetric shifts. In summary:

Result 4: For constant net female and male reproduction rates, a mutant causing a shift of reproduction to earlier ages in at least one of the gametic sexes becomes established in a growing but not in a declining population.

CONCLUDING REMARKS

The greatest advantage of considering intrinsic values apparently lies in their quality of summarizing the effects of a large number of complex variables in a single and intuitively plausible quantity. Thus, it is obvious that a mutant increasing male fertility without reducing the female fertility should gain an at least initial evolutionary advantage. Yet, as is stated in Result 1, this increase does not rule out the possibility that in some age classes male fertility may decrease; all that has to be guaranteed is that the intrinsic male fertility increases. This need not require an increase in resources used for the production of male gametes. However, the intrinsic male fertility cannot be increased indefinitely without being accompanied by a decrease in the intrinsic female multiplication rate or the intrinsic female fertility.

Probably unexpected is the finding (detailed in Result 2) that the role played by age-specific sexual asymmetry for evolutionary success is only secondary. In the present model, this form of asymmetry cannot override the effect of sexual asymmetry between the genotypes as specified by the ratio between intrinsic male and intrinsic female fertilities, and it becomes completely ineffective if there is no sexual asymmetry between the genotypes at all. Applying this to questions on the evolution of sequential hermaphroditism or less extreme forms of alternating female and male flowering across reproduction cycles, the conclusion is that the evolutionary success of these forms of alternating flowering essentially depends on their bearing on the intrinsic multiplication rate and the ratio between the intrinsic male and female fertility (i.e., the amount of sexual asymmetry between the mutant and the resident life history).

In the last analysis, the various forms of sexual asymmetry cannot be evaluated satisfactorily without setting any bounds to the resources available for the production of vegetative, female, and male tissue or without specification of the set of life history strategies considered (where the latter usually results in analyses of evolutionary or competitively stable strategies; see HASTINGS 1978). The analysis in the present paper suggests that these bounds be specified by the net female and male reproduction rates which define the total female and male reproduction output. The initial growth rate of a mutant life history is completely determined by these total net reproduction outputs and the mode according to which net reproduction is distributed across age classes. This cogently led to the analysis of shifts of reproduction to earlier or later ages for fixed total net reproduction outputs. The result (stated as Result 4) is surprising but nevertheless
desirable, since it reveals the ecological aspects of pure shifts of reproduction.

These shifts must not be confused with the usual analyses based on independent variation of survival and fertility parameters of the single age classes, since such analyses imply changes in both the total net reproduction output and the distribution of net reproduction across age classes. Thus, one of the more widely accepted ecological rules (probably due to Mertz 1971) stating that in non-growing populations increased survival at later ages is advantageous, while it is disadvantageous in growing populations, may rather be a consequence of an implied change in net reproduction rate than of the age shift itself. A similar comment applies to analogous changes in fertility [see Charlesworth (1980), p. 210 and p. 225]. As a consequence, different forms (models) of allocation of vegetative and reproductive resources may lead to opposing results on the evolutionary significance of age-dependent survival and fertility. For example, increasing viability at an early age at the cost of fertility at a later age may be advantageous for one but disadvantageous for another model of allocation of reproduction resources or life history strategy. In such cases the present analysis suggests separate consideration of the effects such models have on the net reproduction rate and the net reproduction distribution.

The observation that pure shifts of reproduction (female, male, or both in any proportions) to earlier ages are advantageous in growing but disadvantageous in declining populations, while shifts to later ages have the opposite consequence, has several interesting implications, some of which coincide with those derived from the above-mentioned earlier analyses. Considering that populations are usually not precisely stabilized at the carrying capacities of their environments and thus experience alternating phases of growth and decline, it is quite unlikely that the conditions for establishment are realized over a sufficiently long period of time to allow the mutant to reach substantial frequencies under climax conditions. This may be different for a colonizing species where the periods of population growth might last long enough for a mutant causing a shift of reproduction to earlier ages to become evolutionarily successful and possibly even to replace the resident type.

An interesting conclusion to be drawn from this finding is that a modified life history is very unlikely to become established and to persist under conditions of fluctuating population size unless this modification implies a relative increase in total female or male net reproduction output. Thus a transition from an annual to a biennial or perennial mode of reproduction (or vice versa), for example, is likely to be evolutionarily successful only if it entails a fitness gain in terms of the net reproduction rate. The same statement applies to the evolutionary fate of asymmetric allocation of sex function.

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