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On the moulding of senescence by natural selection in sexual and partly sexual populations

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Abstract. Under a wide variety of dynamic environmental conditions, natural selection appears to favor reproductive investment in a sexually produced offspring, carrying only half of the mother's genes, over the investment in an asexually produced offspring, genetically identical to her. It is maintained that the same environmental conditions must affect the evolutionary cost and benefit of an investment in the prolongation of one's own life versus an investment in sexual reproduction, in favor of the latter. The effects of different environmental conditions on the division of resources among sexual reproduction, asexual reproduction and prolongation of life are studied.

Keywords. Sex; senescence; changing environment.

1. Introduction

The phenomenon of senescence is characterized by an almost deterministic biological process which is significantly different from that of a random decay. The evolutionary significance of this rather universal phenomenon was first indicated and studied by Hamilton (1966), who tried to explain it on the basis of an optimal reliability model: taking into consideration the inevitable effects of random decay and predation, the chance of a living organism surviving over a certain period of time is bounded by a value that tends to zero as this period increases. Hence, there is no selective advantage in investing too much, even in a vital organ, in order to prolong its functioning beyond a certain time. But once a "decision" is made not to make such an investment, the chance of the organism functioning beyond the period in question diminishes even further. Hence, investment in other, say "cheaper", organs becomes useless as well, with the result of a certain synchronization in the decay of the various organs of the body.

In fact, the theoretical phenomenon observed by Hamilton in respect to living organisms is quite similar in character to a rather general property of optimally designed mechanisms, referred to as "positive aging". In the terminology of reliability theory, this term characterizes a random life-span $T \geqslant 0$ for which the value of the expected time $E(T-t|T \geqslant t)$, remaining to a mechanism (or organism) of age $t \geqslant 0$, is a decreasing function of t.

Yet we know of many biological systems for which the expectation $E(T-t \mid T \geqslant t)$ is, on the contrary, an increasing function of t, at least for a certain, sometimes quite long range of time (in which case, adopting the terminology of the reliability theory, one can speak of a "negative aging"). The fact that this is quite commonly true among juvenile organisms which do not yet invest in reproduction, at least indicates that positive aging may not necessarily be the only result of an optimal investment policy among living organisms. Indeed, such organisms may be distinguished from

all hitherto human designed mechanisms by their ability not only to perform their designated function (i.e. reproduction) but also to invest in rebuilding themselves. Moreover, the decision about which part of the resources available at a certain point should be invested in themselves can be continuously based on relevant up-to-date information. And a specifically relevant piece of information which is always available to a living organism is that he is, indeed, functioning at that very moment, regardless of how low the a-priori probability for this event was at the time he was, so to say, "designed". Thus, from mere arguments of optimality it is not that obvious that an investment in a fully new set of functioning organs (say, a newborn offspring) is preferable over successive investments in self-recuperation. Moreover, in most of the populations which are subject to this investigation, say in sexually reproducing ones, the alternative to an investment in the prolongation of one's own life is not a production of a fully identical copy of the organism but an investment in an offspring whose genes are only 50% identical to those of his parent. Thus, on the face of it, from the viewpoint of inclusive fitness maximization (Hamilton 1964, 1972), the problem seems to be even more serious in a sexual population.

Yet it is a theoretically well-established finding that the very process of sexual reproduction does lead to at least a short-term reduction in the inclusive fitness of the sexually reproducing organism, a reduction which has been referred to as the cost of meiosis (e.g. Maynard Smith 1978, see also Williams 1975), and although one can still argue about the reasons (e.g. Williams and Milton 1973, Williams 1975, Maynard Smith 1978, Hamilton 1980, 1982, Hamilton *et al.* 1981, Bell 1982, Bernstein *et al.* 1985, Weinshall 1986, Bell and Maynard Smith 1987) it is an undisputable fact that under a quite general condition, the process of natural selection does enable either full or partial sexual reproduction. This means that under these conditions, there must be some selective advantage to an investment in a sexually reproduced offspring over a similar investment in an asexually produced offspring, fully identical to the parent. Thus, it may not be that surprising that, under the same environmental conditions, investment in a sexually produced offspring is, all the same, preferred by natural selection over investment in the preservation of one's own organism. Indeed, for the dynamics of the population, preservation of one's own reproductive activity for one more generation is equivalent to the reproduction of a new, genetically identical organism which is to last for exactly one generation. It is therefore, suggested that the evolution and moulding of senescence should be re-examined in connection with the evolution of sexual reproduction.

In this work I take the approach of a permanent, presumably cyclic environmental change as the main cause for the evolution of sex (Hamilton 1980, 1982; Hamilton *et al.* 1981; Weinshall 1986; Weinshall and Eshel 1987; Eshel and Weinshall 1987, and references there). Indeed, in order for natural selection to prefer sexual over asexual reproduction, environmental changes (e.g. due to first parasite adaptation) must be so drastic as to render presently common combinations of genes rather obsolete within one or, at the most, a small number of generations. As such an environmental change affects the selective advantage of investment in asexual offspring, genetically identical to their parents, it appears that it must affect the selective advantage of investment in the prolongation of one's own life as well.

2. A general model for the allocation of investment in sexual reproduction, asexual reproduction and prolongation of life

Assume a large diploid population, either annual or perennial, subjected to the selection pressure of a permanently changing environment. The cause for such permanent environmental change may well be a host–parasite-cycling, yet one can assume any other source of a permanent environmental change as well. The fact that, at least on a theoretical level, such a permanent environmental change can stabilize (pure) sexual reproduction against the alternative of a (pure) asexual reproduction has been established in many theoretical works (e.g. Hamilton 1980, 1982, Hamilton *et al.* 1981, and references there). For the more general case of the stabilization of a given (unbeatable) level of sexual reproduction against any other level of sexual reproduction, see Weinshall and Eshel (1987) and Eshel and Weinshall (1987). Note, however, that all the models cited assume annual populations without overlapping generations for simplicity.

Assume now, more generally, that an individual in the population may survive for any number of periods, depending both on chance (say, outside events) and on the amount of resources invested in the prolongation of its own life. Assume, more specifically, that the total amount of resources available for an individual, already surviving for t periods, is w^t, where $w>0$ is any real value either larger or smaller than 1. This total amount of resources can be divided between sexual reproduction, asexual reproduction and prolongation of one's own life. The allocation of the resources into these three goals is determined by genes carried in one or a few loci which we refer to as the *secondary loci*. Genes carried in another group of loci, namely the *primary loci*, or (referring to a parasite pressure) the *immunological loci*, determine the viability of the carrier in different environmental situations. Let $\pi_1, \ldots, \pi_m$ be the different genotypes, as determined by the primary loci (say, the *immunological genotypes*) and let $S_1, \ldots, S_n$ be the different types of environments within a cycle ($n \geqslant 3$. See Weinshall 1986). Let $d_{i,j} \geqslant 0$ be the chance of an immunological genotype π_i to overcome the selection pressure of an environment of type $S_j (i = 1,, \ldots, m; j = 1, \ldots, n)$.

We assume further that, if an individual of such immunological genotype, while facing an environment of type S_j, invests a proportion α of its available resources in the prolongation of its own life, then its chance of survival to the next period is $d_{i,j} v(\alpha)$, where $v(\alpha)$ is any smoothly increasing function. In other words, $v(\alpha)$ is the chance that an individual, who has invested a proportion $0 \leqslant \alpha < 1$ of its resources in the prolongation of its own life, will survive to the next period, provided it has managed to cope successfully with the specific environmental pressure, independently of its immunological genotype and of the specific environmental pressure. (Note that in the most general case this survival probability may be $v_{i,j,t}(\alpha)$, depending on the immunological type, on the environment and also on the age t).

Assume, further that the proportions of resources invested in asexual and in sexual reproduction are β and τ respectively, $\beta, \tau \geqslant 0$, $\alpha + \beta + \tau = 1$. The numbers of offspring such an individual produces sexually and asexually are assumed to be proportional to the total amounts of resources, τw^t and βw^t respectively, invested in sexual and in asexual reproduction. In the general case it might be that the cost of a sexually produced offspring is different from that of an asexually produced one.

Without loss of generality we denote by τw^t and $(\mu/2)\beta w^t$ the number of sexually and asexually produced offspring, where μ is the cost of sex in term of parents' genes, passed to the next generation. (Thus, if the costs of sexually and asexually produced offspring are equal, then $\mu=2$, see Maynard Smith 1978.)

Note that the survival function $v(\alpha)$, as well as the parameters w and μ are determined by outside restrictions and are different from one population to another. We refer to them as the ecological parameters of the model and they are not supposed to be affected by natural selection. The parameters, α, β and τ, on the other hand, are determined by the individuals. We therefore can think of Evolutionarily Stable values which will be immune to any non-pleiotropic mutation affecting them (Hamilton 1967; Maynard Smith and Price 1973).

Thus, assume that these parameters are fixed in the population and let the frequencies of the immunological genotypes assume a permanent cycle. Denote by $x_{i,j}^{(k)}$ the frequency of a k-periods old π_i-type individual in a season of type S_j ($i=1,2,\ldots,m$; $j=1,2,\ldots,n$; $k=1,2,3,\ldots,$). Denote, further

$$x_{i,j}=\frac{\sum_{k=1}^{\infty} x_{i,j}^{(k)} w^k}{\sum_{l=1}^{m}\sum_{k=1}^{\infty} x_{l,j}^{(k)} w^k}. \tag{1}$$

This is the resources-weighted relative frequency of π_i-type individuals in an S_j-type period.

Denote further

$$y_{j,k}=\sum_{i=1}^{m} x_{i,j}^{(k)}. \tag{2}$$

This is the proportion of individuals of age k in an S_j-type period.

We start by analysing the relatively easy problem of an evolutionarily stable allocation of resources between asexual reproduction and prolongation of life when the proportion of resources invested in sexual reproduction is given.

3. Evolutionarily stable allocation of resources between asexual reproduction and prolongation of life

In this section we assume a mutation $B\to b$ which changes only the proportion of the two non-sexual investments. More specifically assume that the (rare) heterozygote Bb invests $\alpha+\theta$ and $\beta-\theta$ in prolongation of his own life and in asexual reproduction respectively, where θ is either a positive or a negative real number, small in absolute value.

Denote by $\varepsilon_{i,j,k}$ the relative frequency of the heterozygote mutant $\pi_i|Bb$ among k-period-old individuals in an S_j-type period. The (weighted) proportion of $\pi_i|Bb$ individuals of all ages within the population of the S_j-type period is

$$\varepsilon_{i,j}=\frac{\sum_{k=1}^{\infty} \varepsilon_{i,j,k} w^k y_{j,k}}{\sum_{k=1}^{\infty} w^k y_{j,k}}. \tag{3}$$

Denote by $\varepsilon'_{i,[j+1],k}$ and $\varepsilon'_{i,[j+1]}$ the appropriate proportion after one period where

$$[j+1]=\begin{cases} j+1, & \text{if } j<n \\ 1, & \text{if } j=n \end{cases}.$$

In order to facilitate the analysis, let us keep in mind the appearance of an alternative neutral mutation $B \to b'$ which might appear in the same proportions $\varepsilon_{i,j,k}$ at the same time. By $\tilde{\varepsilon}_{i,[j+1],k}$ and $\tilde{\varepsilon}_{i,[j+1]}$ we denote the appropriate proportion of the neutral mutation after one period.

Recall that the amount of sexual reproduction among mutant and non-mutant (or neutrally mutant) individuals is the same, hence the differences $\varepsilon_{i,[j+1],k} - \tilde{\varepsilon}_{i,[j+1],k}$ is given by the sum of the two differences: the difference between the appropriate proportion of surviving individuals and the difference between the appropriate proportion of asexually produced individuals.

Thus, denote by $\delta'_{i,[j+1],1}$ and $\tilde{\delta}_{i,[j+1],1}$ the proportions of $\pi_i|Bb$ and $\pi_i|Bb'$ individuals respectively, being born in the period $[j+1]$ to asexual reproduction among all new offspring (either sexual or asexual) born at this period.

By $\delta'_{i,[j+1],k}$ and $\tilde{\delta}_{i,[j+1],k}$ $(k>1)$ we denote the proportions of $\pi_i|Bb$ and $\pi_i|Bb'$ k-period-old individuals among all k-period-old individuals at the $[j+1]$th period. We get:

$$\delta'_{i,[j+1],1} = \frac{\mu(\beta-\theta)\, d_{i,[j+1]}}{2\bar{W}_j y_{[j+1],1}} \sum_{k=1}^{\infty} y_{j,k}\, \varepsilon_{i,j,k}\, w^k, \tag{4}$$

$$\tilde{\delta}_{i,[j+1],1} = \frac{\mu\beta d_{i,[j+1]}}{2\bar{W}_j y_{[j+1],1}} \sum_{k=1}^{\infty} y_{j,k}\, \varepsilon_{i,j,k}\, w^k, \tag{5}$$

where $\bar{W}_j$ is the average fitness in the population at period S_j. For $k>1$ we get

$$\delta'_{i,[j+1],k} = \frac{v(\alpha+\theta)\, d_{i,[j+1]}\, y_{j,k-1}\, \varepsilon_{i,j,k-1}}{\bar{W}_j y_{[j+1],k}}, \tag{6}$$

$$\tilde{\delta}_{i,[j+1],k} = \frac{v(\alpha)\, d_{i,[j+1]}\, y_{j,k-1}\, \varepsilon_{i,j,k-1}}{\bar{W}_j y_{[j+1],k}}. \tag{7}$$

Hence

$$\varepsilon'_{i,[j+1],1} - \tilde{\varepsilon}_{i,[j+1],1} = -\frac{\mu\theta d_{i,[j+1]}}{2\bar{W}_j y_{[j+1],1}} \sum_{k=1}^{\infty} y_{j,k} w^k \varepsilon_{i,j,k}, \tag{8}$$

and for $k>1$

$$\varepsilon'_{i,[j+1],k} - \tilde{\varepsilon}_{i,[j+1],k} = \frac{[v(\alpha+\theta)-v(\alpha)]\, d_{i,[j+1]}}{\bar{W}_j y_{[j+1],k}}\, y_{j,k-1}\, \varepsilon_{i,j,k-1}. \tag{9}$$

Thus from (3), (4) and (5) one gets:

$$\varepsilon'_{i,[j+1]} - \tilde{\varepsilon}_{i,[j+1]} = \frac{\sum_{k=1}^{\infty} (\varepsilon'_{i,[j+1],k} - \tilde{\varepsilon}_{i,[j+1],k})\, y_{[j+1],k}\, w^k}{\sum_{k=1}^{\infty} y_{[j+1],k}\, w^k} \tag{10}$$

$$= \frac{\{[v(\alpha+\theta)-v(\alpha)]\, d_{i,[j+1]} \sum_{k=2}^{\infty} y_{j,k-1}\, w^k \varepsilon_{i,j,k-1} - \frac{\theta\mu d_{i,[j+1]}}{2} \sum_{k=1}^{\infty} y_{j,k}\, w^k \varepsilon_{i,j,k}\}}{\bar{W}_j \sum_{k=1}^{\infty} y_{[j+1],k}\, w^k}$$

$$=\left\{[v(\alpha+\theta)-v(\alpha)]\,w-\frac{\theta\mu}{2}\right\}\frac{d_{i,[j+1]}\sum_{k=1}^{\infty}y_{j,k}\,w^k\,\varepsilon_{i,j,k}}{\bar{W}_j\sum_{k=1}^{\infty}y_{[j+1],k}\,w^k}.$$

Denote by $\varepsilon'^{(n)}_{i,j}$ and $\tilde{\varepsilon}^{(n)}_{i,j}$ the appropriate proportion of the mutants $\pi_i|Bb$ and $\pi_i|Bb'$ respectively after a full cycle of n periods. From (10) we get

$$\varepsilon'^{(n)}_{i,j}-\tilde{\varepsilon}^{(n)}_{i,j}=\left\{[v(\alpha+\theta)-v(\alpha)]\,w-\frac{\theta\mu}{2}\right\}\Gamma_{i,j}(\|\varepsilon_{i,j,k}\|), \tag{11}$$

where $\Gamma_{i,j}>0$ is of the order of the values $\varepsilon_{r,l,k}$.

It is easy to verify, though, that under the assumption that $B\to b'$ is a neutral mutation in an infinite population, the vector $\{\tilde{\varepsilon}^{(tn)}_{i,j}\}_{i=1}^{m}$ of the neutral-mutant's relative frequencies after t cycles tends to a limit $\{\varepsilon^{*}_{i,j}\}_{i=1}^{m}$ of the same order. From (11) it, therefore follows that the mutant b disappears from the population if and only if

$$[v(\alpha+\theta)-v(\alpha)]w<\frac{\theta\mu}{2}. \tag{12}$$

Thus, given the proportion $0\leqslant\tau<1$ of resources invested in sexual reproduction, the ratio $\alpha:\beta$ between the investments in prolongation of life and in asexual reproduction is evolutionarily stable (i.e. immune to any invasion by non-pleiotropic mutations affecting this ratio, see Hamilton 1967, Maynard Smith and Price 1973) if (12) holds for any value θ, either positive or negative. A necessary condition for this is, indeed, that the ratio $\alpha:\beta$ will be stable against mutations of small effect, say against all mutations with θ's which are small in absolute value.

In this case (12) becomes

$$\theta\left[wv'(\alpha)-\frac{\mu}{2}\right]+\frac{\theta^2 w}{2}v''(\alpha)+o(\theta^2)<0, \tag{13}$$

for any θ sufficiently small in absolute value. Hence we get

Corollary 1

Given a rate of investment in sexual reproduction $0\leqslant\tau<1$, a necessary and almost sufficient condition for the evolutionary stability of (α,β) against mutations of small effect is either one of the following:

$$\left.\begin{aligned}&\alpha=0\\&wv'(0)-\frac{\mu}{2}\leqslant 0\end{aligned}\right\} \tag{14}$$

or

$$\left.\begin{aligned}&0<\alpha<1-\tau\\&wv'(\alpha)-\frac{\mu}{2}=0\\&v''(\alpha)\leqslant 0\end{aligned}\right\} \tag{15}$$

or

$$\left.\begin{aligned}&\alpha=1-\tau\\&wv'(1-\tau)-\frac{\mu}{2}\geqslant 0\end{aligned}\right\}. \tag{16}$$

A sufficient condition is given by (14), (15) or (16) with a sharp inequality. In this case, each of the three conditions guarantees the stronger condition of continuous stability (Eshel 1983) namely: If α satisfies any of the three conditions (14), (15) or (16) and if the investment in prolongation of life is close to α, then natural selection will favor mutations which determine an investment which is even closer to α. It will operate against mutations which determine an investment further away from α.

It is easy to verify that at least one of the 3 alternative conditions for a local evolutionarily stable strategy (ESS) investment in prolongation of life should be satisfied and, except for singular cases, it should be satisfied as a sharp inequality. It is possible, though, that two of the three or, maybe all three will be satisfied simultaneously. Moreover, (15) can, theoretically, be satisfied for different values of α between 0 and $1-\tau$. While each of the solutions will be stable against mutations of small effect, only one of the solutions can be globally stable against all mutations (except for a singular case in which two solutions will be mutually neutral). In order to see this, assume that α_1 and α_2 are values on the interval $[0, 1-\tau]$, each satisfying one of the conditions (14)–(16) for stability against mutation of small effect.

Refer to (12) and insert $\alpha=\alpha_1$, $\alpha+\theta=\alpha_2$, then in order for α_1 to be stable against a mutation determining an investment α_2 in prolongation of life, it is required that

$$v(\alpha_2)-v(\alpha_1)<\frac{(\alpha_2-\alpha_1)\mu}{2w}. \tag{17a}$$

If, on the other hand, we denote $\alpha=\alpha_2$, $\alpha+\theta=\alpha_1$, then the condition for stability of α_2 against a mutation determining an investment α_1 in prolongation of life is

$$v(\alpha_1)-v(\alpha_2)<\frac{(\alpha_1-\alpha_2)\mu}{2w}. \tag{17b}$$

Indeed (17a) and (17b) cannot possibly be satisfied simultaneously.

Note, however, that $w\theta v'(\alpha)+o(\theta)$ is the gain in one's own next period fitness resulting from an increase θ in the investment in the prolongation of one's own life, $\theta\mu/2$ is the expected gain in terms of number of asexual offspring, resulted from an increase θ in the investment in such offspring. The term $\theta[wv'(\alpha)-(\mu/2)]$ is, therefore, the increment in one's own inclusive fitness, resultant from transferring a small proportion θ of the resources from asexual reproduction to the prolongation of one's own life.

Corollary 1 can thus be written as:

Corollary 2

Natural selection, when operating through mutations of small effect on the division of resources between asexual reproduction and prolongation of life, always operates to increase the inclusive fitness of the parent. It increases the proportion of resources invested in prolongation of life if $v'(\alpha)>\mu/2w$ *and decreases this proportion if* $v'(\alpha)<\mu/2w$.

4. Sexual reproduction versus either asexual reproduction or prolongation of life

In contrast to the result of corollary 2 of the previous section, concerning division of resources between asexual reproduction and prolongation of life, investment in sexual instead of asexual reproduction must decrease the immediate inclusive fitness of the parent by a factor $1:\mu$, where μ is the cost of meiosis. Employing corollary 2, one may infer that, as long as an evolutionarily stable division of resources is maintained between asexual reproduction and prolongation of life, any infinitesimal transfer of resources from the latter to sexual reproduction must cause, all the same, an immediate reduction of the inclusive fitness by the same factor of $1:\mu$. Thus corollaries 1 and 2 turn the problem of an ESS allocation of resources $(\alpha^*, \beta^*, \tau^*)$ into a one-parameter problem of an ESS investment τ^* in sexual reproduction, when a functional relation between α^* and β^* is given. Yet, even this problem is complicated and has not yet been solved even for the annual case $\alpha=0$, unless under quite special, rather controversial assumptions (see Weinshall and Eshel 1987, Eshel and Weinshall 1987, which to my knowledge represent the only dynamic models to deal with an evolutionarily stable rate of sexual reproduction rather than with a competition between fully sexual and fully asexual reproduction).

As mentioned above, this work is not concerned with the question of which population dynamics does, in fact, lead to the establishment of sexual reproduction, say to the stabilization of the ESS triple $(\alpha^*, \beta^*, \tau^*)$ with $\tau^*>0$. Instead, it is assumed that since sexual reproduction does, in fact, exist in natural populations, there exists also a population dynamics that stabilizes it, despite the immediate reduction in inclusive fitness entailed. It is assumed, further, that different ecological parameters are likely to affect such a population dynamics in a way that leads to the stabilization of different rates of sexual reproduction (see, for example, Weinshall and Eshel 1987). While all models for the evolution of sex concentrate for simplicity, on the case of annual populations with non-overlapping generations, it should be noticed that, in a perennial population investment in sexual reproduction may replace an investment either in asexual reproduction or in prolongation of life.

One question which we ask in this section is how an increase in the rate of sexuality in the population affects, besides other ecological factors, the stable rate of investment in the prolongation of life and, therefore, the lifespan and possibly, early senescence in the population. Another question to be dealt with elsewhere is how, on the contrary, changes in the rate of investment in prolongation of life due to direct ecological factors [i.e. due to changes in the shape of the survival function $v(\alpha)$] affect the stable rate of sexuality τ^* in the population.

We start with the simplest case in which

$$v'(0)>\frac{\mu}{2w}, \tag{18}$$

and the equation

$$v'(\alpha)=\frac{\mu}{2w}, \text{ with } v''(\alpha)<0, \tag{19}$$

has a unique solution $0<\hat{\alpha}<1$. This is the case when, with the absence of sexual reproduction, there exists a unique optimal division of resources between asexual

reproduction and prolongation of life. We then assume that a stable rate of sexuality $\tau^* \geqslant 0$ exists, depending on various ecological factors.

In this case, if $\tau^* > 1-\hat{\alpha}$, then for any admissible value of α, $\alpha \leqslant 1-\tau^* < \hat{\alpha}$ and, as it follows from (18) and (19), $v'(\alpha) > (\mu/2w)$. From corollary 1 it therefore follows that natural selection will always operate to increase α to its maximal value $\alpha^* = 1-\tau^*$ and the only evolutionarily stable allocation of resources will be given by the triple

$$(\alpha^*, \beta^*, \tau^*) = (1-\tau^*, 0, \tau^*), \tag{20}$$

which allows for no asexual reproduction.

If, on the other hand, $\tau^* < 1-\hat{\alpha}$, it follows from corollary 1 [together with (18) and (19)] that the only evolutionary stable allocation of resources is given by

$$(\alpha^*, \beta^*, \tau^*) = (\hat{\alpha}, 1-\tau^*-\hat{\alpha}, \tau^*). \tag{21}$$

We, therefore, get:

Corollary 4

In the simple case (18)–(19), an increase in the investment τ^* in sexual reproduction from 0 to $1-\hat{\alpha}$ does not affect the evolutionarily stable investment in prolongation of life (which remains $\hat{\alpha}$) and therefore does not affect the distribution of the individual lifespan.

The evolutionarily stable investment $\alpha^* = \hat{\alpha}$ in the prolongation of life will satisfy, in this region, the requirement of the reliability model of Hamilton (1966). Any increase in the investment in sexual reproduction, within this region, will be at the expense of the rate of asexual reproduction. We therefore refer to the region $0 \leqslant \tau^* \leqslant 1-\hat{\alpha}$ as the *Hamiltonian region* of senescence.

On the other hand, as the investment in sexual reproduction surpasses the critical value $1-\hat{\alpha}$, any asexual reproduction disappears and, hence, any further increase in the stable rate of sexual reproduction τ^* must be at the expense of the investment in the prolongation of life. In this case, earlier senescence must be an inevitable result of natural selection in favour of sexual reproduction. This sort of early senescence cannot be explained on the basis of the optimal reliability model. We therefore refer to the region $\tau^* > 1-\hat{\alpha}$, characterized by lack of asexual reproduction, as the *non-Hamiltonian* region of senescence.

We now relax the condition (18)–(19) and assume that the equations

$$v'(\alpha) = \frac{\mu}{2w};\ v''(\alpha) \leqslant 0$$

or

$$v'(0) \leqslant \frac{\mu}{2w},$$

have at least two solutions (including, maybe, the solution $\alpha = 0$). Denote these solutions by α_i, $0 \leqslant \alpha_1 < \alpha_2 < \ldots < \alpha_l$. As we know, only one of them is a global ESS. We denote this one by $\hat{\alpha}$.

In this case, if the stable investment in sexual reproduction τ^* is less than $1-\hat{\alpha}$, then we can still speak on the Hamiltonian rate of investment in prolongation of life. Exactly as in the previous case, any increase in τ^* within the Hamiltonian region $0 \leqslant \tau^* \leqslant 1-\hat{\alpha}$ will be at the expense of asexual reproduction. The stable

investment in prolongation of life will remain intact, obeying the optimal rules of the reliability model. Moreover, as before, once the stable investment in sex, τ^*, surpasses the critical value $1-\hat{\alpha}$, all investment in asexual reproduction disappears, and all further investment in sexual reproduction, at least in the right vicinity of $1-\hat{\alpha}$, must be at the expense of the prolongation of life. Yet, if $0 \leqslant \alpha_1 < \hat{\alpha}$ we know that the investment α_1 in the prolongation of life is at least stable against mutations of limited effect (local ESS). Moreover, this rate is stable against all mutations which cause a decrease of α below α_1. Hence there exists a critical value $\lambda > 0$ such that if $1-\alpha_1-\lambda < \tau^* < 1-\alpha_1$, then the evolutionarily stable rate of investment in the prolongation of life would become α_1. Again, this rate will remain intact as long as τ^* increases from $1-\alpha_1-\lambda$ to $1-\alpha_1$. Within this region, a positive rate of investment $1-\alpha_1-\tau^*$ in asexual reproduction reappears in a discontinuous way and any increase in sexual reproduction will replace asexual reproduction without affecting the life span of the individual. We can therefore speak about a *secondary Hamiltonian region* $1-\alpha_1-\lambda < \tau^* < 1-\alpha_1$ in which arguments of optimization become, again, relevant to the investment in the prolongation of life, although this time under a different restriction about the resources $1-\tau^*$ available for both asexual reproduction and prolongation of life.

A case of specific biological interest maybe the one in which $\alpha_1 = 0$ and $\alpha_2 = \hat{\alpha} > 0$. In this case, as long as $\tau^* < 1-\alpha_2$ one may observe a mixture of sexual and asexual reproduction, with a fixed investment α_2 in the prolongation of life, regardless of τ^*. As the environmental conditions become more favorable to sexual reproduction and τ^* surpasses $1-\alpha_2$, asexual reproduction disappears and any further increase in sexual reproduction leads to a monotone and continuous decrease in life span, as $\alpha = 1-\tau^*$ decreases. Yet as τ^* surpasses some critical value $1 > 1-\lambda > 1-\alpha_2$, a discontinuous change in life-span occurs and the population becomes annual as the new optimal investment in the prolongation of life becomes $\alpha_1 = 0$. At the same time, a proportion $1-\tau^* > 0$ of the resources are re-invested in asexual reproduction. This case may correspond to situations of sibling species, one being perennial with fully sexual reproduction, the other being annual with mixed sexual and asexual reproduction.

5. Theoretical predictions and empirical observations

In a different work (Eshel and Weinshall 1987) it has been shown that, in the simplest case of an annual population with a stably maintained rate of partial sexuality and with any positive cost of meiosis, sexually-produced offspring are not necessarily (and not generally) fitter, on the average, than asexually-produced ones. This finding stands in contrast to the common, intuitive expectation that, in order for sexual reproduction to be maintained in the population, an average advantage of sexually produced genotypes should be manifested and, moreover, be substantial enough to at least compensate for the cost of meiosis (a widespread expectation that, despite the large-scale selection forces involved, has never been substantiated by direct evidence!). Instead it has been established (Eshel and Weinshall 1987) that the selective advantage of sexual reproduction expresses itself in later generations.

This can be intuitively based on the previous finding of Weinshall (1986) that sexual reproduction can be most easily advantageous when the number of different parasite-types in a cycle is larger than the number of relevant immunological

possibilities of a single genotype (e.g. when a specific allele in a specific, single locus is required to cope with each parasite, then the minimal length of a cycle required for the evolution of sex is three). In this case, no asexual clone can possibly survive the whole cycle without any interval of sexual reproduction. It is, thus, clear that in such a case, the chance for survival of an asexually produced offspring decreases as the number of asexual generations in its clone approaches the length of the cycle. Less obvious is the analytically drawn result that the survival probability of sexually produced offspring is also lower if their parents or, to an extent, even one of them, are taken from asexual clones. The intuitive reason for this is that asexual clones, surviving under the same (changing) environmental conditions are prone to lose the same genes and, more importantly, just those genes which are most likely to be required at the next stages of the cycle.

While this result has been obtained for an annual population, it can be easily generalized to a perennial population in which an individual of the age of k is taken to be equivalent to an asexually-produced offspring of a k-long clone. It is therefore predicted that offspring of an old parent (or parents, in the case of a sexually produced one) will be less viable than the offspring of a young one (or ones). Since this is more drastically true for asexually produced offspring, one prediction is that in a perennial population with partially-sexual reproduction, old individuals (e.g. trees) will tend to invest less in asexual reproduction (e.g. in a vegetative one). Another prediction is that a difference in viability (or fitness) between offspring of young and of old parents will still be manifested (though in a less drastic way) among sexually produced offspring. Indeed, this difference should be looked for in natural populations which are still prone to selection by parasite pressure. A relatively simple experiment, comparing the viability of seeds taken from plants of different ages, is now under investigation in Tel Aviv University.

Next, if sexually produced offspring of young parents are, on the average more viable than those of old parents, it is expected that individuals of both sexes and of either age, will prefer younger mates, as long as they are mature. Indeed, preference for young mates among humans as well as in other monogamous populations cannot be taken as an empirical evidence because of the apparent advantage of a mate with higher life expectancy in this case. Yet reports of sneak matings of female baboons with low-rank young males do not yet have any other explanation on the basis of selective advantage.

One observed phenomenon that can strictly distinguish between the model suggested here and the alternative pure reliability model, is that of sexual sterility in still socially active old individuals, common among humans of both sexes, female deer and female elephants. In this case, sexual-senescence cannot possibly be a result of a mere synchronized collapse in the functioning of the organisms since the individual is still active in an efficient way in order to help other individuals in his family. Yet, if the expected viability of his newly born offspring is indeed decreasing with the change of the environment it might be selectively advantageous for the old, though yet active individual, to direct his investment in helping younger reproductive members of his family.

For the special case of short host–parasite cycling with an age-dependent allocation of reproductive resources by the host, the reader is referred to Eshel and Sansone (1990).

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