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ESSAY REVIEW

What evolution maximizes

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There has been controversy in recent years as to the quantities, if any, that are maximized during evolution. Some biologists have been sceptical on whether any quantity is maximized (e.g. Pierce & Ollason, 1987; articles in Dupré, 1987) and others have expressed concern at the lack of a consensus (see Nur, 1987; Maynard Smith, 1978; for reviews). My aim here is to briefly review the problem, and to argue in favour of a 'selfish gene' solution, as advocated persuasively by Dawkins (1976, 1982, 1986). I shall use gene and allele synonymously (following e.g. Crow, 1986). Briefly, Dawkins argues that evolutionary insights are achieved, and pitfalls avoided, by considering the fate of a gene (i.e. an allele) coding for whatever trait(s) is (are) under study. Thus, for example, the study of altruism has been forwarded by considering whether or not a gene that caused individuals to help relatives at some cost to themselves would spread.

Formally, the question is whether or not a gene with specified properties will spread, in a specified population and environment, faster than others with similar but not identical properties. Clearly alleles now common have, at some past time, spread in populations, and have not been displaced by alternative alleles. In other words, such alleles had a higher (i.e. positive) rate of increase when they were spreading, and competitor alleles which were not able to invade, had lower, possibly negative, rates of increase. Hence selected alleles have had a higher rate of increase than their competitors, and this shows that the rate of increase of an allele is a maximized quantity. Rate of increase must decline, of course, as an allele nears fixation and the genetic composition of the population changes. A full analysis should therefore compare the rates of increase of rival alleles at all stages (see below).

Most biologists would surely accept the view, that rate of increase of an allele is maximized. Rate of increase of a gene is straightforward to calculate if all carriers of the gene have the same, known, life cycle and this is shown in detail in the next section. For many theoretical purposes, this will be a sufficient definition of fitness. However homozygous and heterozygous individuals may have different life cycles, so that different genotypes have different selective effects. This is the stuff of classical population genetics and methods are available that use the proportions and rates of increase of the different genotypes to calculate the rate of increase of the component genes (see e.g. Crow, 1986). However, there can be major problems in calculating the rate of increase of a genotype, because the newborn of the genotype are not necessarily the offspring of parents of that genotype (Denniston, 1978; Nur, 1987; Charlesworth, 1970). For example, AB individuals can be the offspring of AA × AB, AA × BB, etc. Hence the rate of increase of a genotype cannot be derived solely from the life table of that genotype, since information is also needed on the life tables of the other genotypes in the population. Consequently the Malthusian parameter for a genotype is not necessarily a good estimator of its rate of increase (Denniston, 1978; Nur, 1987).

However, these problems do not arise in estimating the rate of increase of an allele. In this case the newborn containing the allele are necessarily the offspring of parents that, between them, must contain at least one copy of the allele. Indeed, in the absence of meiotic drive, the chance of a copy in a parent getting into a specified offspring via meiosis is ½ (Mendel's first law). If all carriers of the allele have the same life table, then the Malthusian parameter does estimate the rate of increase of the allele (see below). Thus although the Malthusian parameter of a genotype has sometimes been regarded as the quantity maximized in the evolutionary process, it seems in every way preferable to use the rate of increase of an allele. Following Sibly & Calow (1986a) I shall refer to this as the fitness, F, of the allele, in a specified population and environment (see below).

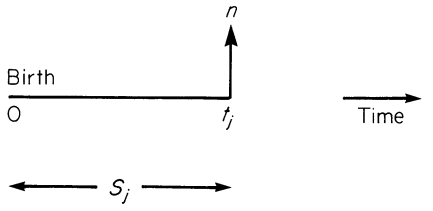

Fig. 1. The semelparous life cycle.

Calculation of the rate of increase of an allele

(1) *Semelparous life cycles*

S_j is the probability of individuals carrying the gene surviving to age t_j, at which point each gives birth (or sires) n offspring, half of which (on average) receive a copy of each allele present in a parent (Fig. 1). Thus there are $\frac{1}{2}n$ copies in offspring for each copy present in a parent. However, individuals survive to breed with chance S_j, so there are $\frac{1}{2}n\,S_j$ copies in offspring at time t_j for each copy present in offspring at time 0.

It is often convenient to use an instantaneous rate of increase, and this is what I am calling fitness, F. A good analogy occurs in the financial markets, where rates of increase are usually presented as instantaneous rates but the annual increment is also sometimes calculated. The annual increment is slightly greater than the instantaneous rate of increase. An investment of one dollar is worth e^F dollars after a year, representing an increase of $e^F - 1$, i.e. slightly more than F.

In the present context there are e^{Ft_j} copies of the gene at time t_j for each copy present at time 0, so $e^{Ft_j} = \frac{1}{2}n\,S_j$. This equation can also be written as

$$F = \frac{1}{t_j}\log_e(\tfrac{1}{2}n\,S_j), \qquad \text{Equation 1}$$

or as

$$1 = \tfrac{1}{2}n\,S_j\,e^{-Ft_j} \qquad \text{Equation 2}$$

for comparison with the equation relevant to more complex life cycles (see below).

(2) *Annual iteroparous life cycle*

The life cycle is as before except that adults survive after reproduction, i.e. there is a chance, S_a, that they live to the following breeding season and they may potentially live for ever (Fig. 2). In other words there is no senescence built into this model. The interval between breeding seasons is the same as the juvenile development period. As in model 1, for each allele in an individual about to breed, $n/2$ copies occur in its offspring (on average) and these survive to breed with chance S_j. Hence $\frac{1}{2}n\,S_j$ copies get to next breeding via offspring. In addition, the adult itself may survive, with chance S_a. Hence in total $\frac{1}{2}n\,S_j + S_a$ copies reach next breeding for each copy in existence now.

Thus $e^{Ft_j} = \frac{1}{2}n\,S_j + S_a$ Equation 3

or $1 = \frac{1}{2}n\,S_j e^{-Ft_j} + S_a e^{-Ft_j}$ Equation 4

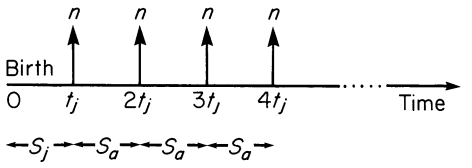

Fig. 2. The annual iteroparous life cycle.

(3) *Simple iteroparous model*

The life cycle is as in model 2 except that the juvenile development period t_j may differ from the interval between breeding attempts t_a (Fig. 3). Since we may henceforth be interested in variation in the timing of breeding, it is attractive to replace survivorship variables (S_j and S_a) by expressions involving t_j and t_a and the appropriate mortality rates, μ_j and μ_a. This is because survivorship declines with time, so that S and t are not independent variables, whereas μ and t are. Survivorship S over a period t when the mortality rate is μ is given by the expression $S = e^{-\mu t}$.

Consider all copies N of the gene that are in adults about to reproduce at some time X. Some copies are in adults which last bred t_a years ago when there were $N\,e^{-Ft_a}$ copies in adults about to breed. These adults survived to X with chance $e^{-\mu_a t_a}$. Thus $e^{-\mu_a t_a} N\,e^{-Ft_a}$ genes come via this route.

The other copies come via offspring born t_j ago, when there were $N\,e^{-Ft_j}$ copies in adults about to breed. Those adults produced n offspring each, so $\frac{1}{2}n\,N\,e^{-Ft_j}$ copies appeared in offspring, which survived to X with chance $e^{-\mu_j t_j}$. Thus $\frac{1}{2}n\,N\,e^{-\mu_j t_j}\,e^{-Ft_j}$ genes came by this route.

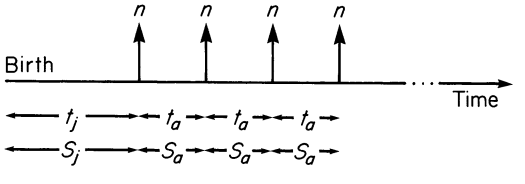

Fig. 3. The simple iteroparous life cycle.

Thus the total number of adults about to breed at time X is

$$N = \tfrac{1}{2}n\,N e^{-\mu_j t_j - Ft_j} + N e^{-\mu_a t_a - Ft_a} \qquad \text{Equation 5}$$

Dividing by N we have

$$1 = \tfrac{1}{2}n\, e^{-\mu_j t_j - Ft_j} + e^{-\mu_a t_a - Ft_a} \qquad \text{Equation 6}$$

(4) *General iteroparous model*

The life cycle is completely general, with n_1 offspring being produced at age t_1, n_2 at t_2 and so on (Fig. 4). Age at last breeding will be written t_Ω. Survivorship from birth to t_i will be written l_i.

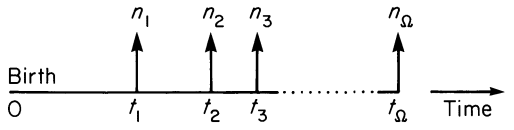

Fig. 4. The general iteroparous life cycle.

Consider all copies N of the gene that appear in offspring at time X. Some copies come from parents which are t_1 years old. The number of copies that appeared in offspring t_1 years ago was $N e^{-Ft_1}$. These copies survived to breeding age with chance l_1 and then each produced $\tfrac{1}{2}\, n_1$ copies in offspring.

Thus $\tfrac{1}{2}\, N e^{-Ft_1}\, l_1 n_1$ genes come from parents t_1 years old. Similarly $\tfrac{1}{2}\, N\, e^{-Ft_2}\, l_2 n_2$ genes come from parents t_2 years old, and so on. The sum of all these is the total number of copies appearing in offspring at time X, i.e.

$$N = \tfrac{1}{2}\, N e^{-Ft_1}\, l_1 n_1 + \tfrac{1}{2}\, N e^{-Ft_2}\, l_2 n_2 + \ldots + \tfrac{1}{2}\, N e^{-Ft_\Omega}\, l_\Omega n_\Omega$$

$$\therefore\ 1 = \tfrac{1}{2} \sum_{i=1}^{\Omega} e^{-Ft_i}\, l_i n_i \qquad \text{Equation 7}$$

Assumptions

In all these models it is assumed that all carriers of an allele have the same life cycle. In particular, life cycles do not differ between sexes or generations. Since fecundity varies with age in model 4, it is necessary here that males mate with females of the same age: otherwise they could not achieve the fecundity characteristic of their ages. In models 3 and 4 it is necessary to assume that the instantaneous rate of increase, F, is constant over some period. In model 3 this period is the greater of t_j and t_a, and in model 4 it is t_Ω. Using this approach no assumption is necessary about the stability of the age distribution.

Linton *et al.* (in preparation) have made some progress in relaxing these assumptions using computer simulation. They chose to model the annual iteroparous life cycle to avoid the timing problems inherent in the more complex models and discovered that the same strategies evolved whether or not the gene was expressed in both sexes. Not surprisingly, however, evolution was faster if the gene was expressed than if it was silent in the second sex. In a second set of simulations they discovered that variation in survivorships between generations had surprisingly little effect on either the rate or the outcome of evolution, even when the standard deviation of the variation was equal to its mean. This condition occurs, for example, when survivorship oscillates between 0 and some maximum value, i.e. a fairly extreme form of variation.

This suggests that the effects of population fluctuations and unstable age distributions will not be marked but there is scope for further work in this area. An initial problem is to decide what types of fluctuation and age distribution should be investigated. Some important mathematical results in this field have been obtained by Tuljapurkar (1982). The evolutionary effects of density dependence have been considered by Sibly & Calow (1986a, 1987) and Bulmer (1985).

Changes as a gene approaches fixation

As a gene spreads in the population, its rate of increase must eventually decline to zero and the pattern of spread that is usually assumed is shown in Fig. 5. As noted in the previous section, it is assumed in estimating rate of increase that this quantity is constant, over a period which is less than or equal to greatest reproductive age (depending on the life cycle assumed, see above). However greatest reproductive age is small compared to the time scale of Fig. 5, so it is a reasonable simplification to suppose that rate of increase is constant over this period.

It is usually the case that the optimal strategy depends at least to some extent on the rate of increase (Sibly & Calow, 1984, 1986a). However, very few genes would be able to modify the life cycle of their carriers in an appropriate way as they spread in the population. Therefore, there is a possibility that genes which were initially very successful might not produce the optimal life cycle when they were at or close to fixation. In this

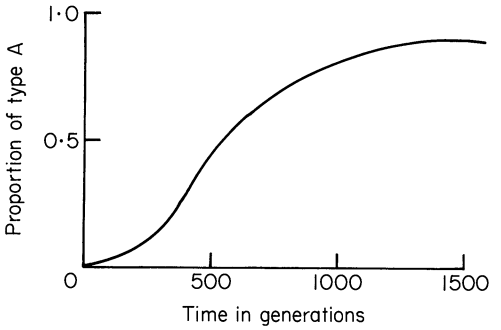

Fig. 5. The spread of a dominant allele A through the population. The *y*-axis represents the proportion of alleles, p_A, that are of type A, and this can be converted to the number of type A, N_A, by multiplying by 2 × population size. Note that if population size is fixed, = K, then the fitness of allele A (as defined in the text) can be estimated by the per capita rate of increase of p_A, i.e. $\frac{1}{pA}\frac{dp_A}{dt}$. This is because $F = \frac{1}{N_A}\frac{dN_A}{dt}$, and since $N_A = 2K\,p_A$, and $\frac{dN_A}{dt} = 2K\frac{dp_A}{dt}$, it follows that $\frac{1}{N_A}\frac{dN_A}{dt} = \frac{1}{2Kp_A}\frac{2Kdp_A}{dt} = \frac{1}{p_A}\frac{dp_A}{dt}$.

situation they would still be vulnerable to invasion by other alleles. The only genes not vulnerable to invasion are those which, at fixation, have a higher rate of increase than their potential competitors. In other words their competitors, if they ever did occur in the population, would decline (i.e. have a negative rate of increase), whereas the optimal gene, being at or close to fixation, would have a rate of increase of approximately zero. The optimal gene would, presumably, have been able to enter the population since it could spread, if only very slowly, against near competitors when the rates of increase of both were close to zero.

Comparisons between approaches

Lifetime reproductive success

It is proved in Appendix 1 that strategies maximize F if and only if they maximize lifetime reproductive success, in fixed-size populations (i.e. with $F = 0$ for the optimal strategy). However since optimal strategies usually depend on F (Sibly & Calow, 1984, 1986a), maximizing lifetime reproductive success will not in general be the same as maximizing F. Differences would occur, for example, if a population spent most of its time growing (positive F) but occasionally experienced selectively-neutral crashes (cf. '*r*-selection').

Reproductive value

Maximizing F is in general not the same as maximizing reproductive value at each age. The equivalences have been thoroughly treated in a discrete age-class model by Caswell (1980), using some general and deep theorems from matrix algebra, and Caswell's conclusions have been checked by Ricklefs (1981), using simpler techniques. This analysis shows that the strategy (i.e. birth-death schedule) that maximizes F will also maximize reproductive value at each age only for a restricted set of trade-off's.

However, a model devised by Schaffer (1974, 1979, 1981; cf. Yodzis, 1981) shows that, subject to the assumption outlined below, reproductive efforts that maximize fitness result in a value of reproductive effort at age x that maximizes reproductive value at age x, for all x. Reproductive effort is here defined as the proportion of the resources available to the animal at a given age that is allocated to reproduction at that age. The assumption is made that reproductive effort at one age can affect fecundities and mortality rates at later ages but not at earlier ages. While this approach is less widely applicable than the approach of maximizing fitness, it can be used to analyse an animal's options at age x between the various reproductive efforts open to it. If for each effort the resulting fecundity at age x, the probability of surviving to age $x + 1$, and the 'residual' reproductive value (at age $x + 1$) are known, then the effort that maximizes reproductive value can readily be identified using the formula:

reproductive value = (fecundity at age x) + (chance of surviving to age x + 1) × (residual reproductive value).

This has proved a popular way to discuss reproductive investments since its introduction by Williams (1966).

Any single fitness component

It is clear intuitively that any allele that increases fecundity will spread, other fitness components being held constant. Taking the most general model, the iteroparous life cycle, consider the effect, on the rate of spread (F), of increasing fecundity n_x at age t_x. Mathematically, we want to show that F is increased by increasing n_x, i.e. $\frac{\partial F}{\partial n_x}$ is positive. We use the partial derivative of F with respect to n_j to indicate that the other fitness components are kept constant.

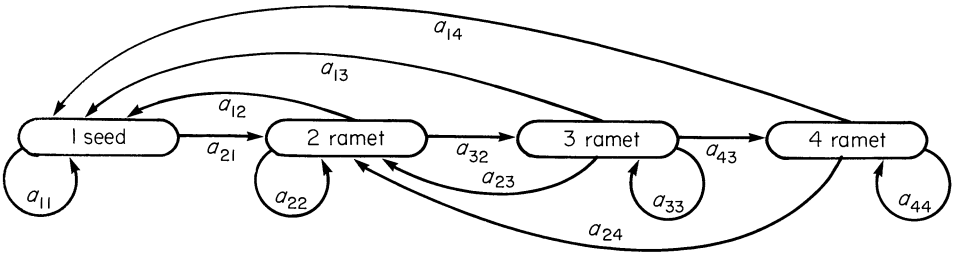

Fig. 6. A life-cycle graph for a plant. Ramets are classified into seeds and three size categories in this example (more can be included if necessary). The transition frequencies a_{ij} between stages are explained further in the text. After Caswell (1985).

Differentiating equation 7 with respect to n_x we obtain

$$\frac{\partial F}{\partial n_x} = \frac{l_x \, e^{-Ft_x}}{\sum_i n_i \, l_i t_i \, e^{-Ft_i}}$$

This is necessarily > 0 because each variable in the right hand side is > 0.

In the same way it can be shown that any allele that reduces mortality rate at any age will spread, other fitness components being held constant (Appendix 2). It is not quite so easy, though, to show that any allele that reduces age at breeding will spread, other fitness components (mortality rates and fecundities) being held constant. However, Sibly & Calow (1986b) have argued strongly that this is probably always true in a constant environment.

In this section we have seen that fitness is increased by increasing fecundity, decreasing mortality rate, or, according to Sibly & Calow (1986b), by breeding earlier, other fitness components being held constant. Specifically, F is increased by increasing n_x, decreasing μ_x, or decreasing i_x, for any value of x, provided that all other values of $\{n_i, \mu_i, t_i\}_{i=1}^{\Omega}$ are held constant. It follows that fitness is maximized by maximizing fecundity, minimizing mortality rate, or breeding as early as possible, other things being equal.

Fitness of plants

Although the above approach seems adequate for animals, some plants have altogether more complex life cycles, because they have alternative modes of reproduction (vegetative or gametic with or without sex). Until recently the only way to approach this problem has been to define a genet as the individual or clone derived from a single zygote (Harper, 1977). The size range of individual genets may therefore extend from a tiny seedling to a clone extending in fragments over a kilometre. The life cycle of a genet can then be defined as its birth/death schedule and the fitness of a gene coding for a particular genet life cycle can be derived as before. Births here refer to the production of offspring genets, i.e. zygotes. The problem with this approach is that in some species there is enormous variability in the life cycles of genets of the same genotype, especially if the genets are clones.

A possible solution is to consider the modular units, of which clones are composed, as the units subject to selection. These are called 'ramets' (Harper, 1977). Examples are leaves, tillers, rhizomes, stolons, rooting shoots, etc. and what it is useful to recognize as a ramet will vary between species. Since ramets are often involved in their own birth/death processes, the reasons for the variability in genet life cycle are evident.

Caswell (1985) has identified a powerful set of analytical tools designed to deal with this sort of complexity. His approach is to choose life-cycle stages appropriate for the study organism, and these might be seed and ramet, and/or the ramets might be classified according to size. An example, classified into seeds and three sizes of ramets, is shown in Fig. 6. Working in discrete time intervals there is then a transition frequency (i.e. a probability, a_{ij}) that seeds or ramets at stage j at time t will turn into ramets at stage i at time $t + 1$. Similarly let a_{1j} be the frequency with which ramets at stage j produce seeds. The transition matrix (a_{ij}) can then be analysed using the methods of matrix algebra, which apply to the stage-specific just as to the age-specific case (Caswell, 1985). In particular the largest eigen-value gives the population's asymptotic growth rate. Although Caswell does not say so, it is easy to use his formulation to derive the rate of increase of a gene, using the methods described above. Specifically, it has to be assumed that at each life-cycle

stage all carriers of the gene have the same transition frequencies. As before changes have to be made to the transition frequency to seeds. Male as well as female offspring must be counted but the resulting frequency must be halved because the chance of a particular allele in the ramet getting through meiosis into the seed is ½. All the other frequencies are unaffected.

This approach gives the rate of increase of a particular allele because the transition frequencies between stages give the probabilities that copies of that allele are transferred between stages. The largest eigen-value of the transition matrix gives the growth rate of the gene, and this, or its natural logarithm, provides a suitable measure of fitness for the complex life cycles of plants.

Conclusions

I have argued in favour of rate of increase of an allele as the preferred measure of fitness in life-cycle studies of what happens in a specified environment and population. For four simple life cycles, I have shown how this measure of fitness is derived. These derivations are subject to the assumption that all carriers of an allele have the same life cycle, but computer simulations, which changed the mode of inheritance, or allowed the sexes different life cycles or perturbed life-cycle components stochastically, suggest that predictions about optimal strategies are robust if made on the basis of this assumption. The approach can also be applied to the complex life cycles of plants, using methods developed by Caswell (1985). Fitness is maximized by maximizing fecundity, minimizing mortality rate, or breeding as early as possible, at each breeding occasion, provided, in each case, the other fitness components are held constant. Maximizing lifetime reproductive success is equivalent to maximizing F in fixed-size populations with $F = 0$ for the optimal strategy. Maximizing F is equivalent to choosing reproductive effort at each age to maximize reproductive value at that age, if it can be assumed that reproductive effort can affect later fecundities and survivorships but cannot be traded-off against earlier fecundities and survivorships.

Acknowledgments

I am indebted to Richard Law for suggesting the theorem in Appendix 1, and to Hans Metz for telling me it was true, at the Founding Meeting of the European Society for Evolutionary Biology, Basel, 26–30 August 1987.

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Appendix 1

Proof that a strategy maximizes fitness, F, *if and only if it maximizes lifetime reproductive success (LRS), in a fixed-size population such that* F = *0 for the optimal strategy*

F is defined as in equation 7 and LRS = $\frac{1}{2}\sum_{i=1}^{\Omega} l_i n_i$.

When a gene has reached fixation in a fixed-size population, $F = 0$, and then LRS = 1.

Consider two genes, A and B, with fitnesses F_A and F_B, and lifetime reproductive successes LRS_A and LRS_B. Suppose $F_A = 0$ and $LRS_A = 1$. F_B is given by

$$1 = \tfrac{1}{2}\sum_i e^{-F_B t_{Bi}} l_{Bi} n_{Bi} \qquad \text{Equation A1}$$

$$\text{and } LRS_B = \tfrac{1}{2}\sum_i l_{Bi} n_{Bi} \qquad \text{Equation A2}$$

I prove first that $F_B < F_A \Rightarrow LRS_B < LRS_A$. Since $F_A = 0$ and $LRS_A = 1$, this is equivalent to proving that $F_B < 0 \Rightarrow LRS_B < 1$. Since $t > 0$, $F_B < 0 \Rightarrow e^{-F_B t_{Bi}} > 1$ for all $t_{Bi} > 0$, $\Rightarrow e^{-F_B t_{Bi}} l_{Bi} n_{Bi} > l_{Bi} n_{Bi}$ for all t, since l_{Bi} and $n_{Bi} > 0$, $\Rightarrow \frac{1}{2}\sum_i e^{-F_B t_{Bi}} l_{Bi} n_{Bi} > \frac{1}{2}\sum_i l_{Bi} n_{Bi} \Rightarrow 1 > LRS_B$ by equations A1 and A2.

I now prove by contradiction that $LRS_B < LRS_A \Rightarrow F_B < F_A$, or equivalently that $LRS_B < 1 \Rightarrow F_B < 0$. Thus suppose that $LRS_B < 1$ and $F_B \geqslant 0$. $F_B \geqslant 0 \Rightarrow e^{-F_B} \leqslant 1 \Rightarrow e^{-F_B t_{Bi}} \leqslant 1$ for all $t_{Bi} > 0$, $\Rightarrow \frac{1}{2}\sum_i e^{-F_B t_{Bi}} l_{Bi} n_{Bi} \leqslant \frac{1}{2}\sum_i l_{Bi} n_{Bi} \Rightarrow 1 \leqslant LRS_B$, contradiction.

Appendix 2

Proof that fitness is increased by reducing a mortality rate

For this analysis it is attractive to describe the life cyle by independent variables (see p.130). Such a description can be obtained for the general iteroparous case as follows. Let $\tau_1 = t_1, \tau_2 = t_2 - t_1, \tau_3 = t_3 - t_2$, and so on, so that τ_i refers to the period between the (i–1)th and the ith breeding. Let μ_i be the mortality rate during this period

$$\text{Thus } t_i = \sum_{j=1}^{i} \tau_j \qquad \text{Equation A3}$$

$$\text{And } l_i = e^{-\mu_1\tau_1} \times e^{-\mu_2\tau_2} \times \ldots \times e^{-\mu_i\tau_i} = e^{-\sum_{j=1}^{i}\mu_j\tau_j} \qquad \text{Equation A4}$$

$$\text{Equation 7 now becomes } 1 = \tfrac{1}{2}\sum_{i=1}^{\Omega} e^{-\sum_{j=1}^{i}(F+\mu_j)\tau_j} n_i \qquad \text{Equation A5}$$

Taking partial derivatives with respect to μ_x, holding τ_i and n_i constant for all i, and holding μ_i constant for $i \neq x$, we obtain

$$0 = -\tfrac{1}{2}\sum_{i=1}^{\Omega} e^{-\sum_{j=1}^{i}(F+\mu_j)\tau_j}\left(\sum_{j=1}^{i}\frac{\partial F}{\partial \mu_x}\tau_j\right)n_i - \tfrac{1}{2}\sum_{i=x}^{\Omega} e^{-\sum_{j=1}^{i}(F+\mu_j)\tau_j}\tau_x n_i \qquad \text{Equation A6}$$

Rearranging,

$$\frac{\partial F}{\partial \mu_x} = -\frac{\sum_{i=x}^{\Omega} e^{-Ft_i} l_i \tau_x n_i}{\sum_{i=1}^{\Omega} e^{-Ft_i} l_i t_i n_i} \qquad \text{Equation A7}$$

Since e^{-Ft_i}, l_i, τ_i and n_i are all necessarily > 0 for $i = 1 \ldots \Omega$, it follows that $\frac{\partial F}{\partial \mu_x} < 0$ for all $x = 1 \ldots \Omega$.

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