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Competition and Body Size

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If being larger than competing conspecifics is important for fitness, then an unstable escalation of body size may result. In asexual populations, a cycling of sizes can occur but for sexual diploids, an irreversible size increase is more likely. Several factors can produce a stable distribution of sizes, but a single body size or even a narrow range of sizes cannot be stable. For example, enough environmental variance can produce stability without any genetic variability in the population. Or, with no environmental variance, a high cost of fighting between similar sizes or, for diploids, an increasing mortality with size may lead to a stable distribution of sizes. A game theory model is used to investigate the existence and form of a stable distribution of body sizes in a population. © 1986 Academic Press, Inc.

1. INTRODUCTION

The classical analysis of evolution in age-structured populations (Charlesworth, 1980) assumes that age-specific rates of mortality and fecundity are independent of the constitution of the population. There are many situations in which this will not be true. In species in which males fight for females, the fitness of a male will depend on his size relative to others. If access to resources depends on status in a hierarchy, it is again relative and not absolute size and strength that matter. Trees are tall because they must compete for light with other trees: a tree growing by itself would not need a trunk.

In such situations we would expect an escalation of sizes, held back by the cost of achieving a large size. But we would not expect one optimal size to result, because if the members of a population were, as adults, all the

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same size, then a mutant which was only a bit larger would suffer only a slight additional cost, but, as the largest member of the population, would gain substantially in fitness. Hence we would expect a stable population, if it exists, to have a distribution of sizes. This paper uses evolutionary game theory to find the form of such a stable distribution and conditions for it to exist.

Haigh and Rose (1980), following a suggestion by Parker (1979), tackled this problem by analysing a modified form of the "war of attrition" game. They found that such models do not readily lead to stable size distributions. However, their model can be criticized on the grounds that it treats total fitness as the sum of a component concerned with probability of survival to a given size and one concerned with success given that the size is actually reached. It seems more logical to treat total fitness as a product of these two components, as we do here. Since we wished to investigate the stabilising effects of such features as cost of competition, increasing force of mortality, environmental variance, and diploid inheritance, it seemed best to reformulate the basic model, rather than pursue further the war of attrition approach.

The evolution of plant life histories, allowing for competition, was treated in an important paper by Mirmirani and Oster (1978), although their criterion of evolutionary stability is open to question (Maynard Smith, 1978). Their model differed from the one analysed here in that it ruled out indefinite increase in size because it was concerned with the optimal allocation of resources between growth and reproduction in a single year. Their conclusions concerning the importance and environmental variance will be compared to ours in the Discussion.

Perhaps the closest approach to our model is one developed by Parker (1983). He assumes that there is significant environmental variance in size (or armament, as he calls it). Instead, we examine models in which heredity (haploid or diploid) determines size. Then we consider the effects of adding environmental variance. Further differences are noted in Section 3 and the Discussion.

A model developed independently by Charlesworth (1983) overlaps our work, and is also considered in the Discussion.

The model discussed here was first formulated by Maynard Smith (1982) but was analysed only numerically.

2. THE BASIC MODEL

We begin by considering an asexual population in which size is genetically determined. Environmental variance of size and diploid inheritance will be introduced later.
For simplicity, we suppose that individuals first grow without breeding, and then breed without further growth: this corresponds closely to the situation in male red deer, which suggested the present model. Suppose first that there are only two size classes, $S$ and $L$, of sizes $m_1$ and $m_2$, $m_1 < m_2$. The probability of surviving to size $m$ is $s(m)$, which is a decreasing function of $m$. For an $S$ in a population of $L$'s, breeding success is $V = a$; for an $L$ in a population of $S$'s it is $V = (a + b)$. For an $L$ among $L$'s, or an $S$ among $S$'s, breeding success is $V = (a + (b/2) - c)$; this assumes that half of all contests are won against an opponent of equal size, but that there is a cost $c$ of fighting. We assume that costly fights occur only between individuals of very similar sizes. The payoff matrix of fitnesses is given in Table I.

There are four possibilities for the dynamics of such a game (Maynard Smith, 1982). If we take $a = 1$ and fix a value of $s(m_2)/s(m_1) = r$ = relative probability of survival of large individuals, then we can show the four possible dynamics as they occur in the $(b, c)$-plane. See Fig. 1. Note in particular the role that the cost-of-fighting parameter, $c$, plays: if $c$ is small, a polymorphism of sizes (region 2 in Figs. 1a and b) is impossible, and as $c$ increases, a polymorphism must eventually arise.

We turn now to the more interesting and realistic case with more than two size classes. Suppose we have sizes $m_1 < m_2 < \cdots < m_i < \cdots < m_n$, and $s(m_{i+1})/s(m_i) = r$ is independent of $i$. Simulations (Fig. 2) show that there can be a cycling of sizes. It is easy to see why this should be so. Suppose we choose $b$ and $c$ corresponding to case 1 (Fig. 1a). Then if we start with a population of size $m_1$, it can be invaded by size $m_2$, and that in turn can be invaded by size $m_3$, and so on. If $c$ is small, smaller sizes are quickly wiped out by invading larger sizes, and the size distribution is a sharp narrow peak moving towards larger sizes. But at some point, $s(m_i)$ is so small that a mutant $m_1$ can invade a population of size $m_i$. At that point, $m_1$ takes over and the cycle starts again. On the other hand, if we increase $c$ (but

<table>
<thead>
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<tbody>
<tr>
<td>$S$</td>
<td>$s(m_1)[a + (b/2) - c]$</td>
</tr>
<tr>
<td>$L$</td>
<td>$s(m_2)[a + b]$</td>
</tr>
</tbody>
</table>

*Note.* The entries are the fitnesses of individuals of the sizes listed on the left, in an ambient population of individuals on the sizes listed on top.
remain in case 1, Fig. 1) then the cost of fighting between equal sizes damps down the sharp peak, and there results a stable polymorphism involving many sizes.

In the above simulation it was assumed that the generation time of all size classes was the same. This is unrealistic because larger individuals would take longer to mature; it is for this reason that we assume that \( s(m) \) is a decreasing function of \( m \). However, the conclusions of the last paragraph do not depend on this assumption. Simulations in which the generation time of size class \( m_i \) was proportional to \( m_i \) gave qualitatively similar results.

In order to get an analytic expression for the stable size distribution observed in our simulations, consider a model with a continuum of possible sizes \( m, 0 \leq m < \infty \). To specify what it means for two individuals

Fig. 1. Dynamics for two asexual genotypes, large \( L \) and small \( S \). The four possible flows on \( 0 \leq p \leq 1 \) (where \( p \) is the frequency of \( L \)) are shown in (a). Open circles are unstable equilibria and solid circles are stable ones. Thus case 1 represents \( L \) going to fixation. The occurrence of these four cases, as parameters \( b \) and \( c \) are varied (with \( a = 1 \)), is shown in (b) for \( r = s(m_2)/s(m_1) = 0.7 \) and in (c) for \( r = 0.4 \).
Fig. 2. Size oscillations assuming a large number of asexual types when cost of fighting, \( c \), is small. The size distribution, \( p(m) \), changes from (a) to (b) to (c) to (a) again, and so on. When \( c \) is close to zero, the travelling peak is very sharp and narrow.

To have the same size, let \( \delta \) be a small number such that an individual of size \( m \) in an encounter with an individual of size \( m' \) will get

\[
(a + b) \quad \text{if} \quad m' < m - \delta,
\]
or

\[
(a + b/2 - c) \quad \text{if} \quad m - \delta < m' < m + \delta,
\]
or

\[
(a) \quad \text{if} \quad m + \delta < m'.
\]

(For the same \( \delta \) to work for all \( m \), we should assume that \( m \) is log size so that additive increments in \( m \) represent multiplicative increments in size. Size is normalized so unit size, or log size \( m = 0 \), simply means the smallest size at which breeding is possible.) Let \( p(m) \) denote the distribution of possible sizes in the adult population. Then

\[
p(m) > 0 \quad \text{and} \quad \int_0^\infty p(y) \, dy = 1.
\]
The fitness of size $m$ in a population $p(m)$ is

$$W(m) = s(m)[a + bz - 2\delta cp(m)]$$

(2)

where $z$ is the fraction of the population smaller than $m$:

$$z = \int_0^m p(y) \, dy.$$  

(3)

For the distribution $p(m)$ to be stationary it must have $W(m)$ constant over the support of $p(m)$. Stability imposes further conditions. In particular there will be a maximum size $R$ because an animal larger than $R$ will always be less fit than one of minimal size $m = 0$. Furthermore, $p(R)$ will be zero because otherwise individuals of size $R + \delta$ would have an advantage.

By solving the equation $W(m) = K$ on $0 \leq m \leq R$, and then finding $K$ and $R$ using

$$p(R) = 0 \quad \text{and} \quad \int_0^R p(y) \, dy = 1$$

(4)

we can find a formula for a stationary distribution. For example, consider the case of “constant mortality.” By this we mean that if the size is increased by a factor $1 + \delta$, $\delta$ small (so log size $m$ becomes $m + \delta$), then $s(m)$ is decreased by a factor $1 - k\delta$, independent of $m$. This implies that $s(m) = \exp(-km)$. Now letting $\beta = (b/2\delta c k)$ and assuming that $c > 0$ and $\beta \neq 1$ we find

$$p(m) = (ak/b) \exp(km) \left[ 1 - \frac{1 - \beta \exp((\beta - 1) km)}{1 - \beta \exp((\beta - 1) kR)} \right].$$

(5)

If we can ignore the term $2\delta cp(0)$ in $W(0) = s(0)[a + 2\delta cp(0)]$ then $R$ is determined by the condition $s(0) a = s(R)(a + b)$; for if $R$ is greater than this, its fitness is necessarily lower than that of $m = 0$. Thus when $2\delta cp(0)$ is small, an approximate $R$ is found by solving $\exp(kR) = 1 + (b/a)$. More generally the exact equation for $R$ is

$$\exp(kR) \frac{[1 - \beta]}{[\exp((\beta - 1) kR) - \beta]} = 1 + (b/a).$$

(6)

This equation has a unique root $R$ which can be found numerically.

Figure 3 shows such a stable continuous distribution of sizes. Simulation of a discrete population with 50 size classes gave a distribution which converged fairly quickly to a stable limit in close agreement with the analytic distribution given by (5). We must emphasize, however, that a stable limit
Fig. 3. Stationary adult size distribution, $p(m)$, assuming a large number of asexual types. A stable equilibrium distribution corresponding to $a = 1$, $b = 4$, and $c = 0.5$ is shown in (a). The unstable stationary distribution shown in (b), which corresponds to $a = 1$, $b = 4$, and $c = 0$, does not appear in simulations. Instead, the population oscillates as in Fig. 2.

is reached only if $\delta c$ is large enough. Figure 3 also shows an unstable stationary distribution corresponding to $\delta c = 0$. When $\delta c$ is small, the population will cycle as in Fig. 2.

So far we assumed a linear function $V(z) = a + bz$ in (2) to express breeding success (ignoring cost of fighting). Simulations of some non-linear success functions are shown in Fig. 4. In both examples the cost of fighting must be above a certain threshold, not much changed from the linear $V(z)$ case, in order to get a stable distribution.

3. ENVIRONMENTAL Variance OF SIZE

Still with asexual inheritance, we now assume that a given genotype produces a normal distribution of sizes. An individual of a particular size has fitness given by (2); these individual fitnesses must be averaged to give the fitness of a genotype.

These assumptions are similar to those of Parker (1983) (although he uses three types of non-normal distributions of size), and our conclusions largely agree with his. However, we give more detail about the dynamics of the instabilities, and the transition to a stable solution as the amount of environmental variance is increased.
Simulations were run with varying values of the environmental variance $\sigma^2$. Three patterns of behaviour were observed:

(i) When $\sigma^2$ was small, we obtained results similar to those in the last section.

(ii) As $\sigma^2$ was increased, the stable distribution (Fig. 3) became unstable and changed to a bell-shaped distribution that moved towards higher and higher mean sizes, until finally small sizes could invade the population. There appeared to be an oscillation between the stable distribution of Fig. 3 and a normal distribution whose mean size was too large to restrict invasion by small sizes.

(iii) As $\sigma^2$ increased further, the normal distribution broadened, and its mean moved to a lower value, until it became the only stable distribution. At this point the resulting normal distribution of sizes was stable, even with zero cost of fighting, $c = 0$. Recall that $c > c_0 > 0$ was required for stability when $\sigma^2 = 0$. Further, the distribution of sizes observed was being generated by a narrow range of genotypes: that is, the variance in size was nearly all environmental, and not genetic.
To explain the results of these simulations with an analytic model, consider an adult population consisting of a single genotype of mean size $m^*$ and variance $\sigma^2$. We assume $c = 0$. We then compute the fitness of a rare mutant genotype of size $m$ and variance $\sigma^2$. For simplicity, we assume that survival probabilities are not averaged; that is, genotype $m$ has survival probability $s(m)$ independent of the size actually achieved when environmental variance is allowed for. Hence

$$W(m) = s(m)(a + bz)$$  \hspace{1cm} (7)

where $z$ is the probability that a random member of the mutant population $m$ is larger than a random member of the main population $m^*$. Let $M$ and $M^*$ be random variables giving the sizes of a random member of the mutant and main populations, respectively. Then $z = \text{Prob}(M - M^* > 0)$. Now $M - M^*$ is normally distributed with mean $m - m^*$ and variance $2\sigma^2$. Hence

$$z = \left[ 1/(2\sqrt{\pi} \sigma) \right] \int_{-\infty}^{\infty} \exp\left\{ -[t - (m - m^*)]^2/4\sigma^2 \right\} dt$$

$$= \left[ 1/(2\sqrt{\pi} \sigma) \right] \int_{-\infty}^{m^*} \exp\left\{ -[t - m^*]^2/4\sigma^2 \right\} dt.$$  \hspace{1cm} (8)

If $m^*$ is to be uninvadable by any $m$, we must select $m^*$ so that $W(m)$ has its maximum value at $m = m^*$. Thus we want, in particular, $0 = (dW/dm)$ at $m = m^*$. Using (7) and (8) we can compute $dW/dm$ and then set $m = m^*$ to get

$$dW/dm = s'(m^*)(a + b/2) + s(m^*) b/(2\sqrt{\pi} \sigma).$$  \hspace{1cm} (9)

Setting this equal to zero gives a condition on $m^*$:

$$s'(m^*)/s(m^*) = -b/(2\sqrt{\pi} \sigma(a + b/2)).$$  \hspace{1cm} (10)

The case of a constant force of mortality, $s(m) = \exp(-km)$, is a borderline case that gives no solution, because the left side of (10) is identically equal to $-k$. But if the force of mortality is increasing (or decreasing), then (10) has a unique solution $m^*$ for given reasonable values of the parameters $a, b, \sigma$.

For example, if $s(m) = \exp(-km^2)$, then

$$m^* = b/[4 \sqrt{\pi} \sigma(a + (b/2))]$$  \hspace{1cm} (11)

and we can check that this $m^*$ gives a local maximum of the fitness function $W(m)$. But it is not necessarily a global maximum. If $\sigma^2$ is small, then $m^*$ is large and the population can be invaded by mutants of small
size. When $\sigma$ is above a certain threshold, $m^*$ does give the absolute maximum of $W(m)$. Then the genetically homogeneous population with mean size $m^*$ and size variance determined entirely by the environment is stable. Our simulations suggest that this stable equilibrium is a global attractor.

Thus if there is an increasing force of mortality, and sufficient environmental variance, there is a stable genetically homogeneous population. However, if there is a decreasing force of mortality, the stationary $m^*$ given by (10) is unstable. A population with mean size larger than $m^*$ would increase in size (until invaded by $m=0$), and a population of mean size smaller than $m^*$ would decrease in mean size.

4. DIPLOID SEXUAL INHERITANCE

We now assume a random mating population of sexual diplolds. Consider first two alleles, $L$ and $S$, at a locus. Assume that $SS$, $SL$, and $LL$ have sizes $m_1 < m_2 < m_3$ respectively and that

$$s(m_2)/s(m_1) = s(m_3)/s(m_2) = r < 1.$$  (12)

This would be the case if, for example, the effects of genes on size were additive and there were a constant force of mortality. The fitness matrix is given in Table II.

The main changes from the haploid case is as follows. It is possible to choose parameter values such that there is escalation from $SS$ to $SL$, and thence to $LL$, and yet for $SS$ to be more fit than $LL$ among $LL$'s: there is no way for the $S$ allele to reinvade, because invasion of $S$ depends on the fitness of $SL$, and not of the rare $SS$, among $LL$'s. The tendency of sexual diploid populations to escalate irreversibility in size is met in a more extreme form when inheritance is polygenic.

**TABLE II**

Fitness Matrix for a Diploid Population

<table>
<thead>
<tr>
<th>Individual</th>
<th>Population</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>$SS$</td>
</tr>
<tr>
<td>$SS$</td>
<td>$s(m_1)[a + (b/2) - c]$</td>
</tr>
<tr>
<td>$SL$</td>
<td>$s(m_2)[a + b]$</td>
</tr>
<tr>
<td>$LL$</td>
<td>$s(m_3)[a + b]$</td>
</tr>
</tbody>
</table>

*Note.* The entries are the fitnesses of individuals with genotypes listed on the left, in an ambient population of individuals with genotypes listed on top.
The case in which size is polygenically determined has been treated by simulating an infinite random-mating population with the following assumptions:

(i) Size is determined by genes at 10 unlinked loci, with two alleles $L$ and $S$ at each locus. The presence of each $L$ allele adds one unit to size, so that the total range of size is from 0 to 20.

(ii) Generations are separate, with each size class having the same generation time.

(iii) A linear function $V(z) = a + bz$ determines reproductive success.

(iv) Linkage equilibrium exists; this is incorrect, but is unlikely to matter for unlinked genes.

Consider first the case with a constant force of mortality and no environmental variance. Regardless of the cost of fighting, size increases until the $L$ allele is fixed at all loci. There is no cycling, as there would be in the corresponding asexual case (Fig. 2), because when the population mean phenotype size rises to a level at which it is susceptible to invasion by individuals of size $m=0$, there is no way in which such individuals can arise (or, if they arise, breed true). If the model were modified to allow "macromutations," then it would oscillate. However, with a fixed set of alleles, each of small effect, the population reaches its maximum size, even though the mean fitness falls very low.

If we assume an increasing force of mortality, but still no environmental variance, then the size increase halts at a stable distribution of sizes. However, this stable distribution is not maintained by competition from smaller sizes, as in the asexual case, but rather by the large increase in mortality associated with being just one size larger. Thus the stability is caused by the discreteness of our simulations; if the same size range were more finely controlled by more loci, then sizes would increase further. Size increases until the next size is too large relative to the preceding one.

It is more realistic to incorporate the effects of environmental variance. This has been done in simulations in which there was no cost of fighting. The results were exactly as expected, by comparison with the asexual case. If there is a constant force of mortality, the mean size increases until the $L$ alleles are fixed at all loci. If there is an increasing force of mortality, a genetically homogeneous population, with the $L$ allele fixed at some loci and the $S$ allele at others, is established, with phenotypic variance being entirely environmental.
5. Discussion

Our conclusions can be summarized as follows:

(i) The dynamics of competitive life histories leads very readily to instability. This is because no matter what the mean size of the population, it always pays to be a bit larger. In the asexual model this leads to cyclical changes. The mean size increases until a stage is reached at which very small individuals can invade the population. The large individuals are then very rapidly eliminated, and a new cycle is initiated as the now small-sized population again increases in size.

(ii) If an individual pays a high cost for competing with others closely similar in size to itself, even if it wins, this can give rise to a stable, genetically polymorphic population.

(iii) The population can be stabilised if there is an increasing force of mortality, and if the environmental variance in size is above a certain threshold. Acting together, these two factors give rise to a stable, genetically monomorphic, but phenotypically variable, population.

(iv) Diploid sexual inheritance is not a stabilizing factor. Instead, it may result in the population increasing indefinitely in size, instead of cycling. This is because, when the population has reached a size at which it could be invaded by a very small individual, there may be no way in which such individuals can arise and reproduce their kind. That is, a rare individual who is homozygous for small genes at most size loci will have offspring that are much larger, because of random mating. These offspring lose most of the benefit of small absolute size. Yet they gain no benefit from relative size, being smaller than everyone else. (However, a "macromutation" could invade the population at this stage, so the difference between asexual and sexual populations is perhaps more apparent than real.) Environmental variance, increasing force of mortality, and cost of competition have the same stabilising effects on sexual as on asexual populations.

It is apparent from Fig. 4 that the phenotypic variability of the population will depend on the form of the function relating breeding success, \( V(z) \), to relative size, \( z \). This agrees with the conclusion of Maynard Smith (1982), who suggested that case (b) corresponds to the case in which large individuals have high fitness, but an alternative strategy is available to the weaker ones, whereas case (a) corresponds to that in which the weakest members of a population (e.g., the lowest in a hierarchy) die, but the rest are equally able to survive.

Maynard Smith (1982) also concluded that, if \( V(z) \) has a steep slope against \( z \) over the whole range \( 0 < z < 1 \), then size would increase until stopped by senescence. This agrees with the present finding that an increas-
ing force of mortality is stabilising. The present analysis shows that senescence must be reinforced by environmental size variance if indefinite size increase is to be avoided, whereas Maynard Smith (1982) did not invoke environmental variance. The difference arises because the present model assumes either continuous size distributions or a large number of discrete size classes.

We agree with Parker (1983) that with sufficient environmental effect on size, there can be "pure ESS" or a stable genetically monomorphic population. But we have shown that with little or no environmental effect, a stable distribution of sizes may still arise. We have also considered the differences between haploid and diploid inheritance.

The model developed by Charlesworth (1984) overlaps with our work. He considers a population subject to normalising selection, and also to frequency-dependent selection for greater size. He concludes that the population will always reach a stable size distribution, larger than the optimal. At first sight this seems to contradict our finding of a continuous size increase. However, the discrepancy is explained by two features of his model:

(i) His normalising selection is Gaussian, so, as the population mean moves away from its optimum, it is equivalent to directional selection with an "increasing force of mortality" in our model.

(ii) He has a different way of calculating the effect of relative size on breeding success. Whereas we suppose that an animal wins against all others smaller than itself, he supposes that its probability of winning a fight is an increasing function of its size advantage over its opponent: that is, we assume a step function where he assumes a ramp.

His assumption (ii) is formally equivalent to introducing some environmental variance into breeding success, so his conclusion agrees with our finding that a combination of environmental variance and increasing force of mortality gives rise to a stable distribution. To check this, we have simulated his assumptions. We find that, with a constant force of mortality, and with his assumption about breeding success, size increases indefinitely, but that if his breeding success assumption is combined with an increasing force of mortality, then, as he says, a stable age distribution is reached.

Since the present model leads so readily to instability, it is worth asking whether indefinite size increase, or continuous cycling, occurs in nature. One could argue that the stabilising factors—increasing force of mortality, cost of fighting with opponents similar in size to oneself, and environmental variance—are so plausible that extreme instability is unlikely to occur. This is reasonable, but carries with it the implication that species with frequency-dependent size competition are likely to start breeding only when
they are reaching senescence. However, it is worth remembering that the fossil record shows that some 90\% of mammalian lineages increase in size. Since, averaged over species, mammals have not continued to increase in size, extinction must have been commoner in large species, and speciation among smaller ones. Combined with the observation that sexual dimorphism tends to be greater in the larger species, this suggests that there has been long-continued selection for increased size in many lineages, concentrated on the male sex, and that this has reduced the chances of survival of the species concerned.

The prediction of cyclical changes in size is still harder to evaluate. Cycles could happen in sexual species only if "macromutations" are possible. It is perhaps more plausible that, when a species reaches an inefficiently large size, it should be replaced by other species. However, the point is of some general importance, because game theoretic models have suggested a number of processes which could result in cycles. In addition to the model discussed here, the "sex war" game (Dawkins, 1976) and the "rock–scissors–paper" game (phenotype B replaces A, C replaces B, and A replaces C) readily lead to cycles. We know of cases (e.g., bluegill sunfish, Gross and Charnov, 1980; solitary bees, Alcock et al., 1977) in which small and large males coexist. These examples are usually interpreted either as stable mixed strategies or as cases in which small individuals are small for non-genetic reasons and are making the best of a bad job. However, it would be rash to overlook the possibility that these cases may be stages in a cycle.

REFERENCES


