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Life History Evolution with Antagonistic Pleiotropy and Overlapping Generations

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Previous results found for selection with antagonistic pleiotropy and discrete generations are extended to cases with overlapping generations. In order to do so, protected polymorphism conditions are found for monoecious and dioecious populations when the intrinsic rate of increase, or Malthusian parameter, is not too large in magnitude. Under such conditions, it is shown that recessive deleterious gene effects foster the maintenance of allelic variants affecting life history. The significance of this result for experimental studies of the evolution of senescence is addressed.

1. INTRODUCTION

Sewall Wright has persistently emphasized the importance of pleiotropy in evolution (e.g., 1977). Caspari (1950), Falconer (1960, 1977), Wallace (e.g., 1959), and Williams (1957) also took up this theme, but it remained largely unnoticed. The last few years have seen a change in this state of affairs. A number of theoretical studies explicitly incorporating pleiotropic gene action have been published (Charlesworth, 1980; Lande, 1980; Templeton, 1980; Rose, 1982; Slatkin, 1982). More importantly perhaps, there have been experimental studies providing evidence for Wright's view (e.g., Simmons *et al.*, 1980; Rose and Charlesworth, 1981a, b). Thus Wright's view is finally receiving the attention it has long deserved.

One of the most interesting aspects of pleiotropy is that it can act to maintain additive genetic variation in fitness components if the multiple effects are antagonistic (Caspari, 1950; Wallace, 1959; Wright, 1977, p. 557; Falconer, 1977; Rose, 1982). That is, if one fitness component is enhanced while another is depressed, then the net effects on fitness can give rise to overdominance, in two-allele, single-locus systems, and its analogues, in more complex genetic systems. In particular, recessive deleterious gene action tends to foster protected polymorphism under a variety of conditions (Rose, 1982, 1983).

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All these results were derived for discrete-generation population genetics models. In view of the known potential importance of pleiotropy in populations with overlapping generations (Charlesworth, 1980; Templeton, 1980), it would be useful to ascertain whether or not comparable results apply to such populations. An analysis of this kind is given below. The lack of theory as wide-ranging as that for discrete generations cases necessarily limits the present analysis to the well-understood single diallelic locus case (Charlesworth, 1976, 1980).

2. Basic Protected Polymorphism Analysis

First, implicit protected polymorphism conditions will be derived, following Charlesworth (e.g., 1980). Consider a single diallelic autosomal locus in a dioecious, discrete-time, age-structured population. Let mating be at random with respect to genotype, but not with respect to age. Let the fertility schedule of a mated female depend only on her genotype, with an abundance of males. Given this assumption, fecundity schedules for males and females may be specified for each genotype. Let the sex ratio be fixed and independent of both age and genotype. Finally, let it be assumed that the population is infinite in size, without density-dependence affecting its life-history characters.

Turning to notation, define A_1 and A_2 as the alleles of interest, with associated ordered diploid genotypes A_iA_j, where A_i came from the individual's mother and A_j came from the father, with no reciprocal effects on life history. Let age be designated by x and time by t. Let $P_{ij}(x, t)$ and $P_{ij}^*(x, t)$ represent the probabilities of survival from age class x at time t to age class $x+1$ at time $t+1$ for A_iA_j females and males, respectively.

If we define $l(x, t)$ and $l^*(x, t)$ as the probability of survival from conception at time $t-x$ to age x at time t for females and males, respectively, then

$$l_{ij}(x, t) = \prod_{y=1}^{x} P_{ij}(x-y, t-y) \tag{1a}$$

$$l_{ij}^*(x, t) = \prod_{y=1}^{x} P_{ij}^*(x-y, t-y). \tag{1b}$$

Let the number of offspring produced in one time interval by A_iA_j females and males aged x at time t be given by $M_{ij}(x, t)$ and $M_{ij}^*(x, t)$, respectively. Let $N_{ij}(x, t)$ and $N_{ij}^*(x, t)$ be the number of A_iA_j females and males aged x

at time t, respectively. Then we must have the total number of zygotes produced at time t, say $B(t)$, as follows:

$$B(t) = \sum_{i,j,x} N_{ij}(x, t)\, M_{ij}(x, t) \tag{2a}$$

$$= \sum_{i,j,x} N^*_{ij}(x, t)\, M^*_{ij}(x, t), \tag{2b}$$

where there are three separate summations covering all age classes and genotypes. Let the frequencies of allele A_i among the maternal and paternal gametes at time t be given by $p_i(t)$ and $p_i^*(t)$, respectively. Evidently, we have

$$p_i(t) = \tfrac{1}{2} \sum_{j,x} [N_{ij}(x, t) + N_{ji}(x, t)]\, M_{ij}(x, t)/B_{(t)} \tag{3a}$$

and

$$p_i^*(t) = \tfrac{1}{2} \sum_{j,x} [N^*_{ij}(x, t) + N^*_{ji}(x, t)]\, M^*_{ij}(x, t)/B(t). \tag{3b}$$

Let $\Psi(x, y, t)$ be the frequency of females of age x mating with males of age y at time t. If we let the sex ratio be a, giving the proportion of females, then we can define sex-specific fecundity schedules as

$$m_{ij} = aM_{ij}(x, t); \qquad m^*_{ij}(x, t) = (1 - a)\, M^*_{ij}(x, t), \tag{4}$$

and net sex-specific reproductive schedules, taking mortality into account, as

$$k_{ij}(x, t) = l_{ij}(x, t)\, m_{ij}(x, t); \qquad k^*_{ij}(x, t) = l^*_{ij}(x, t)\, m^*_{ij}(x, t). \tag{5}$$

Finally, define

$$\bar{p}_i(x, y, t) = \tfrac{1}{2}[p_i(x, t) + p_i^*(y, t)] \tag{6}$$

and

$$\delta_i(x, y, t) = p_i(x, t) - \bar{p}_i(x, y, t)$$
$$= \bar{p}_i(x, y, t) - p_i^*(y, t).$$

With these assumptions and definitions, the genotypic frequencies may be obtained following Charlesworth and Charlesworth (1973). Taking those matings of females aged x and makes aged y, the frequency of

genotypes A_iA_j, say $p_{ij}(x, y, t)$, among the progeny of such matings is given by

$$\begin{aligned} p_{ii}(x, y, t) &= p_i(x, t)\, p_i^*(y, t) \\ &= p_i(x, t)\, \bar{p}_i(x, y, t) + p_i^*(y, t)\, \bar{p}_i(x, y, t) \\ &\quad - \bar{p}_i^2(x, y, t) - \delta_i^2(x, y, t) \\ &= \bar{p}_i^2(x, y, t) - \delta_i^2(x, y, t) \end{aligned} \tag{7a}$$

and, similarly,

$$\begin{aligned} &p_{ij}(x, y, t) + p_{ji}(x, y, t) \\ &\quad = p_i(x, t)\, p_j^*(y, t) + p_j(x, t)\, p_i^*(y, t) \\ &\quad = 2\bar{p}_i(x, y, t)\, \bar{p}_j(x, y, t) - 2\delta_i(x, y, t)\, \delta_j(x, y, t). \end{aligned} \tag{7b}$$

Since the variance of x, V_x, equals $\sum_x f(x)\, x^2 - (\sum_x f(x)\, x)^2$, where $f(x)$ is the frequency distribution of the x_i, we have

$$\sum_x f(x)\, x^2 = V_x + \left(\sum_x f(x)\, x\right)^2.$$

Thus, if $p_{ij}(t)$ is the frequency of A_iA_j zygotes at time t,

$$\bar{p}_i(t) = \sum_{x,y} \Psi(x, y, t)\, \bar{p}_i(x, y, t), \tag{8a}$$

and

$$\delta_i(t) = \sum_{x,y} \Psi(x, y, t)\, \delta_i(x, y, t), \tag{8b}$$

then

$$\begin{aligned} p_{ii}(t) &= \sum_{x,y} \Psi(x, y, t)[\bar{p}_i^2(x, y, t) - \delta_i^2(x, y, t)] \\ &= \bar{p}_i^2(t) - \delta_i^2(t) + V[\bar{p}_i(x, y, t)] \\ &\quad - V[\delta_i(x, y, t)] \end{aligned} \tag{9a}$$

and, similarly,

$$\begin{aligned} p_{ij}(t) + p_{ji}(t) &= 2 \sum_{x,y} \Psi(x, y, t)[\bar{p}_i(x, y, t)\, \bar{p}_j(x, y, t) \\ &\quad - \delta_i(x, y, t)\, \delta_j(x, y, t)] \\ &= 2\bar{p}_i(t)\, \bar{p}_j(t) - 2\delta_i(t)\, \delta_j(t) \end{aligned}$$

$$+2\,\mathrm{cov}[\bar{p}_i(x,y,t),\bar{p}_j(x,y,t)]$$
$$-2\,\mathrm{cov}[\delta_i(x,y,t),\delta_j(x,y,t)]. \tag{9b}$$

With just two alleles, Eq. (9b) becomes

$$\begin{aligned} p_{12}(t)+p_{21}(t) &= 2\bar{p}_1(t)\,\bar{p}_2(t)-2[\delta_1(t)][-\delta_1(t)] \\ &\quad +2\,\mathrm{cov}[\bar{p}_1(x,y,t),1-\bar{p}_1(x,y,t)] \\ &\quad -2\,\mathrm{cov}[\delta_1(x,y,t),-\delta_1(x,y,t)] \\ &= 2\bar{p}_1(t)\,\bar{p}_2(t)+2\delta_1^2(t) \\ &\quad -2V[\bar{p}_1(x,y,t)]+2V[\delta_1(x,y,t)]. \end{aligned} \tag{9c}$$

Note that Eqs (7) to (9) apply irrespective of the gene frequency magnitudes.

For the purpose of analysis of protected polymorphism, it suffices to consider the selection dynamics near the allelic fixation boundaties with $\bar{p}_1(t)=0$ or $\bar{p}_1(t)=1$. Say $\bar{p}_1(t)\approx 1$ and $\bar{p}_2(t)\ll 1$. Thus $O(\bar{p}_2)$ terms can be taken as small. Note that $\delta_i(x,y,t)=O(\bar{p}_2)$, $V[\bar{p}_i(x,y,t)]=O(\bar{p}_2^2)$, and $V[\delta_i(x,y,t)]=O(\bar{p}_2^2)$, for $i=1,2$. Thus we have

$$p_{11}(t)=\bar{p}_1^2(t)+O(\bar{p}_2^2) \tag{10a}$$

and

$$p_{12}(t)+p_{21}(t)=2\bar{p}_1(t)\,\bar{p}_2(t)+O(\bar{p}_2^2). \tag{10b}$$

Equations (10) give

$$N_{ij}(x,t)=aB(t-x)\{\bar{p}_i(t-x)\,\bar{p}_j(t-x)\,l_{ij}(x,t)+O(\bar{p}_2^2)\} \tag{11a}$$

and

$$N_{ij}^*(x,t)=(1-a)\,B(t-x)\{\bar{p}_i(t-x)\,\bar{p}_j(t-x)\,l_{ij}^*(x,t)+O(\bar{p}_2^2)\}. \tag{11b}$$

Using Eqs. (11) in Eqs. (3), we have

$$\begin{aligned} B(t)\,\bar{p}_i(t) &= \tfrac{1}{4}\sum_{j,x}\{[N_{ij}(x,t)+N_{ji}(x,t)]\,M_{ij}(x,t) \\ &\quad +[N_{ij}^*(x,t)+N_{ji}^*(x,t)]\,M_{ij}^*(x,t)\} \\ &= \tfrac{1}{2}\sum_{j,x}B(t-x)\{[\bar{p}_i(t-x)\,\dot{\bar{p}}_j(t-x)][k_{ij}(x,t)+k_{ij}^*(x,t)]+O(\bar{p}_2^2)\}. \end{aligned} \tag{12}$$

Given sufficient time for achievement of a stable age distribution among A_1A_1 genotypes, we may approximate $B(t-x)$ as

$$B(t-x) = e^{-r_{11}x}B(t) + O(\bar{p}_2), \tag{13}$$

(Charlesworth, 1980, p. 169, App. 2). Using (13) in (12) gives

$$\bar{p}_2(t) = \tfrac{1}{2}\sum_x e^{-r_{11}x}\bar{p}_2(t-x)[k_{12}(x,t) + k^*_{12}(x,t)] + O(\bar{p}_2^2). \tag{14}$$

Defining z such that

$$\bar{p}_2(t-x) = e^{-zx}\bar{p}_2(t), \tag{15}$$

we have

$$1 = \sum_x e^{-(r_{11}+z)x}\frac{[k_{12}(x,t) + k^*_{12}(x,t)]}{2} + O(\bar{p}_2^2). \tag{16}$$

From (15), we must have $z > 0$ for protected polymorphism, where z can be found from (16). Given time-independent reproductive schedules, the k_{12} and k^*_{12} may be taken as functions of age alone, where they solely depend on the fertility of A_1A_2 crossed with A_1A_1 genotypes, to a sufficient degree of accuracy (cf. Charlesworth, 1980, p. 169).

3. Explicit Protected Polymorphism Conditions

The protected polymorphism condition on z of Eq. (16) is not algebraically transparent, in that it depends on the root of a complex equation. This root is not available analytically, and instead must be obtained numerically. The condition associated with (16) is only an approximation, accurate to no more than $O(\bar{p}_2)$. Here an explicit protected polymorphism condition will be derived from (16), and then compared with another protected polymorphism result for populations with overlapping generations.

It is assumed that z is $O(\bar{p}_2)$. Since z is an exponent in a gene frequency trajectory equation, its magnitude will not normally be great for alleles of beneficial effects. (It may be great for highly deleterious alleles, but these will not be maintained by protected polymorphism.) With this assumption, a Taylor expansion about $z = 0$ will give an approximation for z:

$$1 = \sum_x e^{-(z+r_{11})x}\left[\frac{k_{12}(x)+k_{12}^*(x)}{2}\right]$$
$$= \sum_x e^{-r_{11}x}\left[\frac{k_{12}(x)+k_{12}^*(x)}{2}\right]$$
$$- z\sum_x x\, e^{-r_{11}x}\left[\frac{k_{12}(x)+k_{12}^*(x)}{2}\right]$$
$$+ \frac{z^2}{2}\sum_x x^2 e^{-r_{11}x}\left[\frac{k_{12}(x)+k_{12}^*(x)}{2}\right] + O(z^3),$$

so that

$$z = \frac{\sum e^{-r_{11}x}\left[\frac{k_{12}(x)+k_{12}^*(x)}{2}\right]^{-1}}{\sum x\, e^{-r_{11}x}\left[\frac{k_{12}(x)+k_{12}^*(x)}{2}\right]}$$
$$+ \frac{z^2 \sum x^2 e^{-r_{11}x}\,\frac{[k_{12}(x)+k_{12}^*(x)]}{2}}{\sum x\, e^{-r_{11}x}[k_{12}(x)+k_{12}^*(x)]} + O(z^3).$$

Since

$$\frac{z^2 \sum x^2 e^{-r_{11}x}\,\frac{[k_{12}(x)+k_{12}^*(x)]}{2}}{\sum x\, e^{-r_{11}x}[k_{12}(x)+k_{12}^*(x)]} > 0,$$

a sufficient protected polymorphism condition is

$$\sum_x e^{-r_{11}x}\left[\frac{k_{12}(x)+k_{12}^*(x)}{2}\right] > 1. \tag{17}$$

In the monoecious case, condition (17) reduces to

$$\sum_x e^{-r_{11}x} k_{12}(x) > 1, \tag{18}$$

where $k_{12}(x)$ gives the age-dependent reproductive schedule of A_1A_2 individuals when mated to A_1A_1 individuals. The standard result for monoecious randomly-mating populations is protected polymorphism when

$$r_{12} > r_{ii} + O(p_j), \qquad j \neq i, \qquad i = 1, 2, \tag{19}$$

(cf. Charlesworth, 1980, p. 170). When the $r_{ij}^2 = O(p_j)$, there is in fact no difference between (18) and (19), as will now be shown. When the r_{ij} are not too large, they may be approximated using a Taylor series expansion:

$$r_{ij} = \frac{\sum_x k_{ij}(x) - 1}{\sum_x x k_{ij}(x)} + O(r_{ij}^2) \tag{20}$$

(Charlesworth, 1980, p. 32). When $O(r_{ij}^2) = O(p_j)$, $r_{12} > r_{ii} + O(p_j)$ is equivalent, to $O(p_j)$, to

$$\frac{\sum_x k_{12}(x) - 1}{\sum_x x k_{12}(x)} > \frac{\sum_x k_{ii}(x) - 1}{\sum_x x k_{ii}(x)}. \tag{21}$$

For concreteness, let $i = 1$. Then (21) becomes

$$\left[\sum_x x k_{11}(x)\right]\left[\sum_x k_{12}(x) - 1\right] > \left[\sum_x x k_{12}(x)\right]\left[\sum_x k_{11}(x) - 1\right]. \tag{22}$$

If we define $\delta_{11}(x)$ such that

$$k_{12}(x) = k_{11}(x) + \delta_{11}(x), \tag{23}$$

then (22) simplifies to

$$\left[\sum_x x k_{11}(x)\right]\left[\sum_x \delta_{11}(x)\right] > \sum_x x \delta_{11}(x)\left[\sum_x k_{11}(x) - 1\right].$$

Using (20), this becomes

$$\sum_x \delta_{11}(x)[1 - r_{11} x] > 0, \tag{24}$$

or

$$\sum_x e^{-r_{11}x} \delta_{11}(x) > 0, \tag{25}$$

or

$$\sum_x e^{-r_{11}x} k_{12}(x) > 1, \tag{26}$$

which is equivalent to (18). Evidently, parallel conditions apply when $p_1 \approx 0$, *mutatis mutandis*. Note that in any particular analysis of protected polymorphism, one of conditions (24), (25), and (26) may be more useful than the others.

4. Mortality and Fecundity Conditions for Protected Polymorphism

Conditions (24) to (26) are couched in terms of the net reproductive schedules, the $k_{ij}(x)$, and differences between them. In natural populations, these characters are not very readily observable within a short time frame, because the $l_{ij}(x)$ functions depend on mortality from birth to age x, and it may be difficult to obtain such data from each stage in the life history. Thus it is of some interest to reformulate the protected polymorphism conditions just derived in terms of differences in mortality and fecundity characters. For simplicity, the monoecious case will be considered here; comparable dioecious results follow readily.

(i) *Mortality Effects*

Let there be a difference in mortality during one age interval, say i to $i+1$, between A_jA_j and A_1A_2, $j=1, 2$. In order to facilitate application to $j=1, 2$, and simplify notation, let the reproductive schedule of individuals having genotype A_1A_2 be given by $k(x)$ and that of A_jA_j be $k'(x)$. From (23) and (25) we have protected polymorphism condition

$$\sum_x e^{r'x}[k(x)-k'(x)] > O(1-p'). \tag{27}$$

By our hypothesis, we have $P(i) \neq P'(i)$. Let the mortality difference be given by

$$\Delta v_i = 1 - \frac{P_i'}{P_i}. \tag{28}$$

This allows the following simplification of the *LHS* of (27):

$$\begin{aligned} \sum_x e^{-r'x}[k(x)-k'(x)] &= \sum_{x=i}^{\infty} e^{-r'x}[k(x)-k'(x)] \\ &= \Delta v_i \sum_{x=i}^{\infty} e^{-r'x}k(x). \end{aligned} \tag{29}$$

This result is easily generalized to a set of mortality effects Δv_i, with $i \in A_v$, a set of indices, and j the minimum of such indices, such that, for all $i \in A_v$, $i \geqslant j$ and $\exists i$ such that $i=j$, where j now does not denote an allele. In this case

$$\sum_x e^{-r'x}[k(x)-k'(x)] = \sum_{x=j} e^{-r'x}m(x)\left[\prod_{i=1}^{x} P(i) - \prod_{i=1}^{x} P'(i)\right]$$

$$= \sum_{x=j} e^{-r'x} m(x) \prod_{\substack{i=1 \\ i \notin A_1}}^{x} P(i) \left[\prod_{\substack{i=1 \\ i \in A_1}}^{x} P(i) - \prod_{\substack{i=1 \\ i \in A_1}}^{x} P'(i) \right]$$

$$= \sum_{x=j} e^{-r'x} k(x) \left\{ 1 - \prod_{i=j}^{x} \frac{[P'(i)]}{[P(i)]} \right\} \Bigg|_{i \in A_v}$$

$$= \sum_{x=j} e^{-r'x} k(x) \left\{ \sum_{\substack{i=j \\ i \in A_v}}^{x} \Delta v_i - \sum_{\substack{i=j \\ i \in A_v}}^{x} \left[\sum_{\substack{n=j \\ n \in A_v \\ n \neq i}} \Delta v_i \Delta v_n \right] \right\}$$

$$+ O(\Delta v_i^3) \tag{30}$$

For high fitness alleles, it will almost always be the case that $O(\Delta v_i \Delta v_n) = O(1 - p')$, especially with multiple effects. This gives, for genes of small effect, protected polymorphism condition:

$$\sum_{x=j} e^{-r'x} k(x) \left[\sum_{\substack{i=j \\ i \in A_v}}^{x} \Delta v_i \right] > O(1 - p'). \tag{31}$$

(ii) *Fecundity Effects*

For fecundity, the algebra is much simpler. Let there be fecundity differences during age intervals $i \in A_f$, $i \geq j$, with $\Delta f_i = m(i) - m'(i)$, $i \in A_f$, much as before. With this notation, it is easy to show that protected polymorphism condition (25) becomes

$$\sum_{\substack{i=j \\ i \in A_f}} e^{-r'i} l(i) \Delta f_i > O(1 - p'). \tag{32}$$

(iii) *Combined Fecundity and Mortality Effects*

Let A_v, Δv_i, A_f, and Δf_i be as above, along with r', p', $k(x)$, etc. There are two cases.

If the maximum value of $i \in A_f$ is less than j_v, where j_v is the minimum $i \in A_v$, then protected polymorphism for genes of sufficiently small effects on mortality requires

$$\sum_{\substack{i=jf \\ i \in A_f}} e^{-r'i} l(i) \Delta f_i + \sum_{x=j} \left\{ e^{-r'x} k(x) \left[\sum_{\substack{i=j_v \\ i \in A_v}}^{x} v_i \right] \right\} > O(1 - p'). \tag{33}$$

When the last age of gene effects on fecundity is greater than j_v, then (33) is not correct, unless it is assume that terms of $O(\Delta v_{i_1} \Delta f_{i_2})$ are of no

greater magnitude than $O(\Delta v_{i_1} \Delta v_{i_2})$ terms. Otherwise, condition (33) must be corrected by at least a factor of

$$-\sum_x e^{-r'x} l(x) \left[\sum_j^x \Delta v_j \Delta f_x \right].$$

For alleles which are not markedly deleterious, condition (33) will normally suffice.

5. Explicit Genetic Models

As in Rose (1982), the effect of different patterns of gene action on protected polymorphism in the diallelic monoecious case will be investigated. Again, the assumption of no overdominant gene effects on particular characters will be made. There are two levels at which such genetic analysis can proceed: that of net reproductive schedules and that of mortality and fecundity.

(i) *Reproductive Schedule Model*

Reverting to the notation of Sections 2 and 3, let there be disjoint sets of indices A and B, such that the following pattern of gene effects arises.

	A_1A_1	A_1A_2	A_2A_2
$k_{ij}(x \in A)$	$k(x) - \varepsilon_x$	$k(x)$	$k(x) + h_x\varepsilon_x$
$k_{ij}(x \in B)$	$k(x) + h_x\varepsilon_x$	$k(x)$	$k(x) - \varepsilon_x$

Here we have $h_x \geqslant 0$ and $\varepsilon_x > 0$. Using (23) and (25), the protected polymorphism conditions become:

$$\sum_{x \in A} e^{-r_{11}x} \varepsilon_x - \sum_{x \in B} e^{-r_{11}x} h_x \varepsilon_x > O(1 - p_2) \tag{34a}$$

$$-\sum_{x \in A} e^{-r_{22}x} h_x \varepsilon_x + \sum_{x \in B} e^{-r_{22}x} \varepsilon_x > O(1 - p_1) \tag{34b}$$

Two conclusions emerge from this result. First, as $h_x \to 0$, protected polymorphism becomes more likely. Conversely, as $h_x \to \infty$, protected polymorphism becomes less likely. This result is similar to those found for discrete-generation models (Rose, 1982). Second, for populations increasing in density, effects expressed at later ages will have less effect, and conversely for populations decreasing in density. (See below for more on this second point.)

(ii) *Mortality and Fecundity Model*

Define A_v and B_v as disjoint index sets for gene effects on mortality, and A_f and B_f as disjoint index sets for gene effects on fecundity. Define h_x^v, v_x, h_x^f, and f_x to correspond with the h_x and ε_x variables of the previous model, with $h_y^z \geqslant 0$ and $z_y > 0$. This gives the following pattern of gene effects on life history characters.

Mortality

	A_1A_1	A_1A_2	A_2A_2
$P_{ij}(x \in A_v)$	$P(x) - v_x$	$P(x)$	$P(x) + h_x^v v_x$
$P_{ij}(x \in B_v)$	$P(x) + h_x^v v_x$	$P(x)$	$P(x) - v_x$

Fecundity

	A_1A_1	A_1A_2	A_2A_2
$m_{ij}(x \in A_f)$	$m(x) - f_x$	$m(x)$	$m(x) + h_x^f f_x$
$m_{ij}(x \in B_f)$	$m(x) + h_x^f f_x$	$m(x)$	$m(x) - f_x$

At any particular age of action, for either genotype (28) gives $\Delta v_i = v_i/P(i)$ or $\Delta v_i = -h_i^v v_i/P(i)$, while Δf_i is analogous to $\delta_{jj}(i)$. Thus (33) becomes, in the case of A_1A_1,

$$\sum_{i \in A_f} e^{-r_{11}i} l(i) f_i - \sum_{i \in B_f} e^{-r_{11}i} l(i) h_i^f f_i + \sum_x \left\{ e^{-r_{11}x} k(x) \left[\sum_{i \in A_v}^{x} \frac{v_i}{P(i)} \right] \right\} - \sum_x \left\{ e^{-r_{11}x} k(x) \left[\sum_{i \in B_v}^{x} \frac{h_i^v v_i}{P(i)} \right] \right\} > O(1 - p_2), \tag{35a}$$

and

$$- \sum_{i \in A_f} e^{-r_{22}i} l(i) h_i^f f_i + \sum_{i \in B_f} e^{-r_{22}i} l(i) f_i - \sum_x \left\{ e^{-r_{22}x} k(x) \left[\sum_{i \in A_v}^{x} \frac{h_i^v v_i}{P(i)} \right] \right\} + \sum_x \left\{ e^{-r_{22}x} k(x) \left[\sum_{i \in B_v}^{x} \frac{v_i}{P(i)} \right] \right\} > O(1 - p_1), \tag{35b}$$

for A_2A_2.

As in the reproductive schedule model, as $h_i^g \to 0$, conditions (35) are more readily achieved. Thus, recessive deleterious gene action fosters protected polymorphism however the life history is characterized.

It should be emphasized that the patterns of gene action examined here are not completely general. Most importantly, it has been assumed that homozygous benefits at some age x are always associated with some heterozygous effect at the same age. (Note that this is not true of deleterious effects in these models.) This of course need not be true, in principle.

The age dependence of gene effect contributions to protected polymorphism is more complex with conditions (35) as compared with conditions (34). The terms in (35a) and (35b) giving the weights of these contributions are of the same form as the $s(x)$ and $s'(x)$ terms in the numerators of the corresponding equations concerning age-dependence of mortality and fecundity effects in Charlesworth (1980, pp. 206–214). As discussed on several occasions (e.g., Hamilton, 1966; Charlesworth and Williamson, 1975; Charlesworth, 1980), these weighting factors usually make life history gene effects less subject to natural selection with increasing age of action, the only exception being genes with late effects on fecundity in sharply declining populations. Thus late-age gene action will have little effect upon the establishment of protected polymorphism.

These results have been found for monoecious populations using condition (25). However, because of the similar form of (17), comparable results are readily obtainable for dioecious populations.

6. Numerical Results

Numerical calculations were performed in order to check the validity of the approximate analytical results and to obtain further information concerning the evolutionary effects of antagonistic pleiotropy in populations with age structure. These calculations were made in FORTH on IBM PC's augmented with Intel 8087 coprocessors for hardware floating-point arithmetic. A monoecious population with 10 age classes was assumed, with a single diallelic locus affecting life history attributes, as in the mathematical analyses above. The Malthusian parameter for each genotype was obtained by iterating the genotype's Leslie matrix, as if the genotype was growing in a pure culture, and calculating its dominant eigenvalue once a stable age distribution had been achieved. Direct iteration of the evolutionary equations revealed that the Malthusian parameters estimated using this procedure gave the correct evolutionary outcome. Thus these estimated Malthusian parameters were used to infer the evolutionary consequences of different life history genetics patterns.

A wide variety of basic life history patterns were examined, with age-specific survival probabilities and age-specific fecundities *both* either increasing or decreasing. Curves of both increase and decrease were linear,

convex, or concave. Eight genetic effects were pseudorandomly imposed upon each basic life history pattern of 20 characters, 20 times for each "dominance-level". This dominance level was the order of magnitude of the h_i dominance parameters. It was varied from 1 to 0.001. The magnitudes of all ε and δ parameters were a few percent of the magnitudes of the life history characters they affected. The genetic effects were contrived so that each beneficial allelic effect was associated with an antagonistic, pleiotropic, deleterious effect on another life history character.

A total of 7680 cases were studied numerically. Of these, in only 14 instances did conditions (35) predict protected polymorphism when fixation was the calculated evolutionary outcome, an error frequency of 0.0068. On the other hand, in 2065 of 7680 cases conditions (35) allowed the possibility that fixation could occur when, in fact, protected polymorphism was predicted numerically. Thus, conditions (35) seem to be fairly reliable *sufficient* conditions for protected polymorphism, but not reliable necessary conditions.

In a previous study of antagonistic pleiotropy in populations with discrete generations, it was found that stable polymorphic equilibria frequently arose without much dominance variance, relative to the magnitude of additive genetic variance, for each life history character (Rose, 1982). While such cases were also discovered numerically in the present numerical study, they did not predominate. In a comparable number of cases at equilibrium, some characters exhibited substantially greater dominance variance, relative to additive genetic variance. Thus, if antagonistic pleiotropy at a number of loci were responsible for the maintenance of genetic variability in life history characters in populations with age structure, then there could be any proportion of additive to total genetic variance.

The analytical conclusion of greatest importance found here and previously (Rose, 1982, 1983) is that recessive deleterious effects foster polymorphism. The numerical results underscore this conclusion. As shown in Tables 1 and 2, irrespective of life history pattern, as the h_i approach zero, polymorphism is more frequent.

Of the life history patterns employed, two factors stood out as facilitating polymorphism: high initial survival probability and increasing fecundity. These patterns are documented in Tables 1 and 2, respectively. In both cases, the adduced pattern held up over a wide variety of cases. In particular, whether an increasing or decreasing age-specific character followed a linear, convex, or concave curve did not seem to matter. Intuitively, both of these effects can be understood in terms of the effective number of life history characters which make a major contribution to fitness. If individuals die off very rapidly or fecundity does not increase with age then gene effects expressed at later ages will be of less importance. Since the

TABLE 1

Effect of Initial Survivorship and Dominance on Polymorphism

Dominance Level	Initial survival probability	Number of runs	Number polymorphic	Percentage polymorphic
1.0	.2	960	41	4.27
	.8	960	60	6.25
0.1	.2	960	356	37.08
	.8	960	563	58.65
0.01	.2	960	617	64.27
	.8	960	803	83.65
0.001	.2	960	776	80.83
	.8	960	872	90.83

numerical cases considered involved eight genetic effects scattered evenly over 10 age classes, some of these genetic effects will be expressed at later ages. It often happened that most of the beneficial effects expressed in the first two or three age classes were due to one of the two alleles. In such cases, low survivorship or fecundity at later ages would militate against protected polymorphism, and conversely. However, results of this kind depend critically on the programmed assumptions, in this case the random scattering of pleiotropic effects over age classes.

TABLE 2

Effect of Fecundity Pattern and Dominance on Polymorphism

Dominance level	Fecundity pattern	Number of runs	Number polymorphic	Percentage polymorphic
1.0	Non-increasing	1040	46	4.42
	Increasing	880	56	6.36
0.1	Non-increasing	1040	409	39.72
	Increasing	880	510	57.95
0.01	Non-increasing	1040	707	67.98
	Increasing	880	713	81.02
0.001	Non-increasing	1040	865	83.17
	Increasing	880	783	88.98

7. Discussion

There are two major points of interest which arise from the present analysis. First, results first obtained for discrete-generation models have also been found for a model with overlapping generations. As found in Rose (1982, 1983), antagonistic pleiotropy allows protected polymorphism in the absence of overdominance in gene effects on individual life history characters. In addition, as found before, recessive deleterious gene action fosters the establishment of such protected polymorphism. These results thus appear to have considerable generality, in that they have been found for most of the well-understood population genetic models. Though such models are of course extremely limited, it is tempting to suggest that these conclusions also hold for more than two loci, more than three alleles at a locus, overlapping generations in continuous time, and so on. Certainly, the consistency of the theoretical results suggests that experiments directed at testing their validity in real populations should be performed.

Second, and more specifically, one point of interest which emerges from these results is that the maintenance of genetic variability affecting early and late life history characters depends primarily on the early effects. Abundant genetic variability in rates of senescence could be maintained in natural populations as a result of antagonistic pleiotropic gene effects at early ages. Thus, there may be a great deal of allelic variability at loci involved in determining age of death and the health of old organisms. This would go some way to explaining the genetic variability in later life history characters detected in Rose and Charlesworth (1981b). The exploitation of such genetic variability to create longer-lived populations could allow a much greater rate of progress in unravelling the physiological genetics of senescence, thereby giving gerontology a significantly improved basis for research into the mechanisms and postponement of senescent deterioration.

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