Selection and the Evolution of Genetic Life Cycles

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ABSTRACT

The evolution of haploid and diploid phases of the life cycle is investigated theoretically, using a model where the relative length of haploid and diploid phases is under genetic control. The model assumes that selection occurs in both phases and that fitness in each phase is a function of the time spent in that phase. The equilibrium and stability conditions that allow for all-haploid, all-diploid, or polyphasic life cycles are considered for general survivorship functions. Types of stable life cycles depend on the form of the viability selection. If mortality rates are constant, either haploidy or diploidy is the only stable life cycle possible. Departures from constant mortality can give qualitatively different results. For example, when survivorship in each phase is a linear, decreasing function of the time spent in the phase, stable haploid, diploid or polyphasic life cycles are possible. The addition of genetic variation at a coevolving viability locus does not qualitatively affect the outcome with respect to the maintenance of polyphasic cycles but can lead to situations where more than one life cycle is concurrently stable. These results show that trade-offs between the advantages of being diploid and of being haploid may help explain the patterns of life cycles found in nature and that the type of selection may be critical to determining the results.

O NE of the most fundamental characteristics of meiotic reproduction is the fact that the entire genome must pass through both a haploid and a diploid phase every generation. The necessity of this genetic cycling imposes a constraint, but evolution can adjust the timing of the events involved. The result is a wide variety of genetic life cycle patterns. The most obvious are the extremes where one or the other phase has been reduced to a single cell; all animals retain a haploid phase only in gametes, while a large number of protists, algae and fungi spend their entire lives as haploids with the exception of a fleeting diploid phase. But these extremes are not the only options available. For example, angiosperms spend the majority of time as diploids but retain a great amount of gene expression in the haploid (pollen) phase, mosses have a vegetative haploid phase that metabolically supports a diploid reproductive phase, and the red algae have haploid and diploid phases that are usually separate individuals and often morphologically indistinguishable.

The evolutionary processes that give rise to this wide diversity in genetic life cycles are poorly understood (Williams 1975; Maynard Smith 1978; Bold, Alexopoulos and Delevoryas 1980; Bell 1982; Willson 1983; Valero et al. 1992). Most previous work has focused on the advantages of diploidy over haploidy, usually stressing the genetical consequences of having one vs. two homologous genomes (Muller 1932; Crow and Kimura 1965; Bernstein, Byers and Michod 1981; Kondrashov and Crow 1991; Peirrot, Richerd and Valero 1991; Otto and Goldstein 1992). Less attention has been given to the maintenance of life cycles that retain both phases (Willson 1983; Couzens 1988; De Wreeede and Klinger 1988; Destombe et al. 1989; Mclachlan 1991; Richerd, Couvet and Valero 1993). Theoretical treatment of both of these issues is still incomplete. The objective of this research is to provide a theory for understanding the diversity of life cycles seen in nature by considering the impact of selection on the evolution of genetic life cycles.

The evolutionary force to be considered here is selection acting on genes that are expressed in both genetic phases. Selection of this type would include ecological and physiological factors that influence the relative advantages of the haploid and diploid phase, such as different morphologies, niches or inherent differences in haploid and diploid cells (such as size or growth rates). Viability in each phase is assumed to be a function of the time spent in that phase. This approach reflects the idea that the length of each genetic phase may be the result of a balance between the advantages of being diploid and of being haploid. The problem of interest is to determine the conditions that will lead to the evolution of all-haploid or all-diploid genetic life cycles or to life cycles that retain both phases.

In order to do this, a theory of the evolution of genetic life cycles is developed in this paper. First, I consider general conditions for stable life cycles when selection is constant (Model 1) and present two special cases to illustrate types of possible life cycles. Since selection on the life cycle is assumed to act through
its effects on viability traits expressed in both phases, the model is expanded to include selection which can lead to the maintenance of genetic variation at a viability locus (Model 2). Equilibria for both the life cycle and the viability locus are then determined and examples presented to illustrate the results.

**EVOLUTION OF THE LIFE CYCLE**

In this section I will examine the evolution of the genetic life cycle in organisms where selection acts in both the haploid and diploid phase. I will assume that viability in each phase is a function of the time spent in that phase and that the total life cycle has a fixed duration, leading to a trade-off between the length of the haploid and diploid phases. General conditions are given for the stability of the life cycle and some simple examples are presented to illustrate the effects of different forms of selection.

**The life cycle:** Consider a population of meiotically reproducing individuals whose genetic life cycle therefore includes both haploid and diploid phases each generation, giving the potential for selection in both phases. The life cycle is characterized by a parameter $t$ that represents the proportion of the life spent in the haploid phase, with $1 - t$ defining the proportion spent as a diploid (Figure 1). A value of $t = 1$ indicates an all-haploid life cycle, meaning that syngamy is followed immediately by meiosis and there is no selection on the haploid phase. Likewise, a value of $t = 0$ indicates an all-diploid life cycle, with meiosis followed immediately by syngamy and no selection on the haploid phase. For polyphasic life cycles ($0 < t < 1$), selection acts in both phases and fitness in each phase is a function of the proportion of the life cycle spent in that phase.

Assume that the life cycle is under genetic control and that any life cycle ($0 \leq t \leq 1$) is possible. If we postulate that syngamy is synchronous, perhaps due to external effects such as seasonality, it is possible for the genotype in the diploid phase to control the entire life cycle. Assume that the amount of time spent in the diploid phase is determined by the individual's diploid genotype, and the time spent in the haploid phase by all offspring of that diploid (regardless of haploid genotype) is determined by the time left until syngamy. This assumption has an additional technical advantage in that it assures constant total life cycle length, regardless of genotype. This is desirable in order to consider evolution due only to the effects of changes in the relative proportions of time spent in each phase and not to changes in total life cycle length.

**General conditions for the stability of life cycles (Model 1):** If the diploid genotype controls the life cycle, the fitness of each genotype is determined by two episodes of selection, selection during the diploid and the haploid phase. For a randomly mating, infinite population with constant selection where $q$ is the frequency of an allele at syngamy, it can be shown that:

$$
\Delta q = \frac{q(1 - q)}{2W} \frac{d\bar{W}}{dq}
$$

with $\bar{W} = \bar{W}_H\bar{W}_D$, where $\bar{W}_H$ is the mean fitness in the haploid phase and $\bar{W}_D$ the mean fitness in the diploid phase. Thus, if relative fitnesses remain constant, $\bar{W}$ is locally maximized at an equilibrium, consistent with standard diploid theory (Wright 1969, p. 32). An allele that codes for a life cycle $\hat{t}$ that maximizes $\bar{W}$ (subject to the feasibility constraint that $0 \leq \hat{t} \leq 1$) will be stable to invasion by other alleles when fixed in the population.

Since the model considers viability selection, haploid and diploid fitnesses for a given life cycle $t$ can be written as the general survivorship functions:

$$
\bar{W}_H(t) = \exp\left(-\int_0^t \mu_H(x)dx\right)
$$

$$
\bar{W}_D(t) = \exp\left(-\int_0^{1-t} \mu_D(x)dx\right)
$$

where $\mu_H(x)$ is the haploid mortality rate at time $x$ since meiosis and $\mu_D(x)$ is the diploid mortality rate at time $x$ since syngamy. If $\bar{W} = \bar{W}_H\bar{W}_D$ is monotonically increasing with respect to $t$, haploidy is the only stable life cycle ($\hat{t} = 1$); only diploidy is stable ($\hat{t} = 0$) if $\bar{W}$ is monotonically decreasing with respect to $t$. In order for polyphasic cycles to be stable, a local maximum for $\bar{W}$ must exist for $0 < t < 1$. The location of such a maximum point is determined by setting $\frac{d\bar{W}}{dt} = 0$ and solving for $\hat{t}$. If $0 < \hat{t} < 1$ and $\frac{d^2\bar{W}}{dt^2} \bigg|_{\hat{t}} < 0$, then $\hat{t}$ describes a polyphase cycle and is stable. For the general survivorship functions given above, these stability conditions become:

$$
\mu_H(\hat{t}) = \mu_D(1 - \hat{t}) \quad \text{for} \quad 0 < \hat{t} < 1, \quad \text{and} \quad (2a)
$$

$$
\mu_D(\hat{t}) > \mu_D(1 - \hat{t}), \quad (2b)
$$

where primes denote the derivative with respect to $t$. 
If Equation 2a is met but 2b is not, there is an unstable polyphasic equilibrium. Polyphasic cycles are stable whenever the increase in \( W \) from removing a unit of time from one phase is equal to the decrease from adding that unit of time to the other phase.

**Special cases:** In order to get an idea of the range of stable life cycles possible for different kinds of selection, two examples are discussed in detail in this section. In the first example, mortality rates are constant over time within each phase. In the second, survivorship in each phase is a decreasing linear function of time spent in the phase, giving mortality rates that increase throughout each phase.

**Constant mortality:** Perhaps the simplest type of time-dependent fitness is that where there are constant mortality rates. Let \( \mu_H \) be the haploid phase mortality rate and \( \mu_D \) the diploid phase mortality rate. Then \( W_H = \exp(-\mu_H t) \) and \( W_D = \exp(-\mu_D (1 - t)) \). The only stable life cycles are haploidy (\( \hat{t} = 1 \)) when \( \mu_D > \mu_H \) or diploidy (\( \hat{t} = 0 \)) when \( \mu_D < \mu_H \). Polyphasic cycles are never stable (Figure 2a). In this case, it is easy to show that the phase with the lower mortality rate is favored, as one might expect intuitively.

**Linear survivorship:** Though constant mortality rates lead to only haploid or diploid stable cycles, deviations from constant mortality can lead to qualitatively different results. As a simple example, consider a linear survivorship function in which \( W_H = 1 - vt \) and \( W_D = 1 - s(1 - t) \). Mortality rates within each phase increase with the time spent in that phase. Selection that operates in this manner is equivalent to a type of senescence. Figure 2b illustrates the stable life cycles possible for each combination of haploid and diploid selection coefficients. Stable life cycles are:

\[
\hat{t} = \begin{cases} 
0 & \text{for } s \leq \frac{v}{1 + v} \\
\frac{v}{2uv} - \frac{s}{2w} & \text{for } \frac{v}{1 + v} < s < \frac{v}{1 - v} \\
1 & \text{for } \frac{v}{1 - v} \leq s.
\end{cases}
\]  

Life cycles that include both phases (\( 0 < \hat{t} < 1 \)) are stable for a large range of parameters. For each point within this polyphasic region, a unique value of \( \hat{t} \) obtains; that is, there are never multiple stable life cycles for a given set of parameters in this case. For example, for every combination of parameters lying along the dashed line in Figure 2b (where \( s = v \)), the stable life cycle is one with equal haploid and diploid phases (\( \hat{t} = \frac{1}{2} \)).

For this type of viability selection, the range of selection parameters that give a stable polyphasic life cycle is small when selection is weak in both life cycle phases, implying that monophasic cycles are favored. The opposite is true when the selection coefficients are large in both phases, implying polyphasic cycles are favored. This result makes sense in light of the fact that, for very small values of \( s \) and \( v \), the linear survivorship function is closely approximated by a constant mortality function.

**JOINT EVOLUTION OF THE LIFE CYCLE AND A VIABILITY TRAIT**

In the analysis considered above, selection on the life cycle is assumed to act through its effects on viability traits expressed in both phases. If there is genetic variation available for such traits, evolution may proceed in two ways in response to selection: the haploid/diploid life cycle ratio can shift and gene frequencies at the viability loci may change. In this case, the marginal relative fitnesses of alleles at the life cycle controlling locus are no longer constant in time, and the evolutionary problem is more complicated. Does the addition of genetic variation at a viability locus change the qualitative results found above? To address this issue, the evolution of the life cycle is analyzed for two examples where selection can maintain variation at a viability locus. First, I present the conditions for evolution of a viability locus which is expressed and is under selection in both the haploid and diploid phase. An optimization approach is then developed to determine stable equilibria for the life cycle and the viability locus, and the equilibria found are tested with a two-locus stability analysis. Finally, two special cases are discussed to illustrate the types of life cycles possible.

**Evolution of a trait under haploid and diploid selection:** Several authors have previously considered the evolution of a trait in a genetic system that includes both haploid and diploid phases (Scudo 1967; Wright 1969; Ewing 1977; Gregorius 1982). The life cycle in each instance was assumed fixed. As it is important for purposes of this model to understand how any genetically determined trait will evolve if it is under selection in both the haploid and diploid phase, the basic results are reviewed here.

Assume that there is a genetic locus \( A \) controlling a viability trait that is expressed in each phase. Locus \( A \) segregates for two alleles; let \( p \) be the frequency of allele \( A_1 \) in haploids after meiosis and before selection. This represents a different census point than that used in the analysis of the diploid-controlled life cycle (Model 1). In this case, censusing at the beginning of the haploid phase allows the derivation of a useful optimization criterion (see below).

Let \( w_i \) be the fitness of allele \( A_i \) in the haploid phase and \( w_{ij} \) be the fitness of genotype \( A_iA_j \) in the diploid phase. In a randomly mating, infinite population with
constant selection, the one-generation change in allele frequency at locus $A$ is

$$
\Delta p = p(1 - p)(w_1^2w_{11}p + w_1w_2w_{12}(1 - 2p) - w_2^2w_{22}(1 - p))/\hat{W}
$$

(4)

$$
= p(1 - p) d\hat{W}/dp
$$

where:

$$
\hat{W} = w_1^2w_{11}p^2 + 2w_1w_2w_{12}(1 - p) + w_2^2w_{22}(1 - p)^2
$$

(5)

That is, allele frequencies will evolve to maximize $\hat{W}$ (WRIGHT 1969, p. 56).

Analysis of these equations shows that a viability locus equilibrium at $\bar{p} = 1$ is locally stable when $w_1w_{11} > w_2w_{12}$, and $\bar{p} = 0$ is locally stable when $w_2w_{22} > w_1w_{12}$. If both $w_1w_{11} < w_2w_{12}$ and $w_2w_{22} < w_1w_{12}$, a polymorphism is stable ($0 < \bar{p} < 1$). Unlike the standard diploid model, overdominance in the diploid phase is neither necessary nor sufficient for the establishment of a stable polymorphism, and directional selection favoring the same allele in both phases is sufficient, but not necessary, for fixation of a single allele (SCUDO 1967; WRIGHT 1969; EWING 1977). It is apparent from the conditions above that if a locally stable equilibrium exists at $\bar{p} = 1, \bar{p} = 0$, or both, it is not possible to also have a stable equilibrium that is polymorphic ($0 < \bar{p} < 1$).

Simultaneous evolution of the life cycle and viability locus (Model 2): Assume that fitness in each life cycle phase is both a function of the genotype at a viability locus $A$ and also of the time spent in that phase. The focus of the rest of this analysis is to consider the evolution of the life cycle when the life cycle and a viability trait are coevolving. We might conjecture that the life cycle, $t$, still evolves as shown in Model 1, to maximize $\hat{W} = \hat{W}_H\hat{W}_D$, while allele frequencies at the viability locus, $A$, simultaneously evolve to maximize $\hat{W}$, as described above. For purposes of comparison, Equation 5 can be rewritten in the form:

$$
\hat{W} = \hat{W}_H\hat{W}_D
$$

(6)

where

$$
\hat{W}_H = w_1p + w_2(1 - p)
$$

is the mean viability of the haploid phase,

$$
\hat{W}_D = w_1p^* + 2w_2p^*(1 - p^*) + w_2(1 - p^*)^2
$$

$$
= w_{11}(w_1p)^2 + 2w_{12}(w_1w_2p(1 - p)) + w_{22}(w_2(1 - p))^2
$$

(7)

is the mean viability of the subsequent diploid phase, and $p^*$ is the frequency of allele $A_1$ in diploid zygotes before selection. The intuition of this result is clear if we consider that zygote frequencies are determined by the products of uniting haploid gametes that may have different viability.

The viability locus therefore evolves to maximize a different value ($\hat{W} = \hat{W}_H^2\hat{W}_D$) than does the life cycle locus (where $\hat{W} = \hat{W}_H\hat{W}_D$ is maximized). The difference is due to the fact that the viability alleles are expressed in both the haploid and diploid phases, allowing selection to operate on both haploid and diploid genotypes. Selection acts only on the diploid genotype of the life cycle locus, for the diploid genotype controls both the haploid and diploid phenotype.

The strategy used to determine the evolutionarily stable life cycle $\bar{t}$ involves two steps. First, candidate equilibria are identified using an optimization approach. This approach is based on the conjecture that the life cycle, $t$, evolves to maximize $\hat{W} = \hat{W}_H\hat{W}_D$, while allele frequencies at the viability locus, $A$, evolve to maximize $\hat{W} = \hat{W}_H^2\hat{W}_D$. In order to find candidate
equilibria, values for $t$ and $p$ are found that jointly maximize these two expressions. Once these equilibria are identified, the second step is to verify their stability using a formal two-locus genetic model. The two-locus model developed to do this is presented in APPENDIX A. This model assumes that there are two autosomal loci: locus $M$, a life cycle modifier that determines the value of $t$, and locus $A$, a viability locus that is expressed in each phase. Each locus segregates for two alleles. The model assumes that $M_i$ is the common allele; $M_iM_j$ homozygotes give a life cycle characterized by $t$. The two-locus analysis determines whether a rare allele $M_k$, that may code for any other possible life cycle, will spread in the population. The fitness functions used in each case described below are substituted into the general two-locus recursion equations and standard linear stability analysis is used to verify stability of the equilibria found from the optimization analysis.

Special cases: Because the interest here is in cases that may lead to polymorphism at the viability locus, I will focus on two situations that can maintain variation: a case where diploid heterozygotes have over-dominant fitness and a case of antagonistic pleiotropy, where directional selection on the viability trait acts in opposite directions in the two phases. In each example, fitness in each phase is also a function of time spent in the phase. For each case discussed below, values for $t$ and $p$ were determined using the optimization approach discussed above. Stability of each of these equilibria has been verified by the two-locus analysis presented in APPENDIX A.

A cautionary note must be made about the interpretation of the “selection coefficients” $v$ and $s$ used in the following examples. As can be seen from the way they are used in the fitness functions (Table 1), they do not represent precisely the same parameter in each case. Therefore, it is hard to make direct comparisons concerning the type of stable life cycle possible for a given combination of $v$ and $s$ in the different examples.

Overdominance: One mechanism for retaining variation is selection favoring the heterozygote (CROW and KIMURA 1970). I have analyzed both the constant mortality case (Table 1a) and the linear survivorship case (Table 1b) assuming symmetric overdominance at locus $A$ in diploids and equal viabilities in the haploid phase. Analysis shows there is a stable equilibrium of $p = \frac{1}{2}$ at the $A$ locus (for all values of $s$ and $v$) for both survivorship functions.

When mortality is constant (Table 1a), the only stable life cycles are haploidy ($t = 1$) when $s > 2v/3$ or diploidy ($t = 0$) when $s < v - e^{-(2s-v)}$ (Figure 3a). As in Model 1, polyphasic cycles are never stable. An interesting difference does occur in that for the small region where $2v/3 < s < v - e^{-(2s-v)}$, both haploidy and diploidy are locally stable; which equilibrium is reached will depend on the initial conditions of the population.

When the survivorship function is linear (Table 1b), stable haploidy, diploidy or polyphasic cycles can occur (Figure 3b). Stable life cycles are:

$$
\hat{t} = \begin{cases} 
0 & \text{for } s \leq \frac{2v}{3 + 3v} \\
\frac{3vs - 2v + 3s}{6vs} & \text{for } \frac{2v}{3 + 3v} < s < \frac{2v}{3 - 3v} \\
1 & \text{for } \frac{2v}{3 - 3v} \leq s.
\end{cases}
$$

As in Model 1, there is a range of intermediate combinations of haploid and diploid selection coefficients that results in stable polyphasic life cycles, with the length of each genetic phase determined by a balance between the strength of selection in each phase. For each point within this polyphasic region, a unique value of $t$ obtains; that is, there are never multiple stable life cycles for a given set of parameters in this case. For example, for every combination of parameters lying along the dashed line in Figure 3b (where $s = 2v/3$), the stable life cycle is one with equal haploid and diploid phases ($t = \frac{1}{2}$).

As seen from these results, the main effect of the addition of overdominance at locus $A$ in this example for either survivorship assumption appears to be to shift the range of parameters that give each type of stable life cycle. The results with respect to the maintenance of polyphasic cycles are equivalent to Model 1 for both survivorship functions. Haploidy and diploidy are still the only stable life cycles if mortality rates are constant, while the linear survivorship function results in stable haploid, diploid or polyphasic cycles. The addition of this type of variation at the viability locus may, however, lead to situations where more than one life cycle is concurrently stable, as in the constant mortality example.

Antagonistic pleiotropy: Another mechanism that may maintain variation at the $A$ locus is antagonistic pleiotropy (ROSE 1982), where directional selection on the viability trait acts in opposite directions in the two genetic phases. In the situation considered here, where both the life cycle and the viability trait can evolve, this type of selection has the interesting properties that more than one equilibrium value of $p$ is possible, a stable polymorphism ($0 < p < 1$) is never possible and, for certain values of the selection coefficients, stable equilibria at both $p = 1$ and $p = 0$ are possible. For each given equilibrium at $A$, however, there is no difference from Model 1 with respect to the maintenance of polyphasic cycles; constant mortality still gives only stable haploid or diploid cycles while linear survivorship can lead to stable haploidy, diploidy and polyphasic cycles. Antagonistic pleiotropy can, however, lead to situations where more
TABLE 1

Fitness functions: \( w_i = \) the viability of allele \( A_i \) in the haploid phase and \( w_{ij} = \) the viability of genotype \( AA_j \) in the diploid phase

<table>
<thead>
<tr>
<th>Overdominance</th>
<th>Antagonistic pleiotropy</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Haploid fitness</strong></td>
<td><strong>Diploid fitness</strong></td>
</tr>
<tr>
<td>a. Constant mortality:</td>
<td>( w_1 = \exp(-ut) )</td>
</tr>
<tr>
<td>( w_2 = \exp(-ut) )</td>
<td>( w_{12} = \exp(-2s(1-t)) )</td>
</tr>
<tr>
<td>b. Linear survivorship:</td>
<td>( w_1 = 1 - vt )</td>
</tr>
<tr>
<td>( w_2 = 1 - vt )</td>
<td>( w_{12} = 1 - s(1-t) )</td>
</tr>
</tbody>
</table>

\( 0 \leq t \leq 1, 0 \leq v \leq 1 \) and \( 0 \leq s \leq 1 \).

Figure 3.—Simultaneous evolution of the life cycle and viability locus \( A \) (Model 2). (a). Stable life cycles: overdominance and constant mortality. Stable life cycles: overdominance and linear survivorship. The dashed line \( s = 2v/3 \) is included for reference: for each point along this line the stable life cycle is one with equal haploid and diploid phases \( (i = \frac{1}{2}) \). (c). Equilibria for locus \( A \) and corresponding stable life cycles: antagonistic pleiotropy and constant mortality. (d). Equilibria for locus \( A \) and corresponding stable life cycles: antagonistic pleiotropy and linear survivorship. \( \text{H:0} = \) haploidy \( (i = 1) \) and \( \beta = 0 \); \( \text{P:0} = \) polyphasic \( (0 < i < 1) \) and \( \beta = 0 \); \( \text{P:1} = \) polyphasic \( (0 < i < 1) \) and \( \beta = 1 \); \( \text{D:1} = \) diploidy \( (i = 0) \) and \( \beta = 1 \).

than one life cycle is concurrently stable. To illustrate, examples of antagonistic pleiotropy with constant mortality or linear survivorship are analyzed in detail below and the results illustrated in Figure 3, c and d.

First, assume that mortality rates are constant and fitnesses take the form shown in Table 1c; the \( A_2 \) allele is favored in the haploid phase and the \( A_1 \) allele is favored in the diploid phase. As in all previous examples with constant mortality rates, only haploidy and diploidy are stable life cycles, as shown in Figure 3c. Stable equilibria exist at \( i = 1 \) and \( \beta = 0 \) when \( s > v/2 \) and at \( i = 0 \) and \( \beta = 1 \) when \( s < 2v \). In the region where \( v/2 < s < 2v \), both haploidy and diploidy are stable, in concert with the different \( A \) locus equilibria. Which equilibrium is reached depends only on the initial conditions of the population.
Now assume a linear survivorship function with fitnesses as shown in Table 1d. Allele $A_2$ is again favored in the haploid phase and $A_1$ in the diploid phase. The following equilibria are stable (Figure 3d):

$$i = 0, \hat{p} = 1 \text{ for } s \leq \frac{2v}{1 + 2v'}$$

$$i = \frac{2vs - 2v + s}{4vs}, \hat{p} = 1 \text{ for } \frac{2v}{1 + 2v} < s < \frac{4v^2 - 8v + 8v + v^2}{5 - 8v - 4v^2}$$

$$\hat{i} = \frac{2vs - v + 2s}{4vs}, \hat{p} = 0 \text{ for } \frac{v + 4v^2 - v\sqrt{25 - 4v + 4v^2}}{4v^2 + 4v - 8} < s < \frac{v}{2 - 2v'}$$

$$\hat{i} = 1, \hat{p} = 0 \text{ for } \frac{v}{2 - 2v} \leq s.$$  \hspace{1cm} (8)

Again, for many combinations of $s$ and $v$, two locally stable $A$ locus equilibria are possible simultaneously, one at fixation of the $A_1$ allele and one at fixation of the $A_2$ allele. As was true in the other cases of linear survivorship analyzed, polyphasic life cycles are stable over a large portion of the parameter space, with the basic pattern of stable life cycles possible for each individual equilibrium at $A$ similar to the results from Model 1. In this case, however, for many combinations of selection coefficients, two different stable life cycles may evolve, in concert with the different $A$ locus equilibria. The equilibrium state reached under these conditions will be determined by the initial conditions of the population. Therefore, as can be seen in Figure 3d, this type of selection can lead to a wide variety of different stable life cycles.

**DISCUSSION**

The central conclusion of this analysis is that selection operating on each phase of the life cycle can result in stable haploidy, diploidy or life cycles that retain both phases. If selection in each phase is a function of the time spent in that phase, the life cycle reflects a trade-off between advantages of being diploid and of being haploid. The ability of selection to maintain polyphasic life cycles, however, depends on the form the survivorship function takes with respect to time spent in each phase. If mortality rates per unit time are constant, either haploidy or diploidy is the only stable life cycle possible. Deviations from constant mortality can give qualitatively different results. For example, when survivorship in each phase is a linear, decreasing function of the time spent in the phase, stable haploid, diploid or polyphasic life cycles are possible. In this case, there is a range of intermediate combinations of haploid and diploid selection coefficients that result in stable polyphasic life cycles, with the length of each genetic phase determined by a balance between the strength of selection in each phase.

In the examples considered here, the addition of genetic variation at a coevolving viability locus does not qualitatively affect the outcome with respect to the maintenance of polyphasic cycles. For a given survivorship function, different types of viability selection within a phase appear to simply adjust the range of parameters that support each type of stable life cycle. As shown in the examples, however, a coevolving viability locus can lead to situations where more than one life cycle is concurrently stable (Figure 3). Populations under identical selection pressure may evolve to have different stable life cycles, depending only on the initial state of the population. In addition, results from the antagonistic pleiotropy example are more complex than those of the other types of selection considered in that more than one stable viability locus equilibrium is possible. Patterns of associated stable life cycles show a great deal of variation in this case.

Previous analyses (WRIGHT 1969; CROW and KIMURA 1970; EWING 1977) indicated that when selection favors one viability allele in the haploid phase and another in the diploid phase, evolution can result in a stable polymorphism. This outcome is the result of an implicit assumption that the genetic phases are fixed and cannot evolve. The work presented here shows that if both the life cycle and a trait can evolve, genetic variation for the trait cannot be maintained under this model. An analysis of the values of $\hat{i}$ possible for the antagonistic pleiotropy examples discussed above shows that, for every value of $\hat{i}$ such that $0 \leq \hat{i} \leq 1$, a stable equilibrium exists at either $\hat{p} = 1$ or $\hat{p} = 0$, or both; an equilibrium at $0 < \hat{p} < 1$ cannot also be stable. In all cases considered, both the viability locus and the life cycle evolve until a stable equilibrium is reached at fixation for one or the other viability allele.

The model as presented here assumes that the life cycle is determined completely by the diploid genotype of a life-cycle controlling locus. I have analyzed the case of haploid life cycle control as well, and find that, under certain conditions, the results are equivalent. In the haploid-control case the assumption was made that no interbreeding occurs between haploid gametes of different life cycle genotype; this restriction was made to assure that the total life cycle length remains constant regardless of genotype. The analysis thus reduces to one of comparison of mean fitnesses between reproductively isolated groups and may be biologically less compelling than the diploid-control model developed above.

Some caveats must be added to the conclusions drawn from this model. First, the model assumes that any life cycle ($0 \leq t \leq 1$) is possible, implying that the life cycle that maximizes fitness is attainable. If variation is limited to only certain values of $t$, other equi-
There may exist additional polymorphic equilibria which have not been identified.

Discussion of the evolution of genetic life cycles in the literature involves two different approaches. First, most theoretical work to date considers genetic arguments for the advantages of diploidy over haploidy, without addressing the possibility of polyphasic life cycles. The classical explanation for the evolution of diploidy is that a diploid genome allows for complementation and thus for protection against the expression of deleterious mutations (Muller 1952; Crow and Kimura 1965; Bernstein, Byers and Michod 1981). Three recent studies have introduced theoretical models that aim to determine the conditions under which this explanation will be sufficient. Kondrashov and Crow (1991) studied a mutational load model where selection against the multilocus epistatic effects of deleterious mutations can support either all-haploid or all-diploid life cycles. Their model suggests that truncation selection may result in an advantage to diploidy when deleterious mutations are mostly recessive and when the mutation rate exceeds one deleterious mutation per genome. Perrot, Richerd and Valero (1991) also considered directional selection against a recurring deleterious mutation using a genetic model with a single viability locus and a second locus coding for either an all-haploid or all-diploid life cycle. Results indicated that diploidy is favored under weak selection when mutations are recessive; the stronger the selection against the deleterious mutation, the more recessive its effects must be to result in a diploid advantage. Otto and Goldstein (1992) extended the Perrot, Richerd and Valero model to examine the effect of differing rates of recombination, and found that, while diploids may invade haploid populations when recombination rates are high, they are never able to do so when recombination rates are very low.

The model presented here differs from these previous theoretical studies in two ways. First, the possibility of the retention of both genetic phases in the life cycle is explicitly included here. The previous models have not provided for this possibility; as such, it is not apparent whether the evolutionary forces they discuss could explain the polyphasic life cycles seen in nature. Second, rather than considering selection against continuously recurring deleterious alleles and the potential benefits of diploid complementation, as was done previously, this model focuses on the importance of selection operating on traits expressed in both phases of the life cycle. This selection can take a number of general forms, including selection against deleterious alleles, but the model does not consider recurrent mutations.

The second approach used to study the evolution of genetic life cycles considers ecological and physiological differences between phases as a way of explaining observed patterns. A variety of potential differences have been noted. These include the ability to take advantage of different environments (Willson 1983; Cousens 1988; de Wrede and Kling 1988; Destombe et al. 1989) or different morphological alternatives (Buss 1987), possible advantages in the haploid phase due to potential for faster growth, increased fecundity and wider dispersal (Cavalier-Smith 1978; Willson 1983; Destombe et al. 1989; McLachlan 1991) and the potential for evolution of gamete competition and subsequent high rates of evolution due to efficient selection on the haploid stage (Mulcahy 1979; Cousens 1988). Theoretical treatment of these differences as explanations for observed patterns is incomplete. In a recent study, Richerd, Couvet and Valero (1993) develop a theory for the maintenance of polyphasic cycles based on an additional mechanism. Their model assumes that meiosis and fertilization occur half as often in the polyphasic cycle than in all-haploid or all-diploid cycles. This suggests that polyphasic cycles may have an advantage over haploidy or diploidy through a reduction in the cost of sex. Their results indicate that, if the sex ratio is equal, polyphasic cycles may enjoy a twofold advantage over other cycles.

The model discussed in this paper addresses the impact of ecological differences on the evolution of the life cycle and focuses on the importance of selection operating on traits expressed in both genetic phases. Results indicate that the stable life cycle does depend on the interaction of selective forces across the two phases and may depend critically on the form this selection takes. The conclusions drawn from this work are consistent with the patterns of genetic life cycles found in nature. Haploidy and diploidy are common, but stable life cycles that retain both haploid and diploid phases also exist in a large number of taxa. However, a great deal of further field work is necessary to determine whether selection does operate in any of the ways postulated in this model.

There has been little empirical investigation into the patterns of genetic life cycles found in different temporal or spatial environments. Most comparative research to date considers environmental correlates of the alternation of morphological generations, which may or may not correspond to the alternation of genetic phases (Bold, Alexopoulos and Delvoye-Ryas 1980; Raghavan 1989). Studies that have looked for different genetic life cycles in different environments have found evidence that selection does affect the life cycle. Chlamydomonas species spend differing amounts of time in the diploid stage in different
environments (Sleigh 1989). Many ferns retain only the haploid phase in some locations and the normally alternating polyphasic life cycle in others (Farrar 1967). In many polyphasic algae, the ratio of haploid to diploid individuals varies greatly over time and from one location to another (De Wreede and Klinger 1988; Destombes et al. 1989). Some species of the isomorphic red algae Iridaea show a pronounced seasonal genetic life cycle in higher latitudes but appear to cycle yearly or over several years in lower latitudes (De Wreede and Green 1990).

These observations imply that the interaction of selective forces across the genetic phases of the life cycle can be an important evolutionary force and suggest that such selection may be common. The simple theoretical model presented here suggests that this type of selection may help to explain the patterns of genetic life cycles we encounter in nature. It also indicates that further empirical work is needed, not only on actual patterns of genetic life cycles and their association with different environmental conditions, but also on the ways that selection acts on haploid/diploid organisms.

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LITERATURE CITED


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APPENDIX A

A full two-locus genetic model has been developed to verify the stability of the equilibria found by the optimization analysis described in the text. This is a non-overlapping generations model that assumes a haploid/diploid genetic system, meiotic reproduction and random union of gametes after haploid selection. The life cycle proceeds as shown in Figure 1, with selection acting in both phases. The model assumes there are two genetic autosomal loci, each segregating for two alleles. Locus A is a viability locus that is
expressed in each phase and locus $M$ is a life cycle modifier whose diploid genotype determines the value of $t$.

General notation for fitnesses of the two-locus genotypes are as follows:

<table>
<thead>
<tr>
<th>Haploid</th>
<th>Diploid</th>
</tr>
</thead>
<tbody>
<tr>
<td>$M_1^*$</td>
<td>$M_1^{**}$</td>
</tr>
<tr>
<td>$A_1$</td>
<td>$w_1$</td>
</tr>
<tr>
<td>$A_2$</td>
<td>$w_2$</td>
</tr>
<tr>
<td></td>
<td>$A_2A_2$</td>
</tr>
</tbody>
</table>

where:

$*$ = haploids from homozygote diploid parents ($M_1M_1$ or $M_2M_2$)

$^{**}$ = haploids from heterozygote diploid parents ($M_1M_2$)

Haploid phenotypes, and thus haploid fitnesses, are controlled by the haploid genotype of locus $A$ but by the locus $M$ genotype of the diploid parents. Coupling and repulsion double heterozygotes are assumed to have identical fitness.

The model assumes that the $M_1$ allele is the common allele and that the diploid genotype $M_1M_1$ controls a life cycle described by the $t$ found by the optimization analysis. The two-locus analysis determines whether allele $M_2$ will spread in the population when rare. The frequency of allele $M_2$ is assumed small enough that terms that are quadratic or higher with respect to this frequency can be neglected. The heterozygote $M_1M_2$ can code for any other possible life cycle, that is, $(t + e)$ as a haploid and $1 - (t + e)$ as a diploid (for $-t < e < 1 - t$). The value of $t$ is substituted into the appropriate fitness function from the examples discussed in the text and the $w_i$'s and $w_j$'s calculated.

Let $x_1$, $x_2$, $x_3$, and $x_4$ represent the frequencies of the $M_1A_1$, $M_1A_2$, $M_2A_1$ and $M_2A_2$ gametes at a given generation after haploid selection and $r$ the recombination fraction between the $M$ and $A$ locus. Then the gamete frequencies the next generation are:

\[
\begin{align*}
  x'_1 &= w_1(w_1x_1^2 + w_2x_2x_3) + w_3(w_1x_1x_3 + w_2x_2x_4) - \frac{r}{W}w_1w_4x_1x_4 \\
  x'_2 &= w_2(w_1x_1x_2 + w_2x_2^2) + w_4(w_1x_1x_3 + w_2x_2x_4) + \frac{r}{W}w_1w_4x_1x_4 \\
  x'_3 &= w_3(w_3x_1^2 + w_4x_3x_4) + w_4(w_1x_1x_3 + w_2x_2x_4) + \frac{r}{W}w_1w_4x_1x_4 \\
  x'_4 &= w_4(w_3x_1x_4 + w_4x_4^2) + w_4(w_1x_1x_4 + w_2x_2x_4) - \frac{r}{W}w_1w_4x_1x_4
\end{align*}
\]

where

$D = x_1x_4 - x_2x_3$ and

$W = \text{sum of the numerators}$.

The values of the selection coefficients and of $t$ were determined for each special case as discussed in the text. The two-locus fitnesses were then substituted into recursion equations (A1) and standard linear stability analysis was performed. I verified the stability of all equilibria discussed in the text using this approach.