Ecology and the Evolution of Biphasic Life Cycles

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ABSTRACT: Sexual eukaryotes undergo an alternation between haploid and diploid nuclear phases. In some organisms, both the haploid and diploid phases undergo somatic development and exist as independent entities. Despite recent attention, the mechanisms by which such biphasic life cycles evolve and persist remain obscure. One explanation that has received little theoretical attention is that haploid-diploid organisms may exploit their environments more efficiently through niche differentiation of the two ploidy phases. Even in isomorphic species, in which adults are morphologically similar, slight differences in the adult phase or among juveniles may play an important ecological role and help maintain haploid-diploidy. We develop a genetic model for the evolution of life cycles that incorporates density-dependent growth. We find that ecological differences between haploid and diploid phases can lead to the evolution and maintenance of biphasic life cycles under a broad range of conditions. Parameter estimates derived from demographic data on a population of *Gracilaria gracilis*, a haploid-diploid red alga with an isomorphic alternation of generations, are used to demonstrate that an ecological explanation for haploid-diploidy is plausible even when there are only slight morphological differences among adults.

Keywords: haploidy, diploidy, alternation of generations, *Gracilaria*.

An alternation between haploid and diploid nuclear phases is a necessary consequence of eukaryotic sexuality. Variation in the relative timing of meiosis and syngamy allows organisms to vary widely in the size and duration of these two phases. In diplonts, the haploid phase is limited to one or a few cells and undergoes little, if any, development. Similarly, in haplonts the diploid phase is limited, and vegetative growth occurs primarily in the haploid phase. Many algae, ferns, moss, and fungi have a biphasic life cycle in which both the haploid and diploid phases undergo substantial development (Bell 1994). We refer to such biphasic organisms as haploid-diploids to avoid confusion with the term haplodiploidy, which is commonly used to refer to species with haploid males and diploid females. Phycologists have also used the terms "diplohaploid" and "diplohaplontic" to refer to biphasic life cycles.

In land plants and higher animals, there is an evolutionary trend toward increased dominance of the diploid phase, and much of the early thinking about ploidy evolution focused on finding advantages of diploidy that would explain this trend (reviewed in Valero et al. 1992; Mable and Otto 1998). There are several classic arguments for the adaptive benefit of diploidy. Since deleterious mutant alleles are generally rare, a sexual diploid is unlikely to carry two copies. Thus, diploids can mask the effects of most deleterious mutations by compensating with a second, normal allele (Crow and Kimura 1965). Furthermore, masking allows deleterious mutations to be retained over longer periods of time, providing genetic variability that may prove advantageous in the event of environmental change (Raper and Flexer 1970). In addition, since diploids carry twice as much DNA as haploids, they might be expected to accumulate new beneficial mutations at a higher rate (Paquin and Adams 1983). Diploids may also evolve more rapidly because they carry "extra" alleles that can evolve new functions, while the old alleles continue to perform their original functions (Lewis and Wolpert 1979).

However, many extant taxa are exclusively haploid or undergo significant development in both phases. It also appears that the haploid phase has increased in dominance over time in some taxa (Bell 1997). Hypotheses that predict an adaptive benefit only to diploidy are unable to satisfactorily explain the evolution and persistence of haplonts and haploid-diploids, so attention has turned to understanding the maintenance of a diversity of life cycles (Mable and Otto 1998). Recent theoretical work has shown that although masking may give diploids an advantage, it need not always be so. Deleterious mutations tend to be purged more rapidly from haploid populations because they are not masked. Under certain conditions, this may select for an expansion of the haploid phase (Kondrashov and Crow 1991; Perrot et al. 1991; Otto and Goldstein 1992; Jenkins and Kirkpatrick 1995; Otto and Marks 1996).

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Furthermore, diploids do not always evolve faster. Although beneficial mutations arise more frequently in diploids, they are often partially masked, which reduces the effectiveness of selection and decreases the chance that they will be incorporated within a diploid population (Orr and Otto 1994).

Since these genetic arguments can be shown to favor haploidy in some situations and diploidy in others, it seems reasonable to expect that in some cases these influences will balance out and a biphasic life cycle will be favored. However, most genetic models have not fulfilled this expectation, leaving unanswered the question of what accounts for life-cycle diversity in general, and haploid-diploidy in particular (see Willson 1981; Valero et al. 1992; Klinger 1993; Bell 1994; Mable and Otto 1998 for perspectives and reviews). An explanation that has yielded more promising results is that haploid-diploidy may reduce the cost of sex. If the duration of the life-cycle phases are equal, then haploid-diploids will have sex half as often as either haplonts or diplonts. Richerd et al. (1993, 1994) showed that this intrinsic advantage can cause biphasic life cycles to be favored wherever the cost of sex is high. However, in such situations one might reasonably expect that this cost could be reduced through the evolution of asexual reproduction (Mable and Otto 1998).

Most other attempts to account for haploid-diploidy concern potential ecological advantages of biphasic life cycles (Stebbins and Hill 1980; Willson 1981). The purpose of this article is to examine the theoretical validity and possible relevance of the hypothesis that niche differences between haploid and diploid phases may cause the evolution of biphasic life cycles to be favored.

Many multicellular haploid-diploid organisms are heteromorphic; that is, the haploid and diploid phases of their life cycles differ in morphology—and presumably also in physiology and ecology. In algae, one phase may be small and resistant to environmental stresses such as grazing or desiccation, while the other may be large and well adapted to exploit favorable environmental conditions (Klinger 1993). This observation has led to the idea that biphasic life cycles may exploit a broader range of environmental niches (Willson 1981), which could cause haploid-diploid life cycles to be favored, especially in environments that vary over space or time.

A major criticism of this idea has been that the haploid and diploid phases of several organisms are isomorphic, with little distinguishing the two phases (Valero et al. 1992; Klinger 1993). Thus, while the advantage conferred by having more than one phenotype could lead to the maintenance of haploid-diploidy once differences in morphology between haploid and diploid phases had evolved, it is difficult to explain how haploid-diploidy could arise or be maintained in isomorphic species.

This difficulty may be more apparent than real, however. First, it is not clear that isomorphic phases are ecologically identical. Classification is, in most cases, based on gross morphological characteristics, and differences in survival and resource use could easily be missed. In those cases in which life-history parameters have been measured in the field, significant ecological differences have been found between the two phases of isomorphic species (e.g., Destombe et al. 1993; Dyck and DeWreede 1995).

A second point is that even if adult haploids and diploids are ecologically identical, their unicellular propagules need not be. Algal propagules display considerable diversity in characters such as cell size, motility, energy reserves, and the presence of mucilage (Clayton 1992). In particular, diploid cells are often larger than their haploid counterparts as a consequence of the fact that diploids have twice as much DNA and thus have larger nuclei that can direct the production of more protein (Adams and Hansche 1974). Cavalier-Smith (1978) drew attention to the fact that this difference in size may have direct physiological and ecological consequences. Large cells have lower surface area to volume ratios than small ones of the same shape and often have longer division times as well. In multicellular organisms, these differences can be minimized by adjusting the total number of cells so that gross morphology is maintained. However, for single cells, such as gametes, spores, or zygotes, or for juveniles with few cells, these differences in geometry can have major consequences for reproductive, competitive, and dispersal abilities (Cavalier-Smith 1978; Lewis 1985; Destombe et al. 1992).

In order for biphasic life cycles to be favored, it does not matter what the particular differences between the two ploidy phases are, only that they are sufficiently different to exploit an environment more efficiently together than either could alone. However, the tendency for diploids to be larger is particularly interesting because it seems to be a direct consequence of ploidy. Any differences that are not a direct consequence must evolve after the two phases are established. Thus, while they might be able to account for the maintenance of a biphasic life cycle, they could not account for its initial evolution.

Here, we develop a model that investigates the evolution of genes that control the life cycle of an organism with ecological differences between haploid and diploid phases. We use life-history data from the marine red alga *Gracilaria gracilis* (Stackhouse) Steentoft (Destombe et al. 1989, 1992, 1993; renamed from *Gracilaria verrucosa* by Steentoft et al. 1995) to explore whether an ecological hypothesis can explain the maintenance of biphasic life cycles even in species, such as *G. gracilis*, with an isomorphic alternation of generations.

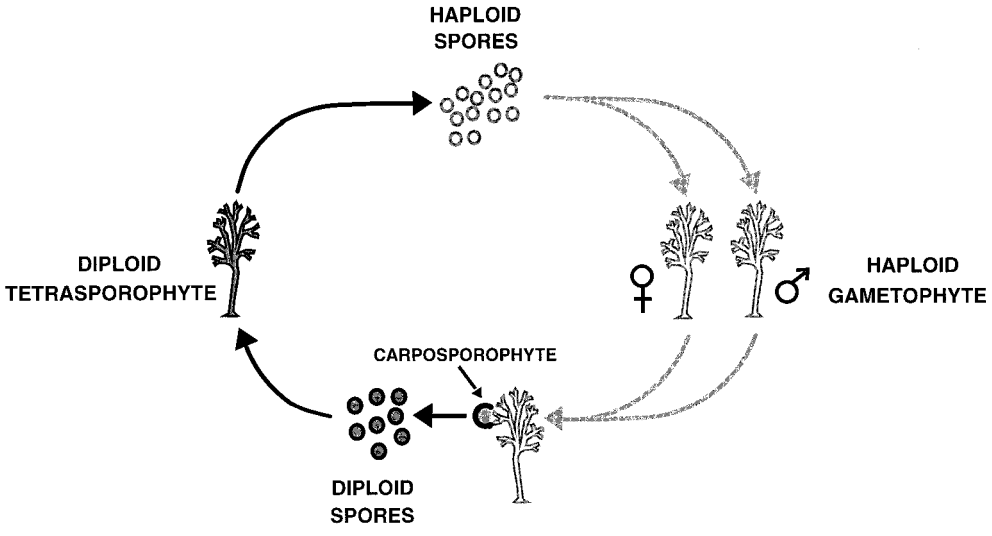

Figure 1: Life cycle of *Gracilaria gracilis*. Diploid tetrasporophytes release haploid spores, which grow into independent haploid gametophytes. Male gametophytes release gametes into the water column. Females retain their gametes, and fertilization occurs on the female gametophyte. Each diploid zygote develops into a small vegetative carposporophyte, from which many identical diploid tetraspores are released. These spores grow into independent tetrasporophytes to complete the cycle (after Destombe et al. 1989). Diploid structures are shaded, and haploid structures are clear.

Methods

Competition and Life-Cycle Evolution

To model growth, survival, and reproduction of haploid-diploid populations in environments with limited resources, we adapt a discretized version of the Lotka-Volterra competition model for two species (Roughgarden 1996). The main difference between our model and the Lotka-Volterra model is that the two competing forms, haploids and diploids, are linked through sexual reproduction, rather than separate species. That is, in a haploid-diploid population, haploids give rise to diploid zygotes, and diploids give rise to haploid spores. Another difference is that we consider a perennial population with overlapping generations and allow adults to survive from census to census with some probability ($1 - d_h$ for haploids and $1 - d_d$ for diploids). An annual population can easily be studied by setting $d_h = d_d = 1$ in the model. It has been pointed out that algae may interact by means other than competition (Paine 1990). For example, spores of some species can coalesce to form one cytoplasmically continuous plant, which may have competitive advantages (Santelices et al. 1996). Unfortunately, there is not enough known about the frequency and effect of this phenomena for us to consider it explicitly, so in our model we have ignored this and other possible noncompetitive interactions.

To allow the extent of the haploid and diploid phases to evolve, we include in our model a genetic locus that alters the life cycle. Specifically, the genotype of an adult determines whether it will undergo a regular alternation of generations or a contracted epiphytic form of the next phase and subsequently produce propagules that have the same ploidy level as the adult. In other words, the life-cycle locus controls whether haploid adults undergo a haploid-diploid or a haplontic life cycle and whether diploid adults undergo a haploid-diploid or a diplontic life cycle. This model is motivated by typical and variant life cycles observed in *Gracilaria gracilis*.

Gracilaria is a perennial haploid-diploid alga with an isomorphic alternation of generations (fig. 1). In the typical life cycle, haploid gametophytes are free-living male or female plants. Male gametophytes release nonmotile sperm directly into the water column, some of which fertilize female gametes retained on the female parent. After fertilization, the diploid zygote develops into a small diploid carposporophyte attached to the female gametophyte. Each carposporophyte releases many identical diploid spores. Those spores develop into free-living sessile diploid adults, known as tetrasporophytes. Adult tetrasporophytes release millions of haploid spores, which give rise to independent haploid gametophytes, thus completing the cycle (Destombe et al. 1989).

In their studies on this species, Destombe et al. (1989) noted that approximately 5% of diploid individuals display "rare sexual phenotypes," which have the common feature that they effectively allow diploids to skip the haploid phase of their life cycle to some degree. Similar phenomena

have been observed among other red algal species (Tokida and Yamamoto 1965; Bird et al. 1977). In these variant life cycles, male and/or female haploid spores were retained on the diploid plant and germinated there to produce haploid reproductive organs, which subsequently released gametes. In some plants, the retained spores underwent a reduced vegetative stage before reproducing. These small epiphytic haploid gametophytes grew on fronds of the parent diploid. These variants essentially had diplontic life cycles, wherein diploid adults contained within them the haploid phase and hence were able to produce haploid gametes. Interestingly, the expression of these phenotypes was unstable over time, with the same individual displaying a rare sexual phenotype at one time and the normal phenotype at others. The degree to which this capacity is genetically determined is unknown, but these observations indicate that strict developmental constraints do not exist that would limit genetic variation for life-cycle type. In this article, we examine the fate of any mutation that would alter the capacity of haplontic, diplontic, and haploid-diploid growth within a population.

We focus on the evolution of biphasic life cycles. However, phycologists often refer to the life cycle of *Gracilaria* as "triphasic" because of the elaboration of the carposporophyte phase (fig. 1). This reduced phase produces many clonal diploid spores from each zygote, increasing the reproductive output per fertilization event at the cost of decreasing the genetic variation among progeny. This might affect the values of life-history parameters, increasing adult fecundity and/or altering juvenile survival. However, it has no effect on the structure of our model or the qualitative results derived from it.

Model in the Absence of Life-Cycle Variation

Consider a perennial population with an alternation of overlapping generations in which juveniles mature by the next breeding season (fig. 2). For clarity, we assume that reproduction occurs in late summer and fall, as in *Gracilaria* (Destombe et al. 1989), and that adult mortality occurs in the winter and spring. Competition for resources could potentially affect juvenile survival, adult survival, or reproduction. Although all of these processes are density dependent to some extent, we assume for simplicity that competition among juveniles for settlement sites is the major locus of density dependence. We census in early summer, after adult mortality and before reproduction. The number of haploid individuals alive at census (H') will equal the number alive last year (H), minus deaths, plus new recruits:

$$H' = H - d_{\mathrm{h}}H + \text{haploid recruits.} \tag{1a}$$

Similarly, the number of diploids (D') will be:

$$D' = D - d_{\mathrm{d}}D + \text{diploid recruits.} \tag{1b}$$

The number of recruits will equal the number of spores released per year (f_{d} from diploid adults and f_{h} from haploid adults) times the probability of establishment of the spores. Fecundity (the number of spores released per individual) is assumed to be density independent, although the results do not depend qualitatively on this assumption. Initially, we assume that the life-cycle locus is fixed on allele M such that diploids skip the haploid phase (diplontic development) with probability p_{MM} and undergo a regular alternation of generations with probability $1 - p_{MM}$. Similarly, haploids skip the diploid phase (haplontic development) with probability p_M and undergo a regular alternation of generations with probability $1 - p_M$. A purely haploid-diploid life cycle would therefore be characterized by $p_{MM} = p_M = 0$, while a purely diplontic population would have $p_{MM} = 1$, and a purely haplontic population would have $p_M = 1$. We let $f_{\mathrm{h}}x_{\mathrm{h}}$ equal the fecundity of skipping haploids and $f_{\mathrm{d}}x_{\mathrm{d}}$ equal the fecundity of skipping diploids, where x_{h} and x_{d} measure the effect on fertility of skipping the regular alternation of generations (fig. 2). Consequently, the total number of haploid and diploid spores produced within the population equals

$$\text{haploid spores} = Df_{\mathrm{d}}(1 - p_{MM}) + Hf_{\mathrm{h}}x_{\mathrm{h}}p_M, \tag{2a}$$

$$\text{diploid spores} = Hf_{\mathrm{h}}(1 - p_M) + Df_{\mathrm{d}}x_{\mathrm{d}}p_{MM}. \tag{2b}$$

Since juvenile haploids compete with both diploids and other haploids for establishment, the survival of haploid juveniles will depend on the abundance of both. The maximum survival of spores in the absence of competitors is s_{h} for haploids and s_{d} for diploids. The survival rate of spores decreases as the number of spores increases, until a juvenile carrying capacity is reached (K_{jh} for haploids and K_{jd} for diploids). The efficacy by which diploids compete for haploid resources is α_{hd}, and the efficacy by which haploids compete for diploid resources is α_{dh}. Assuming logistic density dependence,

$$\text{haploid recruits} = s_{\mathrm{h}}(\text{haploid spores}) \times \left[1 - \frac{(\text{haploid spores} + \alpha_{\mathrm{hd}}\text{diploid spores})}{K_{\mathrm{jh}}}\right], \tag{3a}$$

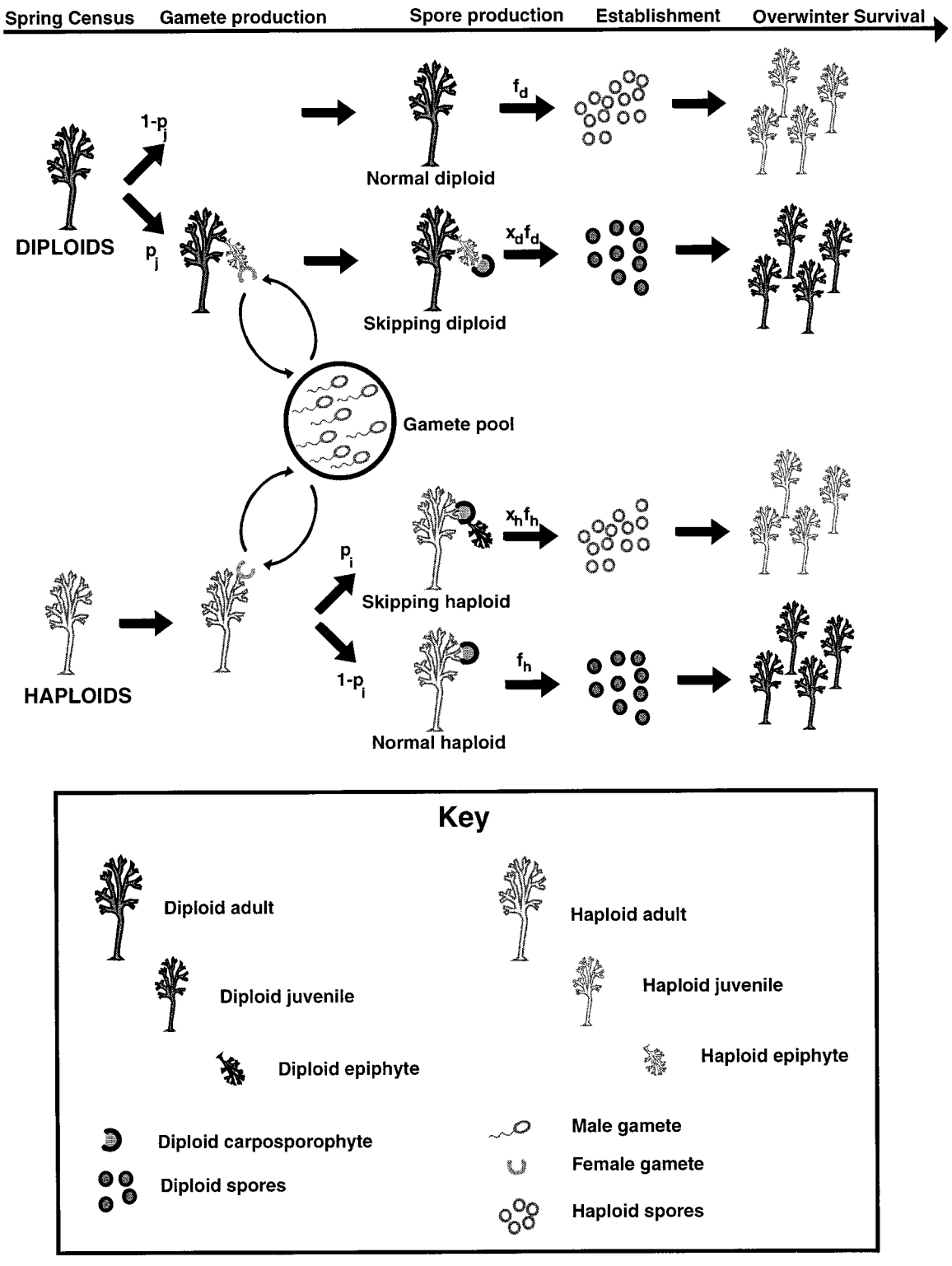

Figure 2: Model life cycle. In early summer, adult diploids produce haploid spores. Some proportion, p_j (j being the diploid genotype *MM*, *Mm*, or *mm*), of diploids may skip the haploid phase of their life cycle by retaining their haploid spores. These spores grow into haploid epiphytes, which subsequently produce and release diploid spores. Thus, skipping diploid individuals give rise to diploids, rather than haploids. In a similar way, some proportion, p_i (i being the haploid genotype *M* or *m*), of haploids may skip the diploid phase. Diploid gametophytes retained on the haploid adult grow into small diploid epiphytes, which release haploid spores. The total number of haploid spores produced is the sum of those released by normal diploids ($f_d D_j[1 - p_j]$) and by skipping haploids ($f_h x_h H_i p_i$). Similarly, diploid spores are produced by both normal haploids ($f_h H_i[1 - p_i]$) and skipping diploids ($f_d x_d D_j p_j$).

$$\text{diploid recruits} = s_{\text{d}}(\text{diploid spores}) \times \left[1 - \frac{(\text{diploid spores} + \alpha_{\text{dh}}\text{haploid spores})}{K_{\text{jd}}}\right]. \quad (3b)$$

Equations (1a)–(3b) fully specify the dynamics of a perennial population of haploids and diploids with resource competition among juveniles. (See table 1 for a complete summary of the terms used in the model.) We have also developed an analogous model with competition among adults, but the results are qualitatively similar and are not reported here.

Note that if the population is haplontic, so that haploid adults give rise directly to haploids ($p_M = 1$), and diploids are absent ($D = 0$), the number of haploids in the next census is

$$H' = H(1 - d_{\text{h}}) + s_{\text{h}} H f_{\text{h}} x_{\text{h}} \left(1 - \frac{H f_{\text{h}} x_{\text{h}}}{K_{\text{jh}}}\right). \quad (4)$$

In this case, we define the equilibrium number of adult haploids as K_{h}, which is the carrying capacity of adult haploids in the absence of diploids when juvenile survival is density dependent. Solving for the equilibrium of equation (4), we find that K_{h} (the adult carrying capacity) is related to K_{jh} in the following way:

$$K_{\text{h}} = K_{\text{jh}} \left(\frac{f_{\text{h}} x_{\text{h}} s_{\text{h}} - d_{\text{h}}}{f_{\text{h}}^2 x_{\text{h}}^2 s_{\text{h}}}\right). \quad (5a)$$

Similarly, the adult diploid carrying capacity is related to K_{jd}:

$$K_{\text{d}} = K_{\text{jd}} \left(\frac{f_{\text{d}} x_{\text{d}} s_{\text{d}} - d_{\text{d}}}{f_{\text{d}}^2 x_{\text{d}}^2 s_{\text{d}}}\right). \quad (5b)$$

For general life cycles, the recursions (1a) and (1b) can be iterated or solved numerically to determine the equilibrium state.

Introducing Life-Cycle Variation

Once a population has reached equilibrium with the *M* life-cycle allele fixed, we introduce a new life-cycle allele, *m*, that alters the probability of haplontic development in haploids to p_m, the probability of diplontic development in heterozygous diploids to p_{Mm}, and the probability of diplontic development in homozygous diploids to p_{mm}. The recursions become

$$H_i' = H_i(1 - d_{\text{h}}) + s_{\text{h}}(\text{spores}_i) \times \left(1 - \frac{\text{haploid spores} + \alpha_{\text{hd}}\text{diploid spores}}{K_{\text{jh}}}\right) \quad (6a)$$

Table 1: Parameters used in the model

Symbol	Interpretation
H	Number of adult haploids in population
D	Number of adult diploids in population
d_{h}	Annual death rate of adult haploids
d_{d}	Annual death rate of adult diploids
s_{h}	Maximum survival rate of haploid juveniles
s_{d}	Maximum survival rate of diploid juveniles
f_{h}	Average number of spores produced by a normal haploid
f_{d}	Average number of spores produced by a normal diploid
x_{h}	Relative fecundity of skipping haploids to normal haploids
x_{d}	Relative fecundity of skipping diploids to normal diploids
p_i	Proportion of haploids of genotype i (i being M or m) that skip the diploid phase
p_j	Proportion of diploids of genotype j (j being MM, Mm, or mm) that skip the haploid phase
K_{jh}	Juvenile haploid density dependence factor
K_{jd}	Juvenile diploid density dependence factor
K_{h}	Adult haploid carrying capacity
K_{d}	Adult diploid carrying capacity
α_{dh}	Competitive effect of haploid juveniles on diploid juveniles
α_{hd}	Competitive effect of diploid juveniles on haploid juveniles

for the number of haploids of genotype i (i being M or m) and

$$D_j' = D_j(1 - d_d) + s_d(\text{spores}_j) \times \left(1 - \frac{\text{diploid spores} + \alpha_{dh}\text{haploid spores}}{K_{jd}}\right) \quad (6b)$$

for the number of diploids of genotype j (j being MM, Mm, or mm), where

$$\text{spores}_M = f_d\left[D_{MM}(1 - p_{MM}) + \frac{1}{2}D_{Mm}(1 - p_{Mm})\right] + f_h x_h\left[H_M p_M\left(p + \frac{q}{2}\right) + H_m p_m \frac{p}{2}\right], \quad (7a)$$

$$\text{spores}_m = f_d\left[D_{mm}(1 - p_{mm}) + \frac{1}{2}D_{Mm}(1 - p_{Mm})\right] + f_h x_h\left[H_M p_M \frac{q}{2} + H_m p_m\left(\frac{p}{2} + q\right)\right], \quad (7b)$$

$$\text{haploid spores} = \text{spores}_M + \text{spores}_m, \quad (7c)$$

$$\text{spores}_{MM} = p\left[f_h H_M(1 - p_M) + f_d x_d\left(D_{MM}p_{MM} + \frac{1}{2}D_{Mm}p_{Mm}\right)\right], \quad (7d)$$

$$\text{spores}_{Mm} = q\left[f_h H_M(1 - p_M) + f_d x_d\left(D_{MM}p_{MM} + \frac{1}{2}D_{Mm}p_M\right)\right] + p\left[f_h H_m(1 - p_m) + f_d x_d\left(D_{mm}p_{mm} + \frac{1}{2}D_{Mm}p_{Mm}\right)\right], \quad (7e)$$

$$\text{spores}_{mm} = q\left[f_h H_m(1 - p_m) + f_d x_d\left(D_{mm}p_{mm} + \frac{1}{2}D_{Mm}p_{Mm}\right)\right], \quad (7f)$$

$$\text{diploid spores} = \text{spores}_{MM} + \text{spores}_{Mm} + \text{spores}_{mm}. \quad (7g)$$

In these equations, we have assumed that haploid eggs are fertilized by sperm from a randomly mixed gamete pool. Haploids and diplontic individuals contribute to the sperm pool in amounts proportional to their fecundity. Consequently, the frequency of M and m gametes in the sperm pool are p and q (which is $1 - p$), respectively, with

$$p = \frac{f_h H_M + f_d x_d[D_{MM}p_{MM} + (1/2)D_{Mm}p_{Mm}]}{f_h(H_M + H_m) + f_d x_d(D_{MM}p_{MM} + D_{Mm}p_{Mm} + D_{mm}p_{mm})}. \quad (8)$$

If haploid-diploidy is favored then, when it arises at low frequency in a population that is predominantly either haplontic or diplontic, it will spread. Our main interest is in determining these invasion criteria, which indicate the conditions under which resource competition can favor the evolution and maintenance of haploid-diploid life cycles. We also determine which life cycle is an evolutionarily stable strategy (ESS) such that, if allele M causes a population to be at ESS, no mutant life-cycle allele can invade.

Results

Conditions for Population Viability

All populations of interest in this analysis must be viable; that is, they must have the capacity to increase at low population densities. This is equivalent to requiring that for each single population, the equilibrium at which no individuals are present is unstable (see appendix for details). Any population is viable when the following condition is satisfied:

$$d_h d_d - d_h p_{MM} f_d s_d x_d - d_d p_M f_h s_h x_h < f_h s_h f_d s_d[(1 - p_M)(1 - p_{MM}) - p_M p_{MM} x_h x_d]. \quad (9a)$$

In particular, a haplontic population ($p_M = 1$, $D_{MM} = 0$) is viable when its maximum recruitment rate is higher than its death rate:

$$d_h < f_h x_h s_h. \quad (9b)$$

Similarly, the condition for viability of a diplontic population ($p_{MM} = 1$, $H_M = 0$) is

$$d_d < f_d x_d s_d. \tag{9c}$$

Note that as long as $x_h \leq 1$ and $x_d \leq 1$, the conditions for viability of haplontic and diplontic populations (eqq. [9b], [9c]) imply that any haploid-diploid population would also be viable (9a).

In our invasion analysis, we will further assume that the population of haplonts (or diplonts) is at equilibrium when a new life-cycle allele arises. A haplontic population reaches a stable equilibrium abundance only if

$$f_h x_h s_h - d_h < 2. \tag{10a}$$

Similarly, a diplontic population reaches a stable equilibrium if

$$f_d x_d s_d - d_d < 2. \tag{10b}$$

These stability conditions are exactly analogous to those of the logistic model in discrete time, in which the intrinsic growth rate must be >0 and <2 for an equilibrium with a positive number of individuals to be stable (Roughgarden 1996). With higher intrinsic growth rates, the dynamics of populations near carrying capacity are unstable and may become chaotic because of time lags in the discrete model.

Conditions for Haploid-Diploid Invasion

Into a population that is fixed on allele M and that is either haplontic ($p_M = 1$, $D_{MM} = 0$) or diplontic ($p_{MM} = 1$, $H_M = 0$), a mutation is introduced that causes an alternation of generations (p_M, $p_{MM} < 1$). We determined the conditions under which this haploid-diploid life-cycle allele can invade the haplontic or diplontic population at carrying capacity. The analysis is described in the appendix, where general results are presented. Here we focus on the case in which the relative fecundities of normal and skipping individuals are equal ($x_d = 1$, $x_h = 1$). In this case, the competitive consequences of haploid-diploidy are not confounded by intrinsic differences in fecundity.

A haploid population ($p_M = 1$, $D_{MM} = 0$) can be invaded by an allele causing a haploid-diploid life cycle ($p_m < 1$) whenever

$$f_d K_d > \alpha_{dh} f_h K_h. \tag{11a}$$

When this condition is met, the survival rate of diploid spores (given by [A3] with $x_d = x_h = 1$) times the fecundity of diploids (f_d) is greater than the death rate of diploids (d_d), allowing diploidy to become established within the population. Similarly, a diploid population ($p_{MM} = 1$, $H_M = 0$) may be invaded by a haploid-diploid life cycle ($p_{Mm} < 1$) whenever

$$f_h K_h > \alpha_{hd} f_d K_d. \tag{11b}$$

If both of these conditions are satisfied, haploid-diploid life cycles are selectively favored over both haplontic and diplontic life cycles. In this case, ecological niche separation is able to maintain a biphasic life cycle. Notice that even if haploids and diploids have identical fecundities and carrying capacities, haploid-diploidy will be favored as long as haploids and diploids experience less intense competition with members of the opposite ploidy level than with members of their own (α_{dh}, $\alpha_{hd} < 1$).

Comparison shows that these conditions are similar in form to the familiar results of the Lotka-Volterra competition equations. According to that model, two competing species, 1 and 2, are expected to coexist if the following conditions are met:

$$K_1 > \alpha_{1,2} K_2, \tag{12a}$$

$$K_2 > \alpha_{2,1} K_1, \tag{12b}$$

where K_1 and K_2 are the carrying capacities of species 1 and 2, $\alpha_{1,2}$ is the relative competitive impact of species 2 on 1, and $\alpha_{2,1}$ is the competitive impact of species 1 on 2. The major difference between our model of competition between life-cycle phases and this traditional model of competition is that haploid and diploid phases are intimately linked through reproduction, while the species considered by Lotka-Volterra are reproductively isolated. It appears, however, that the intricacies of linked reproduction have little confounding effect on the essential effect of competition. If an environment may be exploited more efficiently by two different entities, then these two entities can coexist, whether they are separate species or haploid and diploid phases of the same species.

Evolutionarily Stable Strategy Life Cycle

In the above section, we have specified the conditions under which a haploid-diploid population is able to invade a population composed entirely of haploids or of diploids. In addition, we would like to know whether, for a given set of conditions, a life cycle exists that cannot be invaded by any other life-cycle type. Such a life cycle would represent an evolutionarily stable strategy (ESS), which is "a strategy such that, if all the members of a population adopt it, no mutant strategy can invade" (Maynard Smith 1982, p. 204). When the relative fecundities of normal and skip-

ping individuals is equal ($x_d = 1$, $x_h = 1$), the ESS can be determined explicitly (see appendix for details of the analysis). A population that is fixed on an allele, *M*, such that

$$\hat{p}_{MM} = 1 - \frac{\hat{H}_M f_h (1 - \hat{p}_M)}{\hat{D}_{MM} f_d}, \tag{13}$$

and that has reached the following equilibrium:

$$\hat{H}_M = \frac{f_h K_h - \alpha_{hd} f_d K_d}{f_h (1 - \alpha_{hd}\alpha_{dh})}, \tag{14a}$$

$$\hat{D}_{MM} = \frac{f_d K_d - \alpha_{dh} f_h K_h}{f_d (1 - \alpha_{hd}\alpha_{dh})}, \tag{14b}$$

cannot be invaded by any other life-cycle allele. At this ESS, the number of haploid spores produced is exactly $f_h \hat{H}_M$, and the survival rate of haploid spores is d_h/f_h. This means not only that the number of haploid recruits equals the number of haploid deaths per census (which must be true at equilibrium) but also that the number of haploid spores produced by the population is the same as if all haploids were haplontic, which need not be the case. The same applies to diploids. Notice that the ESS is biologically valid only if $0 \le \hat{H}_M$, $0 \le \hat{D}_{MM}$, and $0 \le (\hat{p}_M, \hat{p}_{MM}) \le 1$. Given these restrictions, any combination of $\hat{p}_M$ and $\hat{p}_{MM}$ that satisfies equation (13) is an ESS. In particular, a population composed of a mixture of haplonts and diplonts (with $\hat{p}_M = \hat{p}_{MM} = 1$) is always an ESS. This suggests an important point to keep in mind: if resources available to haploids and diploids are sufficiently distinct that a haploid-diploid population can be maintained, genetically isolated but sympatric populations of haplonts and diplonts would also be able to utilize the resources to the full extent possible.

The Life Cycle of Gracilaria*: A Numerical Example*

To illustrate the behavior of this model, we iterate recursions (6a) and (6b) to simulate life-cycle evolution, using demographic information on *Gracilaria* to derive estimates of parameter values wherever possible (summarized in table 2). Several parameters remain unknown and have to be chosen. In particular, the effects of competition on haploid and diploid juvenile survival have not been measured. From equation (11a) and (11b), depending on the exact parameter values, we expect the life cycle of a population to evolve toward (a) diplonty, (b) haploid-diploidy, (c) either haplonty or diplonty depending on initial population composition, and (d) haplonty, depending on the exact parameter values. Using the parameters given in table 2, a haploid-diploid population can invade a haplontic population when $\alpha_{dh} < 4/3$, and it can invade a diplontic population when $\alpha_{hd} < 3/4$. To explore all four possible outcomes of life-cycle evolution, we chose the following competition coefficients: (a) $\alpha_{dh} = 0.9$ and $\alpha_{hd} = 0.9$, (b) $\alpha_{dh} = 0.5$ and $\alpha_{hd} = 0.5$, (c) $\alpha_{dh} = 1.5$ and $\alpha_{hd} = 1.0$, and (d) $\alpha_{dh} = 1.5$ and $\alpha_{hd} = 0.5$. In (a), we found that a haplontic population could be invaded and fully replaced by a diplontic population (fig. 3), but that a diplontic population was stable to invasion. In (b), we found that both

Table 2: Parameters used in the simulations

Parameter	Estimate
d_h[a]	.078
d_d[a]	.062
f_h[b,c]	$6\ 10^6$
f_d[b]	$8\ 10^6$
H survival to 1 yr[d]	$2.4\ 10^{-4}$
D survival to 1 yr[d]	$2.2\ 10^{-3}$
s_h[e]	$(2.4\ 10^{-8})$
s_d[e]	$(2.2\ 10^{-7})$
x_h[f]	(1)
x_d[f]	(1)
K_h[g]	(300)
K_d[g]	(300)
K_{jh}[h]	$(3.9\ 10^9)$
K_{jd}[i]	$(2.5\ 10^9)$

Note: Parameters were estimated from studies of the red alga *Gracilaria* whenever possible (values in parentheses were not).

[a] Destombe et al. 1989.

[b] C. Destombe and M. Valero (personal communication).

[c] Destombe 1987.

[d] The survival rate of spores to fixation times the survival rate of fixed spores to 1 yr from laboratory experiments of Destombe et al. 1989. Note that Destombe et al. (1989) did not consider density-dependent effects, so their estimates of juvenile survival are likely to be somewhat lower than the maximum survival of spores in the absence of all competition.

[e] Measured survival of spores to 1 yr times 10^{-4} to estimate lowered survival under natural conditions and to insure that the conditions of population viability (9a), (9b), (9c), (10a), and (10b) are met.

[f] x_h and x_d were set to 1 to ensure that there were no intrinsic fecundity differences between haplontic, diplontic, and haploid-diploid life cycles.

[g] We assume here that the carrying capacities of the adult haploid and diploid *Gracilaria* are equal ($K_h = K_d$). Results (11a), (11b), (13), (14a), and (14b) scale with the carrying capacity, so that changing the magnitude of both K_h and K_d would not affect the evolutionary outcome of the simulations. Changing their relative size, however, would affect the outcome. If K_h is larger than K_d, then we expect haploidy to evolve more readily within the population and vice versa.

[h] From equation (5a).

[i] From equation (5b).

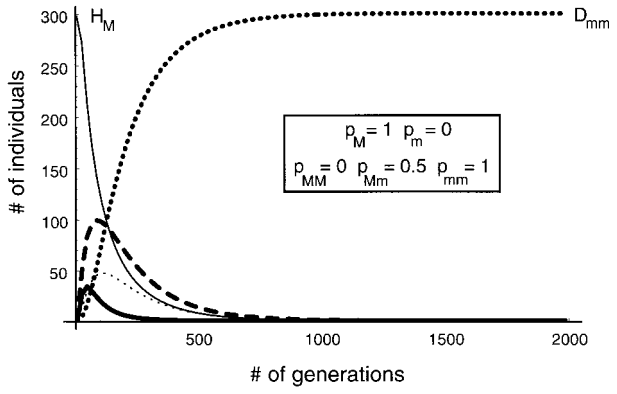

Figure 3: Evolution of diplonty. With $\alpha_{dh} = 0.9$ and $\alpha_{hd} = 0.9$, a haplontic population ($H_M = 300$, $D_{MM} = 0$) can be invaded by any life-cycle allele that increases the number of diploids within the population. In particular, the *M* allele can be fully replaced by an allele *m* that causes the population to become diplontic, as illustrated ($p_m = 0$, $p_{mm} = 1$). Parameters are given in table 2. Thick curves represent diploids, while thin curves represent haploids. Solid curves represent *M* and *MM* genotypes, the dashed curve represents the *Mm* genotype, and dotted curves represent the *m* and *mm* genotypes.

haplontic and diplontic populations would be invaded by a haploid-diploid life cycle (fig. 4); furthermore, allele *m* would fix in both cases if it caused the population to be at ESS. In (c), an allele encoding the ESS (13)–(14b) was unable to invade either a haplontic population or a diplontic population, demonstrating that the ESS may be unattainable even if it is theoretically valid (see appendix for more details). Finally, in (d), a diplontic population could be invaded and fully replaced by a haplontic population (fig. 5), but a haplontic population was stable to invasion. In each case, we assumed that the life-cycle allele was additive, $p_{Mm} = (p_{MM} + p_{mm})/2$, although the final outcome of life-cycle evolution did not depend on this assumption.

Discussion

We have shown that ecological differences between haploid and diploid juvenile phases can lead to the evolution and maintenance of haploid-diploid life cycles, even in species with isomorphic adults. In particular, we have identified the range of conditions under which a biphasic life cycle will be favored over evolutionary time. We have also identified the evolutionarily stable life-cycle strategy (ESS). As long as resource competition between haploids and diploids is sufficiently weak (i.e., $f_d K_d > \alpha_{dh} f_h K_h$, $f_h K_h > \alpha_{hd} f_d K_d$, and $1 > \alpha_{hd}\alpha_{dh}$), an ESS haploid-diploid population exists that is able to invade either a haplontic population or a diplontic population and is stable to invasion against all other life-cycle alleles.

According to the data of Destombe et al. (1989), diploids have higher adult survival, higher fecundity, and higher juvenile survival. All else being equal, this should cause alleles that increase the dominance of the diploid phase to be favored because diploids that directly give rise to more diploids will do better than diploids that give rise to haploids, which have lower survival and fecundity. One reason why diploidy might not evolve in this case is that individuals that skip the haploid phase may pay a high reproductive cost ($x_d < 1$). However, the abundance of organisms with diplontic life cycles suggests that this cost

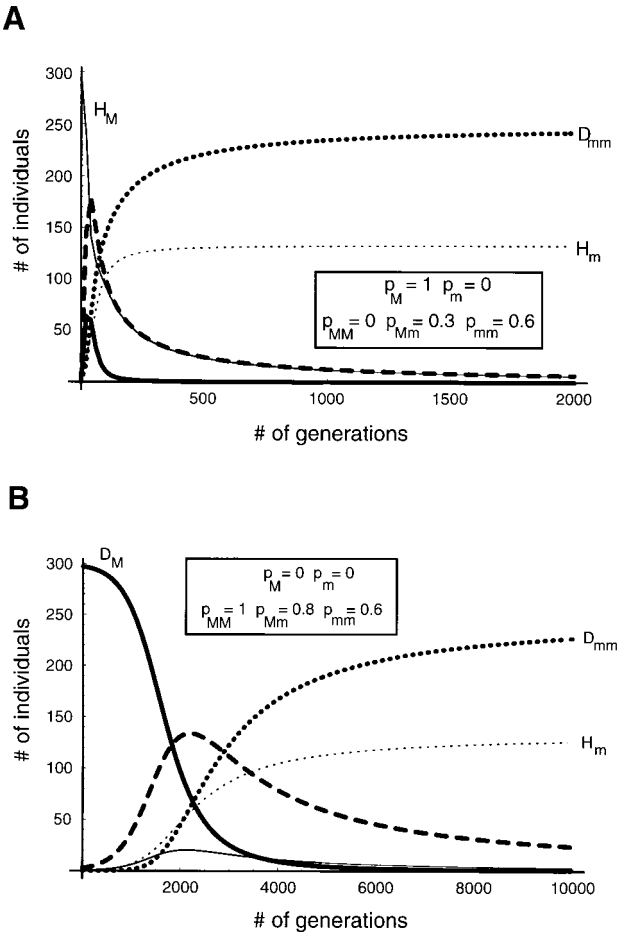

Figure 4: Evolution of haploid-diploidy. With $\alpha_{dh} = 1/2$ and $\alpha_{hd} = 1/2$, both a haplontic population ($H_M = 300$, $D_{MM} = 0$) and a diplontic population ($D_{MM} = 300$, $H_M = 0$) can be invaded by any life-cycle allele that causes a regular alternation of generations, as shown in parts *A* and *B*, respectively. In both cases, the invading allele brought the population to the ESS ($p_m = 0$, $p_{mm} = 0.6$) and, eventually, fully displaced the *M* allele. See figure 3 for more details.

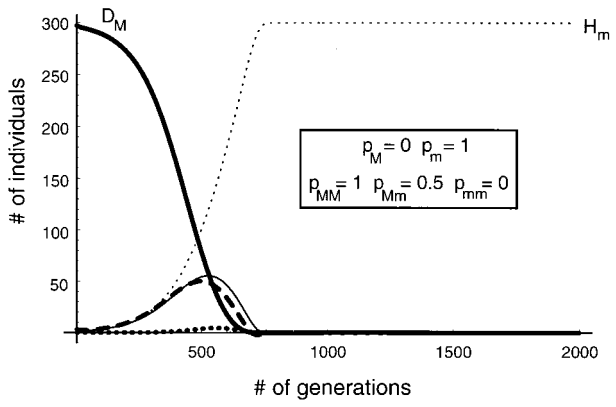

Figure 5: Evolution of haplonty. With $\alpha_{dh} = 1.5$ and $\alpha_{hd} = 0.5$, a diplontic population ($D_{MM} = 300$, $H_M = 0$) can be invaded by any life-cycle allele that increases the number of haploids within the population. In particular, the *M* allele can be fully replaced by an allele *m* that causes the population to become haplontic, as illustrated ($p_m = 1$, $p_{mm} = 0$). See figure 3 for more details.

can be reduced and need not bar the evolution of diploidy. The other possibility is that differences in competitive ability overcome the advantage of higher diploid survival and fecundity. This could happen in two ways. First, if haploids and diploids occupy different niches ($\alpha_{hd}, \alpha_{dh} < 1$), so that diploid spores are poor competitors for haploid resources, then haploids may persist even if diploid spores are more abundant. Second, haploids can compensate by being more resistant to competition ($K_{jh} > K_{jd}$) or by being better overall competitors ($\alpha_{hd} < \alpha_{dh}$). The life cycle that is favored depends on the balance of all of these factors. For example, in simulation a, haploids are more resistant to competition ($K_{jh} > K_{jd}$) and occupy slightly different niches than diploids ($\alpha_{hd}, \alpha_{dh} = 0.9$), but this is insufficient to overcome the reproductive and survival advantages of diploids, and diplonty is favored (fig. 3). If, however, haploids have a strong competitive advantage ($\alpha_{hd} \ll \alpha_{dh}$), diploids can be excluded (simulation d; fig. 5). Both haploid and diploid phases of *Gracilaria* can be maintained when niche differentiation and haploid resistance to competition are sufficient to compensate for higher diploid survival and reproduction but are not so great that the diploid phase is lost altogether (simulation b; fig. 4).

Some evidence suggests that these hypothesized differences in competitive ability may actually exist. Destombe et al. (1993) studied the growth and survival of haploid and diploid juvenile holdfasts under different environmental conditions. They found that haploids grew better than diploids in low-nutrient, natural seawater. When the water was enriched with nutrients, diploids gained the advantage, suggesting that haploids better exploit low-resource environments, while diploids are more vigorous when resources are abundant. If juveniles compete for nutrient resources, then haploids may in fact resist competition better than diploids and may grow under a broader range of conditions.

There are also some theoretical reasons to expect haploids to have a competitive advantage, at least when they are small (Lewis 1985). Haploid cells tend to be smaller than diploid ones, with a higher surface area to volume ratio. Since the nutrient requirements of cells are proportional to their volume, while nutrient uptake rate is proportional to surface area, haploid cells with high surface area to volume ratios will better exploit low-nutrient environments. The effect of resource competition is to lower the abundance of available resources. Thus, haploid cells should be less affected by competition than diploid ones. The experiments of Adams and Hansche (1974) on unicellular yeast are consistent with this expectation. They found that haploids outcompeted diploids when growth was limited by a single nutrient, which caused fitness to depend on the ability to transport this nutrient efficiently across the cell membrane.

As a caveat to his argument that haploids should have a competitive advantage, Lewis (1985) pointed out that the association between cell surface area and organism surface area becomes less close as multicellular organisms become larger. Thus, his hypothesis is most relevant for organisms composed of one or a few cells. Even if haploid cells are smaller, multicellular haploid individuals need not be. However, even completely isomorphic adults must have been young and small once. In the early phases of growth from a unicellular spore, the linkage between cell surface area and organism surface area remains, and Lewis' nutrient limitation hypothesis may still be relevant.

Destombe et al.'s (1993) study shows that ecological differences between the isomorphic phases of *Gracilaria* do exist, and it seems plausible that these differences fulfill the theoretical conditions for haploid-diploid advantage. How general are these results? This is difficult to assess directly, as little relevant information has been gathered for other species. Attempts to look for ecological differences in other isomorphic algae have met with varying success. In some cases, differences in biochemistry, distribution, and seasonality have been observed, while in others no differences could be detected (see Dyck and DeWreede 1995 for summary). It is encouraging to note that in all cases in which life-history parameters were measured in the field, differences have been observed. However, there are so few of these studies that it is difficult to generalize with much confidence. More studies are needed to assess how prevalent ecological differences between phases are and how generally useful this model is at ac-

counting for observed life-cycle variation. In particular, more demographic data, especially on the effects of competition, would provide parameter estimates to help test this model directly.

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APPENDIX A

Population Viability

A haploid-diploid population with a fixed life-cycle allele, M, will be viable if it is able to grow from an initially small size. For this to occur, the following matrix obtained from equation (1) must have a leading eigenvalue that is positive and >1:

$$\left.\begin{pmatrix} \frac{\partial H'}{\partial H} & \frac{\partial H'}{\partial D} \\ \frac{\partial D'}{\partial H} & \frac{\partial D'}{\partial D} \end{pmatrix}\right|_{\substack{H=0 \\ D=0}} = \begin{pmatrix} 1-d_h+f_h s_h x_h p_M & f_d s_h (1-p_{MM}) \\ f_h s_d (1-p_M) & 1-d_d+f_d s_d x_d p_{MM} \end{pmatrix}. \tag{A1}$$

An examination of the eigenvalues of (A1) indicates that the leading eigenvalue is strictly positive unless $1-d_h+f_h s_h x_h p_M$ and $1-d_d+f_d s_d x_d p_{MM}$ both equal 0, a special condition that we assume does not hold. The leading eigenvalue is then >1 whenever condition (9a) is met. A purely haplontic population ($p_M = 1, D = 0$) is viable whenever the upper left-hand element of (A1) is >1, giving (9b). Similarly, a purely diplontic population ($p_{MM} = 1, H = 0$) is viable whenever the lower right-hand element of (A1) is >1, giving (9c).

Invasion of a Haplontic Population by Haploid-Diploidy

A haplontic population fixed on allele M with $p_M = 1$ will reach a stable equilibrium with K_h adults when conditions (9b) and (10a) are met. If an alternative life-cycle allele, m, appears within the population with $p_m < 1$, invasion will occur if the leading eigenvalue of the stability matrix near this equilibrium is real and >1. The local stability matrix describing the dynamics of carriers of allele m is given by

$$\left.\begin{pmatrix} \frac{\partial H'_m}{\partial H_m} & \frac{\partial H'_m}{\partial D_{Mm}} \\ \frac{\partial D'_{Mm}}{\partial H_m} & \frac{\partial D'_{Mm}}{\partial D_{Mm}} \end{pmatrix}\right|_{\substack{H_M = K_h,\, D_{MM} = 0 \\ H_m = 0, \\ D_{Mm} = D_{mm} = 0}} = \begin{pmatrix} 1-\frac{1}{2}d_h(1-p_m) & \frac{d_h f_d (1-p_{Mm}+\frac{1}{2}p_{Mm}x_d x_h)}{2 f_h x_h} \\ f_h(1-p_m)\text{survival} & 1-d_d+\frac{1}{2}f_d x_d p_{Mm}\text{survival} \end{pmatrix}, \tag{A2}$$

where "survival" is the rate of diploid spore survival ([number of diploid recruits]/[number of diploid spores]) when the haplontic population is near its carrying capacity, which equals

$$\text{survival} = s_d - (f_d s_d x_d - d_d)\frac{\alpha_{dh} f_h x_h K_h}{f_d^2 x_d^2 K_d}. \tag{A3}$$

We assume that this survival rate is nonnegative. That is, density dependence will, at worst, reduce diploid spore survival to 0 but cannot cause negative recruitment. If diploid spore survival equals 0, then the leading eigenvalue of (A2) is ≤1, and the *m* allele will not increase in frequency when rare. Therefore, assuming that diploid spore survival is positive, that all individuals have some fertility (f_h, f_d, x_h, $x_d > 0$), and that some death occurs (d_h, $d_d > 0$), the elements of (A2) are strictly positive. The Perron-Frobenius theorem (Gantmacher 1989) ensures that the leading eigenvalue of such a positive matrix will be real and nonnegative. Following a straightforward proof, it can be shown that the leading eigenvalue of (A2)will be >1 if diploid spore survival is greater than

$$\frac{d_d x_h}{f_d(1 - p_{Mm} + p_{Mm} x_h x_d)}. \tag{A4}$$

Therefore, a new life-cycle allele will invade a haplontic population whenever

$$f_d x_d K_d\Big(\{f_d x_d s_d - d_d[(x_h x_d)/(1 - p_{Mm} + p_{Mm} x_h x_d)]\}/\{f_d x_d s_d - d_d\}\Big) > \alpha_{dh} f_h x_h K_h, \tag{A5}$$

which indicates that competition from haplontic juveniles must fall below a threshold value for alternative life cycles to invade. Note that we have not specified the type of life cycle encoded by the new *m* allele other than to say that it does not also cause a haplontic life cycle ($p_m < 1$), so that haploids must undergo some alternation of generations. If $x_h = x_d = 1$, condition (A5) reduces to (11a).

Invasion of a Diplontic Population by Haploid-Diploidy

A diplontic population fixed on allele *M* with $p_{MM} = 1$ will reach a stable equilibrium with K_d adults when conditions (9c) and (10b) are met. Performing an analysis similar to that used to derive (A5), it can be shown that an alternative life-cycle allele, *m*, with $p_{Mm} < 1$, will invade whenever

$$f_h x_h K_h\left(\left\{f_h x_h s_h - d_h\left[\left(x_h x_d\right)/\left(1 - \frac{p_m}{2} + \frac{p_m}{2} x_h x_d\right)\right]\right\}/\{f_h x_h s_h - d_h\}\right) > \alpha_{hd} f_d x_d K_d, \tag{A6}$$

as long as haploid spore survival is positive, all individuals have some fertility (f_h, f_d, x_h, $x_d > 0$), and some deaths occur (d_h, $d_d > 0$). If $x_h = x_d = 1$, condition (A6) reduces to (11b).

ESS Analysis

The evolutionarily stable life-cycle strategy is calculated as follows. Suppose the population is fixed for an allele coding for the ESS values ($\hat{p}_M$, $\hat{p}_{MM}$), which results in an equilibrium number of haploids and diploids, $\hat{H}_M$ and $\hat{D}_{MM}$, respectively. Because ($\hat{p}_M$, $\hat{p}_{MM}$) is an ESS, no mutant allele, *m*, can invade the population. This means that the equilibrium with $H_M = \hat{H}_M$, $D_{MM} = \hat{D}_{MM}$, and $D_{Mm} = D_{mm} = H_m = 0$ must be stable to invasion by any alternative allele, *m*, with (p_m, p_{Mm}) ≠ ($\hat{p}_M$, $\hat{p}_{MM}$). (Note that we do not consider *mm* homozygotes, which are negligible in frequency in the case of random mating.) The stability of the ESS equilibrium to invasion by a new life-cycle allele is determined by the leading eigenvalue, λ, of the local stability matrix

$$\left.\begin{pmatrix} \frac{\partial H_m'}{\partial H_m} & \frac{\partial H_m'}{\partial D_{Mm}} \\ \frac{\partial D_{Mm}'}{\partial H_m} & \frac{\partial D_{Mm}'}{\partial D_{Mm}} \end{pmatrix}\right|_{\substack{H_M = \hat{H}_M,\, D_{MM} = \hat{D}_{MM} \\ H_m = D_{Mm} = D_{mm} = 0}}, \tag{A7}$$

with stability requiring that λ be at most 1.

Because λ = 1 when $p_m = \hat{p}_M$ and $p_{Mm} = \hat{p}_{MM}$, this point must represent a local maximum of λ as a function of p_m and p_{Mm}, or else certain alleles that alter the life cycle could invade. Thus, to qualify as an ESS, ($\hat{p}_M$, $\hat{p}_{MM}$) must satisfy

$$\left.\frac{\partial \lambda}{\partial p_m}\right|_{\hat{p}_M, \hat{p}_{MM}, \hat{H}_M, \hat{D}_M} = 0, \tag{A8a}$$

$$\left.\frac{\partial \lambda}{\partial p_{Mm}}\right|_{\hat{p}_M, \hat{p}_{MM}, \hat{H}_M, \hat{D}_M} = 0. \tag{A8b}$$

These two conditions are calculated by implicitly differentiating the characteristic polynomial of (A7)and simplifying using the requirement that $\lambda|_{\hat{p}_M, \hat{p}_{MM}, \hat{H}_M, \hat{D}_{MM}} = 1$. An additional condition is provided by the fact that $\hat{H}_M$ and $\hat{D}_{MM}$ must be an equilibrium solution of (6a) and (6b). When $x_h = x_d = 1$, we were able to solve these three conditions for the four unknowns, $\hat{p}_M$, $\hat{p}_{MM}$, $\hat{H}_M$, and $\hat{D}_{MM}$, giving us (13)–(14b). Note that, rather than a single ESS, there are an infinite number of different combinations of $\hat{p}_{MM}$ and $\hat{p}_M$ on the line given by (13) that satisfy the ESS conditions and that all lead to the same number of adult haploids and diploids, (14a) and (14b). Also note that the ESS is biologically valid (with a positive number of haploids and diploids) only if conditions (11a), (11b), and $(1 - \alpha_{hd}\alpha_{dh}) > 0$ are all true or are all false.

The above analysis indicates that a population at the ESS given by (13)–(14b) will not be invaded by a new *m* allele that causes a slight change to the life cycle. We were further able to show that the leading eigenvalue of (A7)evaluated at (13)–(14b) is always equal to one, demonstrating that the ESS is neutrally stable to invasion by any new life-cycle allele.

While no new life-cycle allele will be able to increase geometrically in frequency within an ESS population, it is not necessarily true that a population away from ESS will approach the ESS. In fact, we already know that a haplontic population cannot be invaded by an allele causing a haploid-diploid life cycle (including the ESS configuration) unless condition (11a) is met. Similarly, a diplontic population cannot be invaded by an allele bringing the population toward the ESS unless condition (11b) is met. Therefore, for the ESS to be biologically valid and evolutionarily attainable (at least starting from a haplontic or diplontic population) requires that conditions (11a), (11b), and $(1 - \alpha_{hd}\alpha_{dh}) > 0$ all are true.

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