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WHEN CAN A CLONAL ORGANISM ESCAPE SENESCENCE?

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Abstract.—Some clonal organisms may live for thousands of years and show no signs of senescence, while others consistently die after finite life spans. Using two models, we examined how stage-specific life-history rates of a clone's modules determine whether a genetic individual escapes senescence by replacing old modules with new ones. When the rates of clonal or sexual reproduction and survival of individual modules decline with age, clones are more likely to experience senescence. In addition, the models predict that there is a greater tendency to find senescence in terms of a decline in the rate of sexual reproduction with clone age than in terms of an increase in the probability of clone mortality, unless rates of sexual reproduction increase dramatically with module stage. Using a matrix model modified to represent the clonal lifestyle, we show how a trade-off between sexual and clonal reproduction could result in selection for or against clonal senescence. We also show that, in contrast to unitary organisms, the strength of selection on life-history traits can increase with the age of a clone even in a growing population, countering the evolution of senescence.

Senescence is an unavoidable part of the life histories of unitary organisms (Partridge and Barton 1993). It seems, however, that clonal organisms that produce genetically identical modules may escape the ravages of age at the level of the genetic individual, or genet. Although modules (ramets) may experience senescence (i.e., show decreasing survival or reproduction with age), production of new modules could preclude senescence of the genet. For example, there are quaking aspen clones estimated to be over 10,000 yr old that span 81 ha (Cook 1985), creosote bush (*Larrea tridentata*) clones to be 11,700 yr old (Vasek 1980), bracken ferns to be 1,400 yr old (Oinonen 1967), anemones to be hundreds of years old (Hughes 1989, p. 169), and weevil clones in Europe to have persisted since the retreat of the Pleistocene glaciers (Hughes 1989, pp. 76, 204). At the same time, genets of other clonal organisms appear to decline in vigor with clone age. All natural strains of the fungus *Podospora anserina* die, although strains vary in their life spans (Griffiths 1992). In contrast, only in some strains of fungi in the genus *Neurospora* do survival rates fall with clone age. Asexual metazoans (the oligochaete *Paranais litoralis* and the rhabdocoel *Stenostomum incaudatum*) experience senescence (Martinez and Levinton 1992), as

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do rotifer clones of more than one species (Lansing 1942*a*, 1942*b*, 1947). Even in long-lived quaking aspen clones, there appears to be environmental and genetic variation between clones in the rate of senescence (Shields and Bockheim 1981). In this article, we investigate the aspects of a clonal life history that may enable genets to escape senescence, and why some clonal organisms experience senescence nevertheless.

Senescence is usually viewed as a decreasing probability of survival or rate of fertility with age (Finch 1990). Evolutionary arguments posited for the senescence of unitary organisms are that organisms accumulate mutations later in life or that there are trade-offs between early and late reproduction and survival because of pleiotropy (Partridge and Barton 1993). Both of these arguments depend on the decline of the contribution of reproduction and survival to fitness with age (Medawar 1946, 1952; Hamilton 1966). In contrast to unitary organisms, clonal organisms continually produce, without undergoing meiosis, new physiological individuals (ramets). Ramets may be produced by processes such as budding or fragmentation (animals) or by elongation of meristems that lead to new shoots (plants). Even if ramets experience senescence, the probability of mortality of the genet may decline if the rate of ramet production exceeds that of ramet death (Cook 1979, 1983).

We examine clonal senescence of a genet from three angles, two of which are demographic, and the third, evolutionary. In the demographic sense, we interpret senescence to mean changes in an organism that adversely affect its probability of survival or its fertility with increasing age. In the evolutionary sense, we view there to be selection that can lead to senescence if the strength of natural selection declines with the age of a clone. Thus, it may be possible for a clone to experience evolutionary pressure that precipitates senescence but not to experience senescence from a demographic standpoint; this occurs if the chance of genet mortality declines with clone age, but nevertheless selection is stronger on traits displayed earlier rather than later in the life of a genet.

Recent investigators argued that senescence is possible but not obligatory in clonal organisms (Watkinson and White 1985; Fagerstrom 1992; Orive 1995; Pedersen 1995), but these investigators have not provided a general understanding of when and why senescence occurs and when and why it does not. We use two models to clarify the aspects of reproduction and survival that determine when a clonal organism may escape senescence. The first is a stage-based transition matrix (Lefkovitch 1965) modified for aspects unique to clonal organisms. The second describes the genet as a combination of old and young ramets, where the transition probabilities among different states are derived from age-specific rates of mortality and reproduction of ramets. In both models, we compute the change in the probability of genet mortality and the genet-wide rate of (sexual) reproduction with time and evaluate which combinations of parameters lead to demographic senescence—that is, an increasing chance of genet mortality or a decreasing rate of sexual reproduction with time. In the matrix model we also evaluate how rates of clonal and sexual reproduction affect the sensitivity of fitness to changes in ramet life-history rates (the strength of selection) as a clone ages, potentially resulting in selection to escape senescence.

MODEL I: MODIFIED STAGE-STRUCTURED MATRIX (MSSM)

We use a stage-structured matrix model (cf. Caswell 1985; Orive 1995) to describe an organism that clones new modules. The stage-based matrix model used by Caswell (1985) and Orive (1995) follows the dynamics

$$\mathbf{N}(t + 1) = \mathbf{A}\mathbf{N}(t), \tag{1}$$

where

$$\mathbf{N}(t) = [n_1(t), n_2(t), n_3(t), \ldots, n_x(t)]^T, \tag{2a}$$

with $n_i(t)$ the number of ramets in stage i at time t. The specific example that we will consider has

$$\mathbf{N}(t) = [n_1(t), n_2(t), n_3(t), n_4(t)]^T, \tag{2b}$$

where stage $i = 1$ represents seeds; $i = 2$ small ramets, e.g., ramets with one to three leaves; $i = 3$ medium-sized ramets, e.g., ramets with four to six leaves; and $i = 4$ large ramets, e.g., ramets with seven or more leaves. The matrix $\mathbf{A}$ describes transition probabilities between states

$$\mathbf{A} = \begin{bmatrix} 0 & f_1 & f_2 & f_3 \\ g_0 & l_1 & c_{12} & c_{13} \\ 0 & g_1 & l_2 & c_{23} \\ 0 & 0 & g_2 & l_3 \end{bmatrix}. \tag{3}$$

In equation (3), f_i represents the fecundity (sexual reproduction) of ramets in the ith stage, g_i represents the probability of growth from stage i to $i + 1$, and l_i represents the survival of ramets in stage i. The entries c_{ji} above the diagonal represent the production of new ramets in stage j by ramets in stage i. Caswell (1985) used such a matrix for clonal organisms with the interpretation that it represents a population composed of both clonally produced ramets and sexually produced genets. The dominant eigenvalue λ gives the asymptotic population growth rate for a hypothetical population that is homogeneous with respect to life-history parameters. It thus gives a measure of the fitness of a genotype (Caswell 1985, 1989*a*, 1989*b*; Pedersen 1995) with those rates of growth, survival, and clonal and sexual reproduction, so that selection favors an increase in λ. Although using λ as a measure of fitness requires some restrictive assumptions (Charlesworth 1980), it is widely used in investigations of both clonal and unitary organisms (Caswell 1985; Stearns 1992; Pedersen 1995).

Following these analyses of life history, we assume that the eigenvalue λ of matrix $\mathbf{A}$ is a good measure of the fitness of a genotype, a fact that we will use later when examining how selection on reproductive trade-offs indirectly affect senescence. However, λ has two shortcomings for evaluating the probability of mortality of a genet, the critical information for investigating whether a clone experiences senescence. First, the standard Lefkovitch matrix mixes clonal and sexual reproduction so that one cannot discriminate between the clonal produc-

tion of ramets of a genet from sexual production of new genets. This makes computing the life span of a particular genet tricky if one cannot assume that the ramets composing a particular genet are at equilibrium (although with the assumption that ramets of a genet have reached equilibrium, genet life span can be obtained by computing absorption times; Hoel et al. 1972; Orive 1995). Second, a ramet may actually shrink from state i to j by herbivory, disturbance, or resource scarcity. This contributes to c_{ji} in precisely the same way as clonal reproduction accompanied by death of the ramet in stage i, that is, by increasing c_{ji} and decreasing l_i. As a consequence, one cannot distinguish shrinkage of older ramets that may be experiencing senescence from reproduction of new, young ramets. This difficulty in separating retrogression from clonal growth was also noted by Silvertown et al. (1993).

To tailor a Lefkovitch matrix to evaluate demographic senescence in a clonal organism, we distinguish ramet from genet dynamics with a modified stage-structured matrix (MSSM). Sexual reproduction is not included in the matrix itself but is calculated separately. To specify the actual probability of mortality of ramets versus shrinkage without mortality, we split the entries above the diagonal into a portion from shrinkage s_{ji} and a portion from clonal reproduction c_{ji}, so that the entries are now $s_{ji} + c_{ji}$, resulting in the matrix

$$\mathbf{B} = \begin{bmatrix} l_1 & s_{12} + c_{12} & s_{13} + c_{13} \\ g_1 & l_2 & s_{23} + c_{23} \\ 0 & g_2 & l_3 \end{bmatrix}. \tag{4}$$

The values of $l_i + g_i + s_{ji} \leq 1$ and $c_{ji} \leq 1$, since we assume that there is only time for a ramet to clone a single daughter ramet in each interval.

Because sexual reproduction is treated separately, the vector $\mathbf{N}(t)$ loses the dimension of seeds and thus has only three entries representing the numbers of small ($i = 1$), medium ($i = 2$), and large ($i = 3$) ramets. A genet begins at $t = 0$ with one ramet in stage 1. Thus, $\mathbf{N}(0) = [1\ 0\ 0]^T$. The life history may be divided into more than three stages but the analysis follows the same principles.

A ramet in stage i may follow one of four courses: survive and remain in the same stage with probability l_i, survive and grow with probability g_i, survive and shrink with probability s_{ji}, or die with probability d_i. Since these are mutually exclusive events,

$$l_1 + g_1 + d_1 = 1, \tag{5a}$$

$$l_2 + g_2 + s_{12} + d_2 = 1, \tag{5b}$$

$$l_3 + s_{13} + s_{23} + d_3 = 1. \tag{5c}$$

The c_{ji}'s are not included in these equations, with the interpretation that in each time period, ramets first produce daughter ramets and then they do one of the following: survive in the same class, survive and grow, survive and shrink, or die. An analysis for an alternative schedule of events is given in appendix A.

We assume that the mortality of ramets is independent of the fate of other ramets (in the Discussion section, we suggest an alternative interpretation of the following method when this assumption does not hold). If mortality of the genet occurs when all its ramets die, then the probability of mortality of the genet at time t is

$$M(t) = d_1^{(n(3,t)c_{13}+n(2,t)c_{12})} d_2^{(n(3,t)c_{23})} \prod_{i=1}^{3} d_i^{n(i,t)}. \tag{6}$$

The first two terms are the probabilities that clonal offspring produced before ramet death also die. It follows that the probability of survival of a genet until time t is

$$L(t) = \prod_{x=1}^{t} (1 - M(x)). \tag{7}$$

The net rate of increase of ramets within a genet is

$$R(t) = \sum_{i=1}^{3} n(i, t) \Big/ \sum_{i=1}^{3} n(i, t-1). \tag{8}$$

Sexual reproduction depends on the fecundity f_i of producing same-sex offspring, $n(i, t)$, and the probability g_0 that those sexual propagules establish and grow into stage 1 ramets. Thus, the number of sexual offspring at time t is

$$S(t) = g_0 \sum_{i=1}^{3} f_i n(i, t-1). \tag{9}$$

We evaluate how changing the fecundities (f_i's) and transition probabilities in **B** affect the probability of genet mortality ($M(t)$), the rate of increase of ramets of a genet ($R(t)$), and the number of sexual offspring produced ($S(t)$). If $M(t)$ increases as t increases or $S(t)$ decreases as t increases, the genet experiences senescence.

To distinguish juvenile mortality from adult mortality, we calculate the average life span $\bar{a}$ of a genet from

$$\bar{a} = \sum_{t=1}^{t_{\max}} tL(t-1)M(t). \tag{10}$$

Thus an individual is "juvenile" for $a < \bar{a}$ and is adult afterward. Changes in mortality that happen much before $\bar{a}$ are associated with juvenile mortality. Calculating the rate of change of genet-wide sexual reproduction and genet mortality around $\bar{a}$ provides a biologically meaningful prognosis for senescence. Otherwise, a decrease in the rate of mortality that occurs after a time when a clone is most likely dead, that is, after $\bar{a}$ might be misinterpreted as an escape from senescence, when in reality virtually no clones live to that point. To calculate a finite $\bar{a}$ for clones that do not experience senescence to compare with clones that

do, we let $M(t_{\max}) = 1$. This makes no difference for the prognosis of senescence, although it underestimates $\bar{a}$. If

$$M(\text{Int}(\bar{a}) + 1) - M(\text{Int}(\bar{a})) > 0, \tag{11}$$

where $\text{Int}(\bar{a})$ means to take the integer value of $\bar{a}$ then a clone experiences senescence by the genet mortality criterion. Similarly, if

$$S(\text{Int}(\bar{a}) + 1) - S(\text{Int}(\bar{a})) < 0, \tag{12}$$

then a clone declines in vigor with age by the fertility criterion. This interpretation of senescence differs from that of Caswell (1985), in which he states that since neither the steady state reproductive values nor the stable stage distribution of ramets need be decreasing functions of size, senescence need not be universal for clonal organisms. However, because the ramets within a genet may never reach a steady state before the genet dies, we suggest that the rate of change in the probability of genet mortality or rate of genet fecundity around the average genet life span provide better indicators of genet senescence than do equilibrium reproductive values and age distribution.

For many clonal plants, genets face a trade-off between clonal and sexual reproduction (Sutherland and Vickery 1988; Geber 1990; Hartnett 1990). The balance of reproduction depends on the relative payoff of a given outlay of clonal versus sexual allocation. Investments in clonal and sexual reproduction, in turn, affect the rate of genet senescence. Since selection favors genotypes that balance clonal and sexual reproduction to yield the highest fitness, selection on reproductive trade-offs also may affect whether genets experience senescence, not because of direct selection for or against senescence but because of selection on reproductive trade-offs that in turn affect senescence. We anticipate that lower rates of clonal reproduction will result in the senescence of a genet. Therefore, a genet might experience selection for senescence indirectly via selection for sexual over clonal reproduction.

To investigate selection on sexual and clonal trade-offs, we recall that λ of the matrix **A**, the per-time-unit rate of change of a genet and its sexual progeny, represents fitness. If the entries c_{ji} in **A** are replaced by $c_{ji} + s_{ji}$, then **A** matches the modifications that were necessary to construct matrix **B** and includes both sexual as well as clonal reproduction, in contrast to **B**. By definition, $\text{Det}(\mathbf{A} - \lambda\mathbf{I}) = 0$, so λ satisfies

$$\begin{aligned} 0 = {} & \lambda[(l_1 - \lambda)(l_2 - \lambda)(l_3 - \lambda) + (c_{13} + s_{13})g_1g_2 \\ & - g_2(c_{23} + s_{23})(l_1 - \lambda) - (l_3 - \lambda)g_1(c_{12} + s_{12})] \\ & + g_0[f_1(l_2 - \lambda)(l_3 - \lambda) + f_3g_1g_2 - g_2(c_{23} + s_{23})f_1 - (l_3 - \lambda)g_1f_2]. \end{aligned} \tag{13}$$

If λ increases with clonal output, c_{ji}, then selection favors clonal reproduction and acts against senescence. Otherwise, if λ decreases with c_{ji}, then selection favors the senescence of a genet. A trade-off between clonal and sexual reproduction implies that resources are divided (perhaps unevenly) between the two. Thus, a given investment in sexual reproduction, represented by f, requires re-

sources that could otherwise be put into clonal offspring, indicated by c, so that $f = \alpha(1 - c)$, where α measures the relative benefit of sexual compared to clonal reproduction. For large α, sexual investment confers a greater payoff than an equal clonal outlay. The value of α for which λ neither increases nor decreases with c is the critical payoff of sexual relative to clonal allocation, above which selection favors sexual reproduction and therefore clonal senescence and below which selection favors clonal reproduction and hence an escape from genet senescence. We present results obtained by assuming that $f_1 = f$, $f_2 = 3f$, and $f_3 = 2f$, although the results are qualitatively similar with alternate assumptions (e.g., increasing fecundity with stage: $f_1 = f$, $f_2 = 3f$, and $f_3 = 5f$; or declining fecundity with stage: $f_1 = 5f$, $f_2 = 3f$, and $f_3 = f$). We also assume that $c_{12} = c_{13} = c$ and $c_{23} = 0$ (clonal daughters start at stage 1) and that ramets do not shrink so $s_{ji} = 0$, although again these assumptions may be altered without changing the qualitative results that we will present. Then we substitute $f = \alpha(1 - c)$ in equation (13), differentiate with respect to c, set $d\lambda/dc = 0$, and solve for α. This produces a threshold value α_{crit}:

$$\alpha_{crit} = \frac{\lambda(g_1g_2 - g_1l_3 + g_1\lambda)}{g_0(\lambda^2 - l_3\lambda - l_2\lambda + 3g_1\lambda - 3g_1l_3 + 2g_1g_2 + l_2l_3)}. \quad (14)$$

That is, α_{crit} is the threshold trade-off between sexual and clonal reproduction at which the switch between selection for or against senescence occurs; it can be determined by the numerical solution of equations (13) and (14).

The theory for unitary organisms shows that selection is stronger on traits that operate early rather than late in life (Medawar 1946, 1952; Hamilton 1966), allowing a decline in fitness with age because of the accumulation of mutations or selection for pleiotropic alleles. The equivalent analysis for a clonal organism requires that one find the magnitude of the effect of a change in the probability of genet survival on fitness when the change in genet survival occurs at different genet ages. If the effect on fitness increases with genet age, then the strength of selection also grows, resulting in selection against the senescence of a clone.

The fitness of a clone from age z onward is the number of ramets produced by the genet that will enter the population in the following time period through either clonal or sexual reproduction, provided that the genet survives, and discounted by the population rate of increase, summed over all future time intervals. Thus,

$$F(z) = \sum_{t=z}^{\infty} \lambda^{-t}(1 - M(t))\left[S(t + 1) + \sum_{i=1}^{3} n(i, t + 1)\right], \quad (15a)$$

which is equivalent to

$$F(z) = \lambda^{-1}(1 - M(z))F(z + 1) \quad (15b)$$

(Mangel and Clark 1988). If one of the life-history rates in the matrix B is perturbed when the genet is age z, resulting in a change to $M'(z)$ and $F'(z)$, then the

TABLE 1

SUMMARY OF PARAMETERS TO DESCRIBE RAMET BIRTH AND DEATH RATES USED TO CALCULATE GENET TRANSITION PROBABILITIES IN THE GS MODEL

Parameter	Explanation
p	Probability of ramet mortality due to external factors (age independent)
p_0	Probability of mortality of a young ramet due to internal factors (age dependent), given that the ramet does not die from external factors
p_1	Probability of mortality of an old ramet due to internal factors, given that the ramet does not die from external factors
h	Probability that a young ramet becomes an old ramet
b_0	Probability that a young ramet produces a new young ramet, given that the original ramet in the young stage does not advance to the old stage
b_1	Probability that an old ramet produces a new young ramet
s_0	Fecundity (sexual) of a young ramet
s_1	Fecundity (sexual) of an old ramet

change in expected fitness per change in genet survival during that interval ($1 - M(z)$) is

$$\Phi_z = \frac{F'(1) - F(1)}{M(z) - M'(z)}. \tag{16a}$$

Since $F(1) - F(z - 1) = F'(1) - F'(z - 1)$ is the portion of fitness that is accrued before the change and that cancels out, we may write

$$\Phi_z = \frac{F'(z) - F(z)}{M(z) - M'(z)}. \tag{16b}$$

If the curve Φ_z versus z rises, then the strength of selection grows with the age of the clone and there is selection against the evolution of senescence.

MODEL 2: STATE OF THE GENET

In the second genet state (GS) model, we wished to assess the robustness of the conclusions reached in the MSSM model by taking a different approach. The GS model is similar to the MSSM model in that stage-specific rates of clonal and sexual reproduction and survival of ramets are used to predict the circumstances in which clones experience senescence from a demographic standpoint. The GS model differs from the MSSM model in that rates of ramet mortality are clearly specified as either dependent or independent of the stage of a ramet. It is a more simple model than the MSSM model, and its primary value is to evaluate whether the same conclusions are reached by using an alternative modeling technique.

We characterize the state of a genet according to the number and stage of its ramets. We derive the transition probabilities among different states from stage-specific ramet birth and death rates (table 1; app. B). Finally, we evaluate how changing those rates affects the change over time of the probability of mortality and the (sexual) fecundity of the genet.

In the simplest case, a genet can be composed of a total of one or two ramets that are either young or old. The possible states of a genet are specified as a vector ($\boldsymbol{O}$, $\boldsymbol{Y}$), where $\boldsymbol{O}$ is the number of old ramets and $\boldsymbol{Y}$ is the number of young ramets; we index these genetic states by g_i ($i = 1$ to 6). For example, $g_1 = (0, 0)$, and so on (app. B).

The transition probabilities $q(i, j)$ of a genet moving from state j to state i are given in appendix B. For example, the probability that a genet with one young ramet dies (goes from g_2 to g_1) is the probability of ramet death from external (age-independent) causes (p) plus the probability of death from internal (age-dependent) causes given that it did not die from external causes (p_0) times the probability that it did not die from external causes ($1 - p$). If a genet already contains two ramets, then the probability of ramet birth is 0. As the size of appendix B illustrates, the derivation of transition probabilities is complicated enough with only two ramets, hence the small maximum number of ramets per genet for analysis (vs. simulation).

We start a genet with one young ramet and compute the probabilities of the genet being in each of the potential genet states in the next time period. In each time period, a ramet may make only one transition. For example, a ramet may either advance to the older stage or give birth in one interval but not both. Ramets may or may not advance to the older stage, since with the coarse characterization of young and old, there is some variation even within the young stage and therefore uncertainty in the aging of young ramets. The probability of genet mortality at time t is

$$M(t) = \sum_{j=2}^{6} q(1, j)\Pr\{\text{genet state is } g_j \text{ at time } t\}. \tag{17}$$

The probability of genet survival until t is

$$L(t) = \prod_{x=1}^{t} (1 - M(x)). \tag{18}$$

As before, the expected life span is

$$\bar{a} = \sum_{t=1}^{t_{\max}} tL(t - 1)M(t), \tag{19}$$

with $M(t_{\max}) = 1$.

The fecundity of a genet in state g_j, which is denoted by $f(j)$, is the probability of creating a new genet through sexual reproduction (table 2). The expected fecundity of a genet at time t is

$$S(t) = \sum_{j=2}^{6} \Pr\{\text{genet state is } g_j \text{ at time } t\} f(j). \tag{20}$$

As in the MSSM model, we do not use the expected sojourn time to reach the absorbing state g_1 (Hoel et al. 1972) since the ramets of a genet may not reach

TABLE 2

STATE-DEPENDENT FECUNDITIES IN THE GS MODEL

State	$f(j)$
g_1 (0, 0)	0
g_2 (0, 1)	s_0
g_3 (0, 2)	$2s_0$
g_4 (1, 0)	s_1
g_5 (2, 0)	$2s_1$
g_6 (1, 1)	$s_0 + s_1$

a steady state before the genet dies. Thus, we do not assume the population is in demographic equilibrium. Instead, the probability distribution of genet states at time t is computed for each time interval based on the distribution in the previous time interval and the transition probabilities between states, with the initial condition that genets begin as a single young ramet ($g_2[0, 1]$). As in the previous model, an increase in the probability of genet mortality or a decrease in the expected genet fecundity indicates senescence of the genet. We use equations (17) and (20) to evaluate whether genets experience senescence.

RESULTS

MSSM Model

The stage-structured matrix model predicts that as the probability of establishing clonal offspring (c_{ji}) grows, the likelihood that a clone avoids senescence increases (figs. 1, 2). A decreasing probability of genet mortality with time (fig. 1*A, B*), increasing rate of sexual reproduction with time (fig. 1*C*), and a higher rate of production of new clonal ramets (fig. 1*D*) accompany an increase in the rate of clonal reproduction. With a high probability of clonal proliferation, a clone ages only if the rate of ramet mortality (d_i) is above the threshold at which ramet mortality balances ramet production (fig. 2). A boundary curve separates the region where a clone experiences senescence and where it does not. When ramet mortality is high both the probability of genet survival and the reproductive rate decline with age.

If, on the one hand, fertility declines, stays constant, peaks at an intermediate ramet stage, or increases modestly with ramet stage, then clones experience senescence over a broader range of ramet production and mortality rates if senescence is determined by a drop in the rate of genet-wide sexual reproduction rather than by an increase in the rate of genet mortality. On the other hand, if the rate of fertility in the third stage (f_3) is much greater than fertility rates in lower stages (f_1, f_2) then the opposite occurs so that one is more likely to detect senescence by the mortality criterion than by the reproductive criterion. This

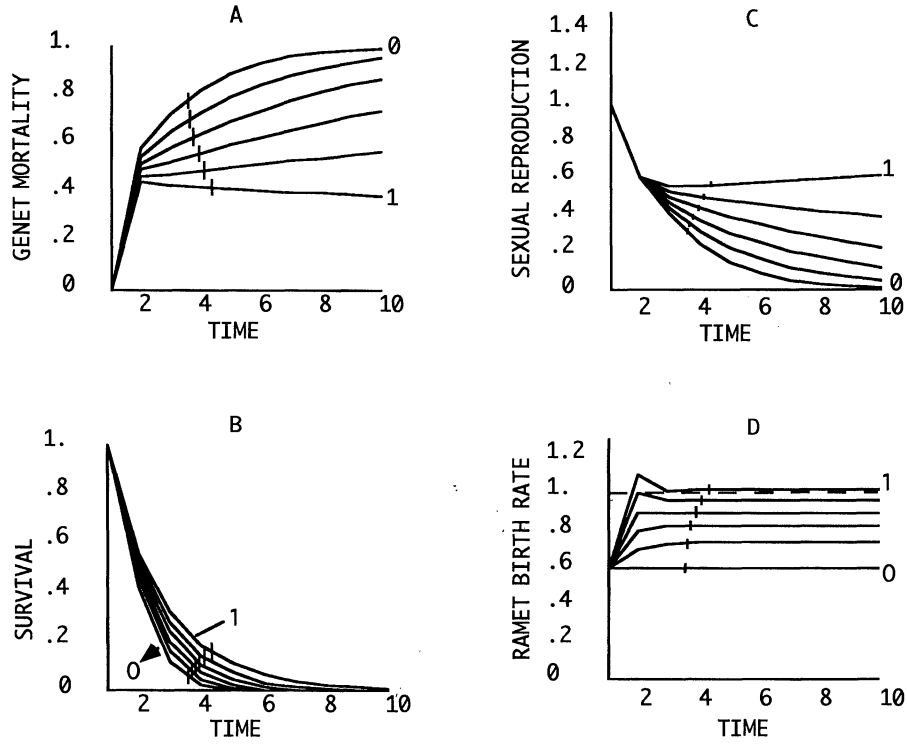

FIG. 1.—Predictions of the MSSM model: genet mortality (*A*), cumulative survival (*B*), sexual offspring (*C*), and production of new ramets (*D*) as a function of time and $c_{12} = c_{13}$ ranging from 0 to 1 in intervals of 0.2. Ticks indicate the expected life span of a clone, $\bar{a}$. (Other parameters are $l_1 = g_1 = l_2 = g_2 = 0.3$, $l_3 = 0.6$, $s_{12} = s_{13} = s_{23} = c_{23} = 0$, $f_1 = 10$, $f_2 = 30$, $f_3 = 20$.)

shifts the curve representing reproductive senescence in figure 2 upward and to the left but has no effect on the curve representing senescence in terms of genet mortality. Unless noted otherwise, in most of the following analyses we assume that $f_2 = 3f_1$ and $f_3 = 2f_1$ since it is particularly interesting if a genet can escape senescence despite the senescence of the modules that compose it. We judge this a reasonable simplification in this discussion since senescence as judged by the probability of genet mortality is unaffected by fertility assumptions.

The threshold for clonal senescence differs if one considers the mortality of small or large ramets or growth versus survival of ramets in a particular class. For low values of clonal reproduction (c_{ji}), senescence is less likely for a given value of mortality (d_3) of large ramets than for an equivalent value of mortality of small ramets (d_1; figs. 2, 3). In contrast, for more frequent clonal reproduction the exact opposite is true; escaping senescence is possible at lower survival rates of smaller ramets than of larger ramets. This result is particularly notable if changes in the rate of ramet growth from the smallest to the middle class (g_1),

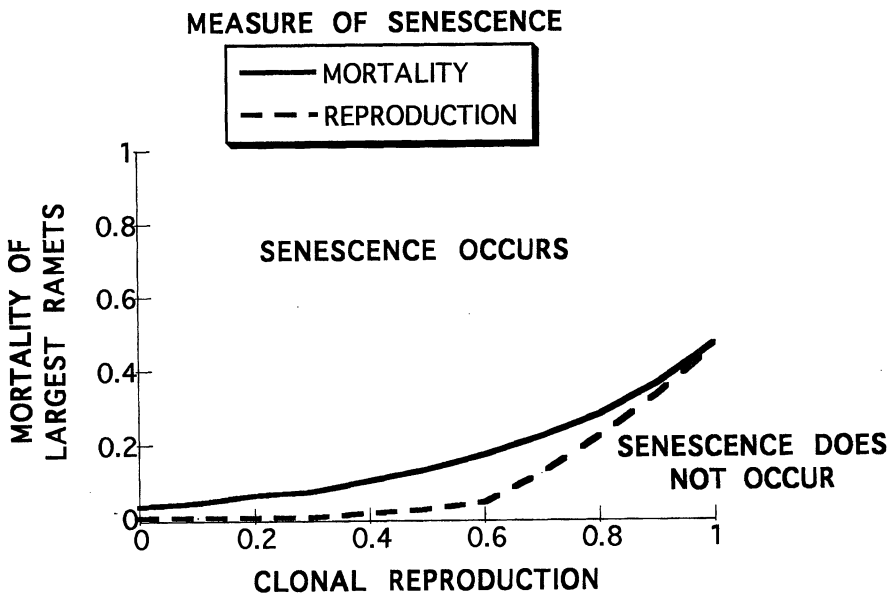

FIG. 2.—The clonal growth/mortality plane can be divided by a boundary that separates parameter values leading to senescence from parameter values that do not. Senescence is measured in terms of an increase in the probability of mortality over time or a decrease in the rate of sexual reproduction over time. As the value of d_3 varies along the Y-axis, $l_3 = 1 - d_3$. Along the X-axis, $c_{12} = c_{13}$ varies. Other parameters are as in figure 1. If $c_{23} > 0$, then the curves are shifted up and to the left, increasing the parameter space where senescence does not occur. Increasing fertility with ramet stage ($f_3 > f_1$) shifts the curve for reproductive senescence upward and to the left but has no effect on the curve for senescence measured by genet mortality.

as compared with remaining in the smallest class (l_1), cause the shift in the rate of ramet mortality. The effect of clonal growth on the threshold value of the mortality of small versus large ramets can be explained by the difference in the size distribution of ramets being shifted toward smaller ramets with higher rates of clonal growth.

There is another difference between the effects of the mortality rates of the largest (d_3) versus the smallest (d_1) ramets. The disparity between genet mortality and genet reproduction as measures of senescence is greater for a given value of mortality of larger than for smaller ramets (fig. 3). This occurs since old ramets have higher (sexual) fecundity than smaller ramets, so there is a greater direct effect on fertility from the survival of older ramets.

Producing ramets in stage 1 (c_{12} or c_{13}) acts more strongly against senescence than an equivalent rate of producing ramets in a later stage (c_{23}), both by the mortality and by the reproductive criteria of senescence. Early stage daughters live, on average, longer than late stage ramets and, therefore, more effectively reduce the probability of genet mortality. Qualitatively, however, other results

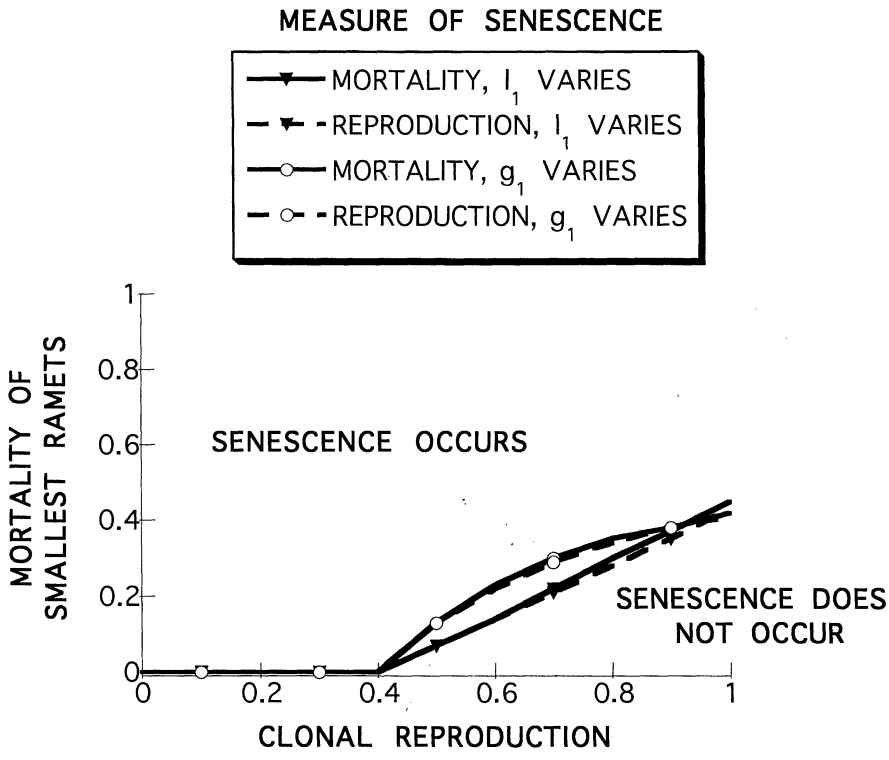

FIG. 3.—Parameter space as in figure 2 except the mortality of ramets in the first class (d_1) varies along the *Y*-axis, so $l_1 = 1 - g_1 - d_1$ when l_1 (survive and remain in the smallest class) varies, and $g_1 = 1 - l_1 - d_1$ when g_1 (survive and grow to the second class) varies. Other parameters are as in figure 1.

are similar, so for brevity we present only the results from analyses where $c_{12} = c_{13} = c$ and $c_{23} = 0$.

Increasing herbivory (s_{ji}; i.e., shrinkage, or a transition from a ramet in the second or third class to one in the first class), even if it does not cause death, can result in senescence (fig. 4). Herbivory is different than clonal growth because a change in herbivory is balanced by a change in ramet survival in the same class, ramet growth to a larger class, or ramet mortality, since the probability of these four alternative transitions must sum to 1 (eq. [5a–c]). A change in clonal reproduction, however, may have no effect on the rate of ramet mortality. For example, we held ramet mortality, d_i, of each class, i, constant and varied stage-dependent shrinkage or herbivory ($s_{12} = s_{13} = s$, $s_{23} = 0$), survival in the same class (l_2, l_3), and growth to the next class (g_2) according to

$$\begin{aligned} l_2 &= g_2 = (1 - s - d_2)/2 \\ l_3 &= 1 - d_3 - s \end{aligned} \tag{21}$$

to find the threshold level of herbivory at which genet senescence occurs. Only

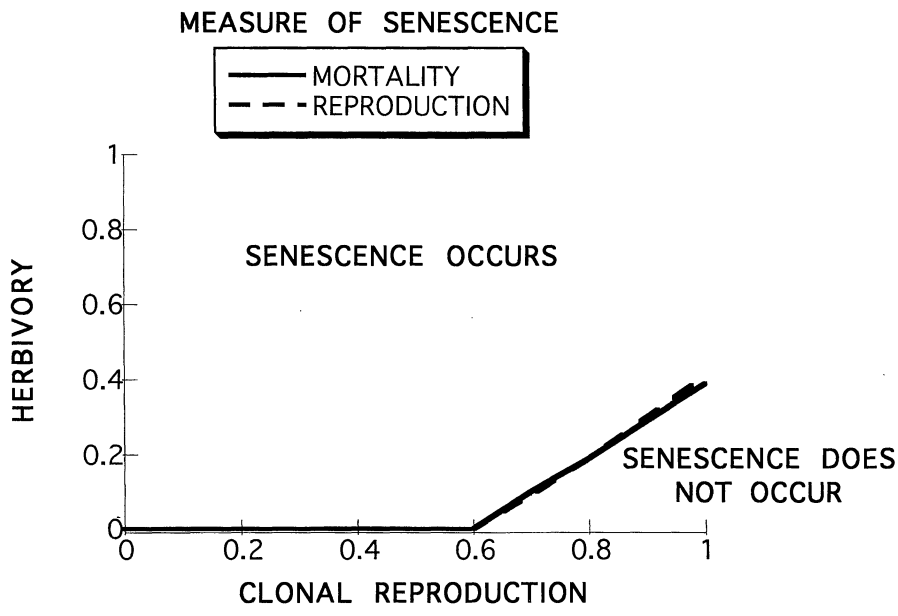

FIG. 4.—As in figures 2 and 3, except herbivory ($s_{12} = s_{13}$) varies along the Y-axis, and $d_1 = d_2 = d_3 = 0.3$, $l_2 = g_2 = (1 - s_{12} - d_2)/2$, $l_3 = 1 - s_{13} - d_3$, $l_1 = 0.4$, $g_1 = 0.3$, $s_{23} = c_{23} = 0$.

for high rates of clonal growth and low rates of herbivory can a genet escape senescence (fig. 4). This results since herbivory reduces a ramet to the first class, which is incapable of clonal reproduction, thus reducing a genet's production of clonal offspring.

At high values of α, sexual reproduction has a higher payoff than clonal reproduction does, λ decreases as the rate of clonal reproduction rises, and selection favors sexual reproduction and, as a result, genet senescence (fig. 5). For $\alpha < \alpha_{crit}$ selection instead works against the evolution of genet senescence. As the mortality rate of ramets increases, α_{crit} drops since less clonal reproduction occurs and a lower payoff of sexual reproduction is sufficient to balance clonal reproduction. Alternative assumptions ($s_{ji} \neq 0$, $c_{23} \neq 0$, fertility peaking at stage 1 or 3 rather than stage 2) alter the value of α_{crit} quantitatively but do not change the qualitative conclusion that for $\alpha > \alpha_{crit}$ selection favors sexual reproduction over clonal reproduction and as a result promotes genet senescence.

One might wonder, why not just set $f_1 = f_2 = f_3 = 0$ in the matrix **A** and calculate λ? Then if $\lambda < 1$, the probability of genet mortality increases with time, eliminating the need to calculate $M(t)$ according to equation (6). However, since the population of ramets that make up a genet is not necessarily at its stable age distribution at $t = \bar{a}$, the point when we evaluate whether a clone ages, one should not simply assume that a clone avoids senescence if $\lambda \geq 1$. Indeed, changes in mortality that occur much before $t = \bar{a}$ could be considered juvenile mortality, and those much after would be irrelevant if all clones had died by that

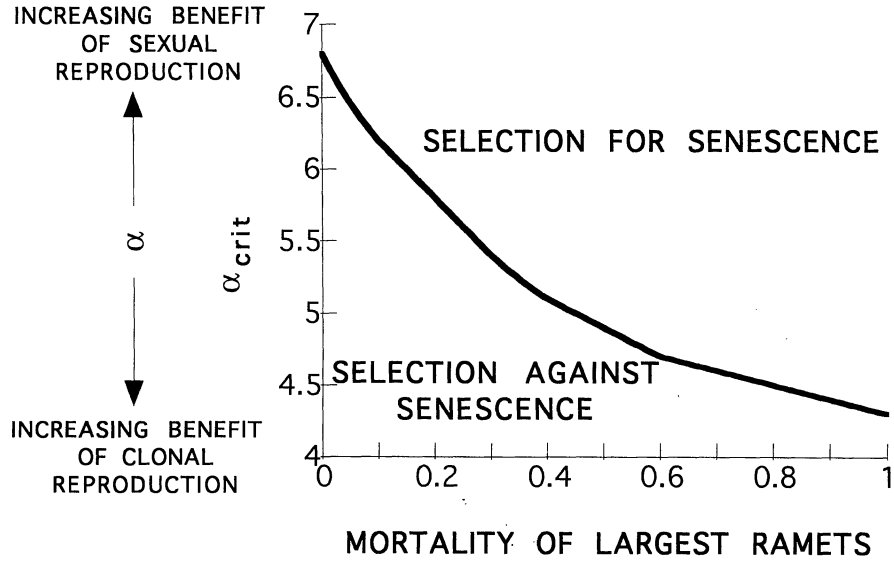

FIG. 5.—The value of α_{crit} versus the mortality rate, d_3, of ramets in stage 3. Parameters are the same as in figure 1, except $f_1 = \alpha(1 - c)$, $f_2 = 3\alpha(1 - c)$, $f_3 = 2\alpha(1 - c)$, and $l_3 = 1 - d_3$.

age. The key point is that one may not assume that the ramets composing a genet have a stable age distribution. In fact, a genet will not have a stable age distribution of ramets as the genet proceeds from the stage of one seedling, through the stage of being a juvenile genet composed of a few young ramets, and finally reaches a stable age distribution after many generations of clonal ramets within a single genet. In contrast, a population of ramets from many genets may be in equilibrium even if the ramets within a particular newly produced genet are not. As Pedersen (1995) notes, the stable age distribution of ramets within a clone is different from the stable age distribution of the total ramet population from all genets since the latter includes ramets from new genets arising from sexual reproduction. However, for these analyses we wish to examine the probability of mortality of a single genet, determined by the demography of that clone's ramets that may not have yet reached a stable age distribution, rather than the fitness of a population of many genets.

The strength of selection at different ages (measured as the change in fitness brought about by a given change in the rate of ramet survival or clonal or sexual reproduction at different ramet ages; eq. [16]) may increase with genet age for very high rates of clonal reproduction and low rates of ramet mortality (figs. 6, 7). Such selection weeds out deleterious mutations occurring in old clones and favors pleiotropic alleles conferring advantages to older rather than younger genets, opposing the evolution of senescence. Rates of clonal proliferation must be high compared with rates of sexual reproduction for the strength of selection to increase with age. If sexual fertility is high, the strength of selection may

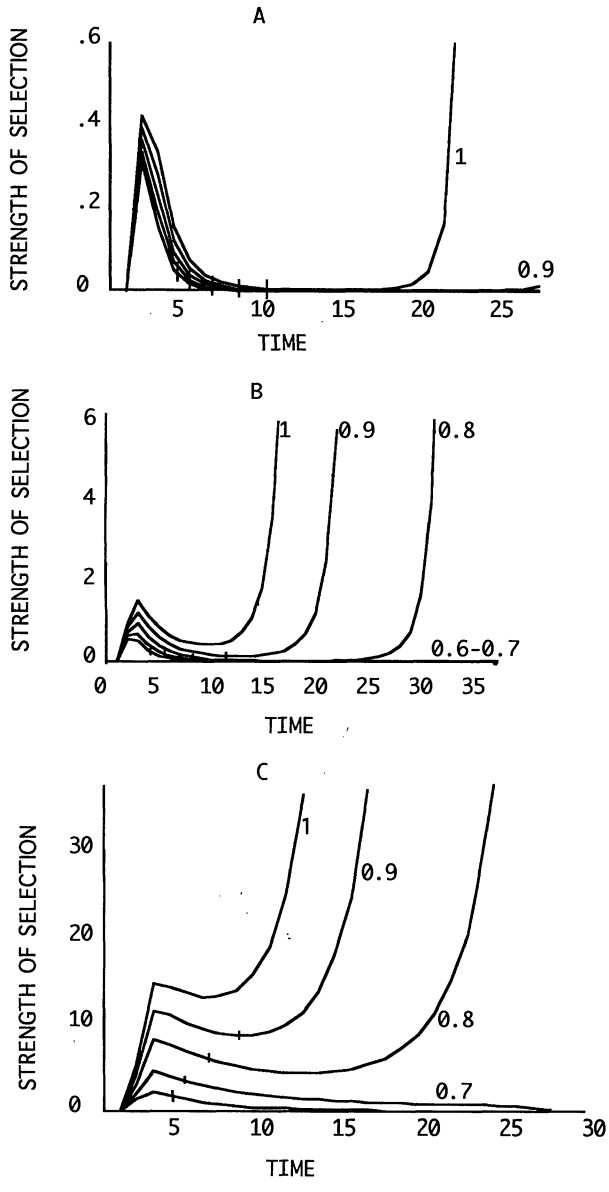

FIG. 6.—The strength of selection on a genet, Φ_z, versus time for $c_{12} = c_{13}$ ranging from 0.6 to 1. Values of $c_{ji} < 0.6$ were not plotted since the strength of selection does not increase as the clone ages after the initial peak. $f_1 = x$, $f_2 = 3x$, and $f_3 = 2x$. *A*, $x = 50$; *B*, $x = 10$; *C*, $x = 0$. Other parameters are $l_1 = l_2 = 0.4$, $g_1 = g_2 = 0.3$, $l_3 = 0.8$, $c_{23} = s_{12} = s_{13} = s_{23} = 0$. To calculate Φ_z using equations (15) and (16) at a given genet age, l_3 was set to half its original value for one time interval. The results are the same if another entry in the matrix **B** is changed instead, for example, l_1.

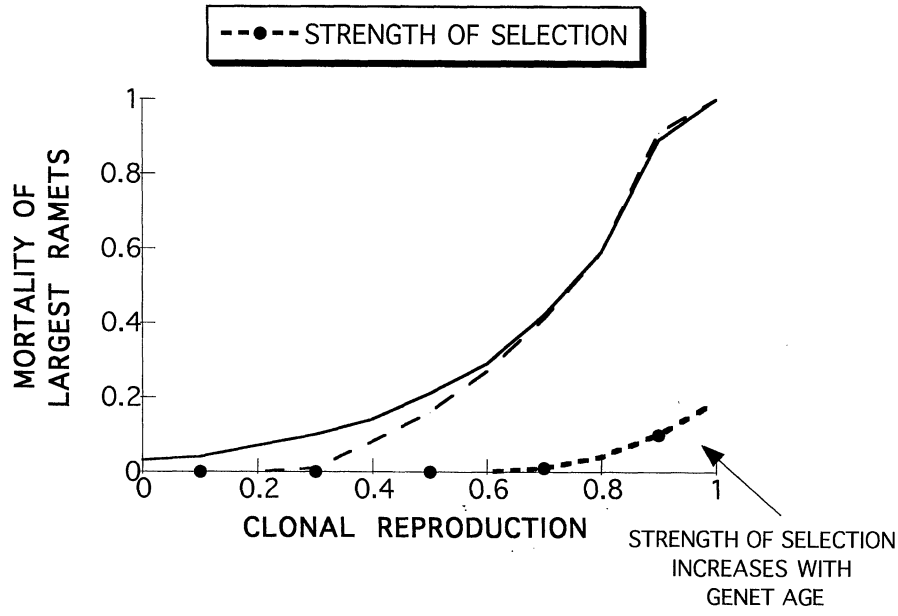

FIG. 7.—Parameter space (MSSM model) for the critical value of the mortality of ramets in the third class and the rate of clonal reproduction $c_{12} = c_{13}$, above which the strength of selection declines with genet age, while below which the opposite is true. The boundary curves for the occurrence of senescence in terms of an increasing probability of genet mortality or decreasing reproductive rate are included as in figure 2. Parameter values are the same as in figure 6*B*.

eventually increase but not until long after most clones would already be dead (fig. 6*A*). Lower rates of sexual reproduction result in an earlier increase in the strength of selection on the genet that begins when a clone is still likely to be alive (fig. 6*B, C*).

GS Model

In the GS model, we calculated the probability of genet mortality and the rate of sexual reproduction for a range of values of each of the mortality parameters (p, p_0, p_1), clonal reproduction parameters (b_0, b_1), and rate of ramet aging, h, separately while the other parameters remained constant. We plotted the probability of genet mortality (figs. 8, 9*A*), cumulative genet survival (figs. 8, 9*B*), and the number of sexual offspring produced (figs. 8, 9*C, D*) versus time for a range of values of b_0 (fig. 8) and b_1 (fig. 9). The number of sexual offspring produced depends on the values of s_0 and s_1, the number of sexual offspring produced by a ramet in the first or second stage, respectively. In figures 8*C* and 9*C*, first-stage ramets were more fecund than second-stage ramets were ($s_0 = 20$, $s_1 = 10$). In figures 8*D* and 9*D*, both young and old ramets were equally fecund ($s_0 = s_1 = 10$). We summarized the rates of change of genet mortality and fecundity in figures 10 and 11.

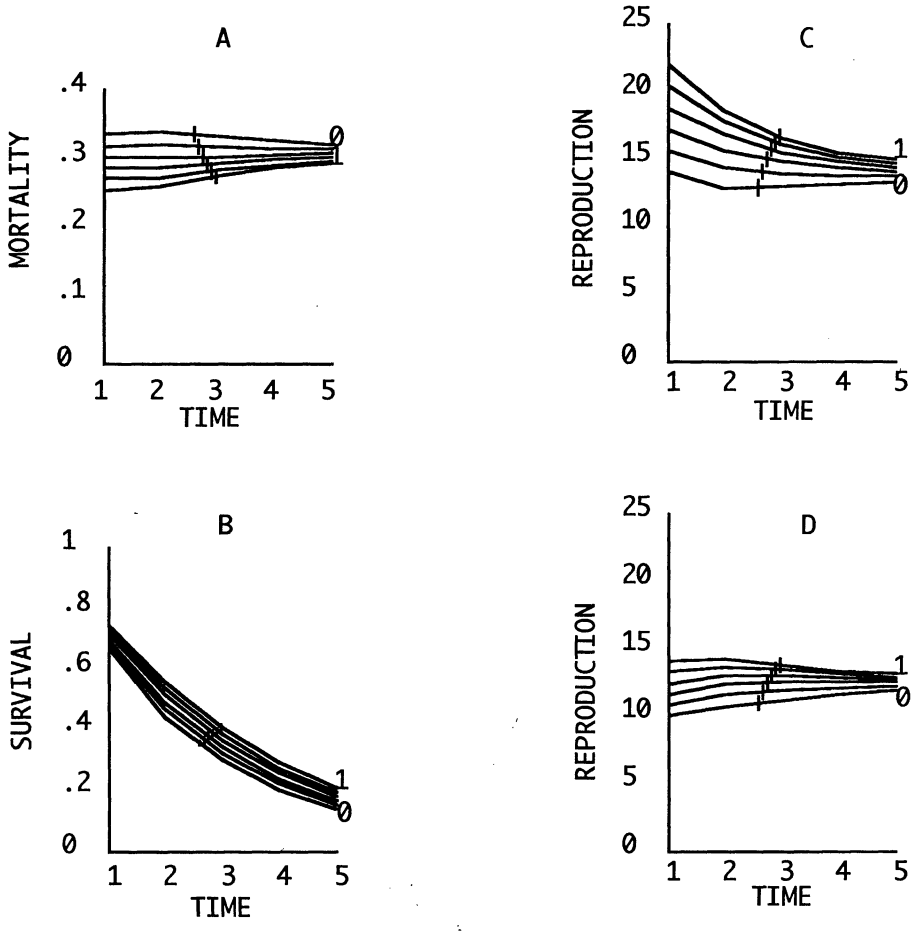

FIG. 8.—Predictions of the GS model for a range of values of b_0 ranging from 0 to 1 in intervals of 0.2 for the probability of genet mortality (*A*), genet survival (*B*), the number of offspring from sexual reproduction of the genet when $s_0 = 20$ and $s_1 = 10$ (*C*), and sexual reproduction when $s_0 = s_1 = 10$ (*D*). The expected genet life spans are indicated by ticks. Other parameters are held at $p = 0.1$, $p_0 = 0.2$, $p_1 = 0.3$, $h = 0.6$, and $b_1 = 0.1$.

The following qualitative patterns emerge. Senescence, in terms of a decline in the reproductive rate, is more likely if the fecundity of younger ramets is higher than that of older ramets ($s_0 > s_1$). The point at which senescence occurs—that is, where mortality begins to increase with age as measured by $M(t)$—does not usually coincide with that measured by reproductive rate, and senescence is more likely to be found in terms of the reproductive than in terms of the mortality criterion. In addition, lower $M(t)$ or $S(t)$ does not mean that a genet escapes senescence. Instead, senescence depends on the slope of the curves at $\bar{a}$ (figs. 10, 11).

As the external rate of mortality (p) rises, the rate of change of $M(t)$ grows

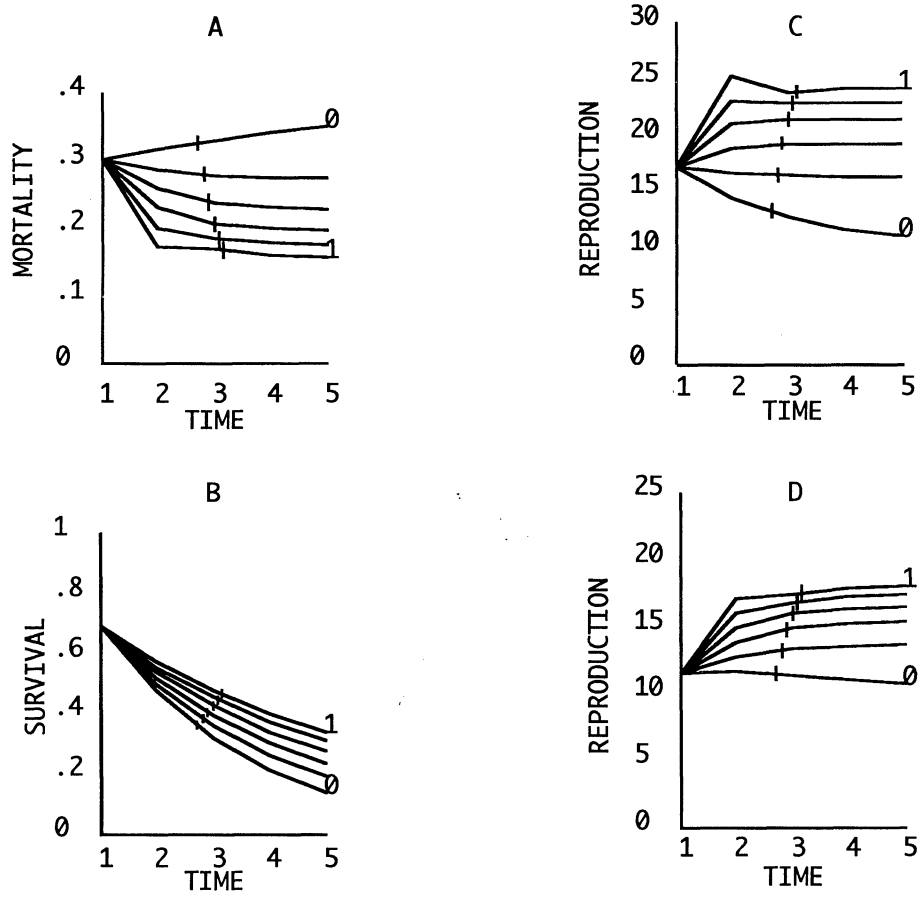

FIG. 9.—As in figure 8 except for a range of values of b_1 and the value of $b_0 = 0.4$

(fig. 10*A*) and that of $S(t)$ shrinks (fig. 11*A*). That is, an increase in externally imposed mortality leads to an increase in the rate of senescence; if there is a large chance that clones will be killed from stage-independent factors, then early reproduction and survival of younger genets is more important than later reproduction and survival of older genets, since a clone is likely to be killed by external causes before internal factors. However, for very large p, the curve $M(t)$ flattens out near 1, when all ramets immediately die. The jumps in figures 10*A* and 11*A* occur from using equations (11) and (12) when the integer part of $\bar{a}$ changes, a result of the discrete nature of our model for a continuous process (aging).

Increasing the mortality rate of younger ramets (p_0) leads to more rapid senescence in terms of reproduction (fig. 11*B*) but slower senescence and, eventually, an escape from senescence altogether in terms of mortality (fig. 10*B*). When a genet starts with one young ramet, $M(t)$ begins very high for large p_0.

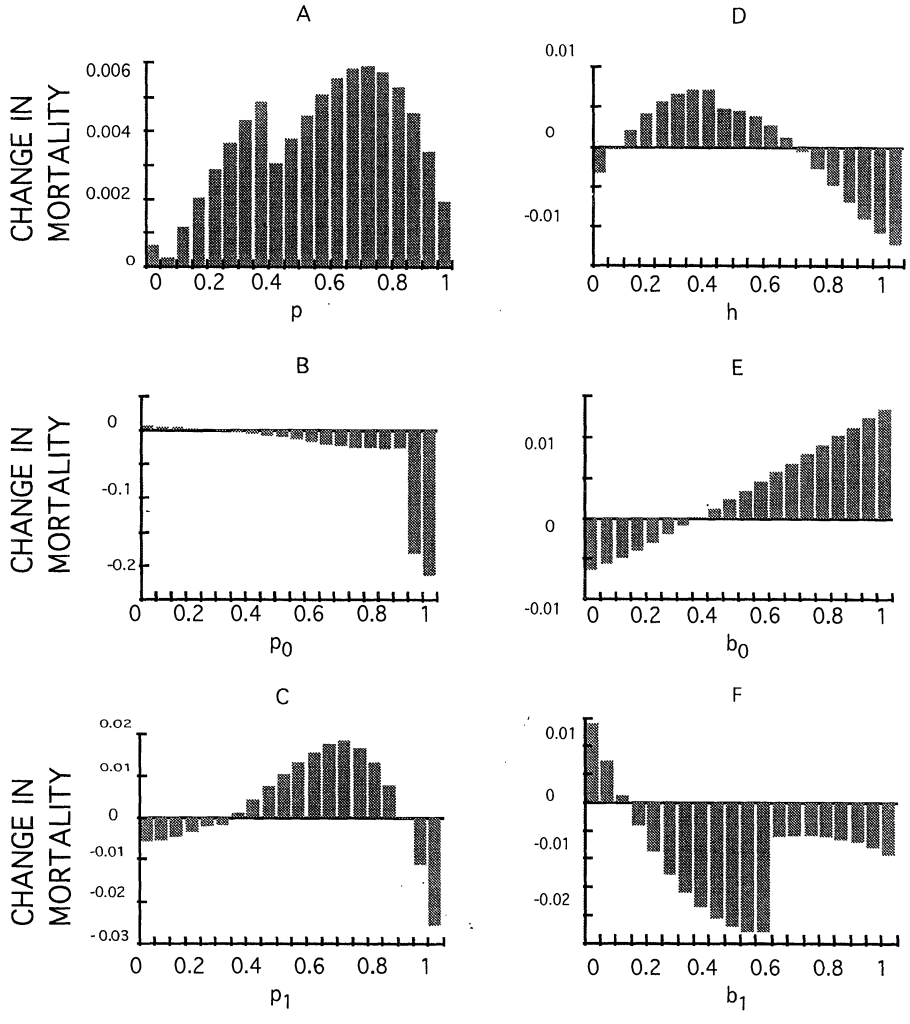

FIG. 10.—The change in the probability of genet mortality at $\bar{a}$ for a range of values of $p(A)$, $p_0(B)$, $p_1(C)$, $h(D)$, $b_0(E)$, and $b_1(F)$. Parameters that do not vary are held at values in figures 8 and 9. Positive changes indicate that the probability of genet mortality increases with time and that senescence occurs.

As the ramets of a genet age, then $M(t)$ drops rapidly. In terms of reproduction, however, high p_0 means that the clonal reproduction of a ramet, which is conditional on the survival of a ramet, rarely occurs, and $S(t)$ drops rapidly at the low $\bar{a}$.

The rate of senescence peaks for intermediate values of the survival of older ramets (p_1) for both reproduction and mortality (figs. 10*C*, 11*C*). The same is true for the rate of aging from a young to an old ramet, h (figs. 10*D*, 11*D*). For very low p_1 or h, as ramets age $M(t)$ declines. High p_1 pushes $\bar{a}$ to a small value,

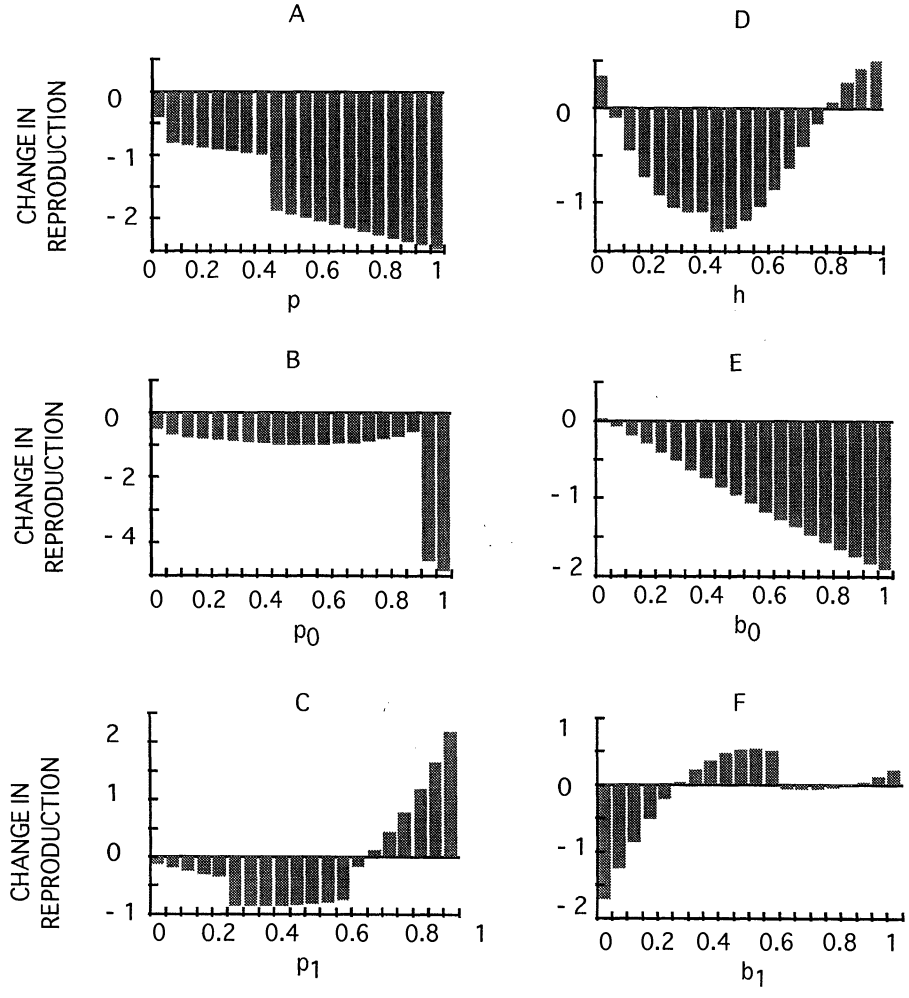

FIG. 11.—The change in the rate of sexual reproduction of a genet when $s_0 = 20$ and $s_1 = 10$. Otherwise the same as figure 10. However, negative values (declines in fertility with time) indicate clonal senescence.

where $M(t)$ and $S(t)$ are changing quickly. In addition, for very high p_1, because this model limits the number of ramets per genet to two, surviving genets more often have two young ramets (with lower mortality) than two old ramets. At low p_1, $\bar{a}$ is much bigger and $M(t)$ and $S(t)$ have already leveled off, so the rate of senescence is close to 0. For intermediate p_1, however, senescence is most rapid since $\bar{a}$ is intermediate and genets are composed of a combination of young and old ramets.

Higher rates of clonal reproduction by young ramets (b_0) lead to faster rates of senescence (figs. 8, 10*E*, 11*E*), while the opposite occurs for the clonal repro-

ductive rate of old ramets (b_1; figs. 9, 10*F*, 11*F*). This occurs because, as ramets age, the clonal reproduction of a genet depends more on the reproduction of older ramets than it did at earlier times. Although the slopes of $M(t)$ and $S(t)$ follow opposite patterns for b_0 and b_1, the clonal birth rates affect the value of $M(t)$ (declines for increasing b_0 and b_1) and $S(t)$ (increases for rising b_0 and b_1) similarly.

DISCUSSION

Our results are in accord with conclusions from other investigations of clonal senescence (Orive 1995; Pedersen 1995), namely, that it is possible for genets to decline in vigor with age despite regeneration through clonal reproduction and that details of life-history parameters determine whether a particular clone experiences senescence. Our results also suggest that one is more likely to find senescence in terms of a decline in the rate of sexual reproduction than in terms of an increase in genet mortality with genet age unless module fertility increases dramatically with ramet stage. This may be viewed as a shift in allocation as a clone ages from sexual to clonal reproduction. Clonal reproduction, particularly of older ramets, enables a clone to avoid senescence. If the rates of reproduction and survival of individual ramets decline with age, the genet is more likely to experience senescence. The senescence of individual zooids has been documented for some bryozoans (Palumbi and Jackson 1983) and may facilitate senescence of genets. When a trade-off between sexual and clonal reproduction is present, there may be selection for or against senescence as a result of selection on mode of reproduction, depending on the relative gain from clonal versus sexual investments. The finding that an increase in the externally imposed rate of mortality facilitates more rapid senescence (figs. 10, 11*A*) agrees with theory developed for unitary organisms (Hamilton 1966; Charlesworth 1970, 1980; Rose 1985) and empirical studies of the clonal bracken fern (Watt 1971).

Pedersen (1995) concluded that the elasticity of fitness to changes in the life-history traits of ramets decreased with ramet age and clone age, facilitating the evolution of ramet and genet senescence. The decline in the strength of selection was not necessarily monotone for all life histories, however, so that there was the possibility for selection against senescence at intermediate ages. Pedersen's results concluded that a significant amount of sexual reproduction must be present for clonal senescence to evolve. Our model also indicates that low rates of sexual reproduction can result in selection against senescence, provided that high rates of clonal reproduction accompany the low sexual contribution.

At the same time, we model senescence from a demographic as well as an evolutionary standpoint, and we show that the designation of clonal senescence may differ depending on the definition. From a demographic standpoint, in contrast to an evolutionary standpoint, the probability of genet mortality may decline for some life histories regardless of whether there is a significant amount of sexual reproduction. A population biologist measuring such a decline in the probability of genet mortality would categorize such a clone as nonsenescent

(Finch 1990; Finch et al. 1990). Eventually, however, if the evolutionary and the demographic interpretations of senescence do not concur, one expects that natural selection would not prevent the accumulation of genes with deleterious effects on survival and reproduction of old clones. Ultimately, this could lead to a decline in the rate of clonal reproduction and the appearance of demographic as well as evolutionary senescence. The same duality, in which demographic and evolutionary senescence do not always correspond, may also occur for unitary organisms; although life-history theory predicts that the strength of selection always falls with age for organisms incapable of clonal reproduction (Medawar 1946, 1952; Hamilton 1966), some empirical studies have detected an increase in the probability of survival of very old individuals (Carey et al. 1992; Curtsinger et al. 1992) .

Perhaps the rate at which the strength of selection (sensitivity of fitness to changes in life history; fig. 6) declines with clone age can help explain why some clones die quickly after going through a set number of cell divisions or reproductive periods (*Euplotes, Paramecium,* Finch 1990; bamboo, Harper and White 1974) and others have a less deterministic, more gradual decline. Our model leads to predictions that higher rates of sexual reproduction and lower rates of clonal reproduction result in a more rapid drop in the strength of selection with clone age following an initial peak. A sharp drop in selection pressure may lead to the sudden die-off of an entire clone in processes like programmed cell death and semelparity. Results from Bell (1984) support the prediction that the strength of selection declines with genet age when organisms engage in a significant amount of sexual reproduction, potentially leading to the evolution of clonal senescence; a comparison of six species of freshwater invertebrates revealed that in the four species in which sexual reproduction occurred, there was a senescent decrease in the rate of survival, but there was not a decline in the survival rates of those species reproducing vegetatively by paratomy.

Sometimes ramets are connected and function at a higher hierarchical level (Tuomi and Vuorisalo 1989; Schmid 1990). In such cases, it may be more biologically reasonable to structure the stages of a life-history matrix in terms of clonal fragments (or integrated physiological units; see Watson and Casper 1984) composed of multiple connected ramets, rather than by stages of single ramets. It is not the size of the ramet but, instead, the size of the clonal fragment that affects the food-gathering ability and, hence, the growth and reproductive rate of the fragment (Hartnett and Bazzaz 1983; Callaghan et al. 1986; McFadden 1986; Hughes 1989; Alpert 1991). Such a modification does not change the analysis or conclusions. It means that transition probabilities should be interpreted as stage-dependent reproductive and survival rates of clonal fragments rather than of ramets.

In addition, some clonal fragments have both age- and size-dependent life-history traits. For example, very young fragments that have attained a certain size through fusion, slightly older fragments that have reached the same size through clonal growth, and much older fragments that have become the same size by fission of larger, older fragments would all occupy different stage classes. This happens in some corals (Hughes and Jackson 1980; Hughes 1985).

Babcock (1991) found that corals that had been injured (fragmented) before were more likely to suffer injury or death than colonies that had never fragmented, and this difference could not be explained by size. Similarly, Hughes and Connell (1987) observed that young coral colonies grew faster than did old colonies at any size and that all size classes included both young and old colonies. Older fragments may grow more slowly since they invest more resources in regenerating damaged tissues (Bak 1983). Our models predict that lower reproductive rates and higher mortality rates for older stages (until unrealistically high mortality rates are reached) lead to faster rates of senescence. For this reason, a traditional matrix analysis (e.g., Orive 1995), in which it is impossible to differentiate between retrogression and clonal growth, might be biased toward finding no senescence.

We assumed that clonal organisms can maintain their genetic integrity (Schmid 1990) so that clonal siblings are essentially genetically identical compared with sexual offspring. Some authors have suggested that somatic mutations are effective in promoting the evolution of modular organisms (Whitham and Slobodchikoff 1981; Gill and Halverson 1984; Klekowski and Kazarinova-Fukshansky 1984*a,* 1984*b;* Whitham et al. 1984; Gill 1986). However, the genotypic variation among ramets of a clone will be much smaller than the variation among clones unless somatic mutation rates greatly exceed gametic ones (Slatkin 1985). Even if somatic mutations are common, intraorganismal selection effectively weeds out deleterious somatic mutations, and genets retain their genetic identity for long periods of time (Otto and Orive 1995). In field situations, it seems that vertical inheritance of somatic mutations is very rare (Hardwick 1985; Watkinson and White 1985). Although over the long term a clone may accumulate mutations, we think that the assumption is justified in this analysis that clonal offspring are essentially part of the same genetic individual as the parent clone.

We have examined two concepts of senescence: demographic, in which the probability of survival or the rate of sexual reproduction declines with genet age, and evolutionary, in which the strength of selection declines with genet age. It is possible that these two interpretations of senescence do not always correspond. According to the MSSM model, it is possible for a clone not to experience senescence in the demographic sense (genet mortality does not increase with age) but, nevertheless, to be exposed to evolutionary pressure that may lead to senescence (stronger selection on characters expressed earlier in the life of the genet than later; fig. 7). Only with low rates of sexual reproduction compared with sufficiently high rates of clonal reproduction does the strength of selection increase with clone age, favoring the evolution of an escape from clonal senescence. Perhaps this can help explain why some cancer cells not only divide more quickly than normal cells do, they also live longer, becoming immortal in culture (Tingley 1996). In contrast, normal animal cells can divide only a limited number of times in culture (Guarente 1996). Thus, a cancerous mutation resulting in the population dynamic process of a faster rate of cell division might boost clonal reproduction high enough to affect an evolutionary process, selecting for the loss of senescence in tumor cells.

ACKNOWLEDGMENTS

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APPENDIX A

Matrix Formulation (MSSM Model) for an Alternate Schedule of Mortality and Clonal Reproduction

In the MSSM model, if survival, growth, herbivory, or death occur before clonal reproduction takes place, then two changes in the analysis provided above are needed. It is still true that

$$l_i + g_i + \sum_j s_{ji} + d_i = 1,$$

but instead of the new c'_{ji} being unconstrained as when clonal reproduction occurred before ramet death, now the probability of clonal reproduction used in the matrix must be conditional on ramet survival. That is,

$$\begin{aligned} c'_{ji} &= \Pr\{\text{ramet survives to state } i \text{ and then clones new ramet in state } j\} \\ &= \Pr\{\text{ramet remains in state } i \text{ and then clones ramet in } j\} \\ &\quad + \Pr\{\text{ramet shrinks to state } i \text{ and then clones ramet in } j\} \\ &\quad + \Pr\{\text{ramet grows to state } i \text{ and then clones ramet in } j\}, \end{aligned} \tag{A1}$$

or, equivalently, in terms of the original c_{ji}, l_i, s_{ji}, and g_i,

$$c'_{ji} = c_{ji} l_i + c_{ji} s_i^* + c_{ji} g_{i-1}, \tag{A2}$$

where s_i^* is the probability of shrinking to state i:

$$s_i^* = \sum_{x=i+1}^{3} s_{ix}. \tag{A3}$$

Since a ramet must survive in order to clone offspring, $c'_{ji} < l_i + s_i^* + g_{i-1} < 1$. This is the first difference from the case when clonal reproduction happens before mortality (in that case $l_i + s_i^* + g_{i-1}$ may be greater than, equal to, or less than c_{ji}, but both $l_i + s_i^* + g_{i-1} \leq 1$ and $c_{ji} \leq 1$). The matrix **B** is the same except that c'_{ji} replaces c_{ji}. The second difference is that the probability of genet mortality does not require the death of clonal offspring, only the death of all the ramets:

$$M(t) = \prod_{i=1}^{3} d_i^{n(i,t)}. \tag{A4}$$

The results using equations (A1)–(A4) are virtually identical to those in the main text.

APPENDIX B

TABLE B1

TRANSITION PROBABILITIES BETWEEN STATES FOR THE GS MODEL

Transition from (j) to Transition (i)	Transition Probability $q(i, j)$
g_1 (0, 0):	
$g_{2\text{-}6}$	0
g_2 (0, 1):	
g_1 (0, 0)	$p + (1 - p)p_0$
g_2 (0, 1)	$(1 - p)(1 - p_0)(1 - h)(1 - b_0)$
g_3 (0, 2)	$(1 - p)(1 - p_0)(1 - h)b_0$
g_4 (1, 0)	$(1 - p)(1 - p_0)h$
g_5 (2, 0)	0
g_6 (1, 1)	0
g_3 (0, 2):	
g_1 (0, 0)	$p^2 + 2p(1 - p)p_0 + p_0^2(1 - p)^2$
g_2 (0, 1)	$2[p + (1 - p)p_0](1 - p)(1 - p_0)(1 - h)(1 - b_0)$
g_3 (0, 2)	$(1 - p)^2(1 - p_0)^2(1 - h)^2 + 2[p + (1 - p)p_0](1 - p)(1 - p_0)(1 - h)b_0$
g_4 (1, 0)	$2[p + (1 - p)p_0](1 - p)(1 - p_0)h$
g_5 (2, 0)	$(1 - p)^2(1 - p_0)^2h^2$
g_6 (1, 1)	$2(1 - p)^2(1 - p_0)^2h(1 - h)$
g_4 (1, 0):	
g_1 (0, 0)	$p + (1 - p)p_1$
g_2 (0, 1)	0
g_3 (0, 2)	0
g_4 (1, 0)	$(1 - p)(1 - p_1)(1 - b_1)$
g_5 (2, 0)	0
g_6 (1, 1)	$(1 - p)(1 - p_1)b_1$
g_5 (2, 0):	
g_1 (0, 0)	$p^2 + 2p(1 - p)p_1 + p_1^2(1 - p)^2$
g_2 (0, 1)	0
g_3 (0, 2)	0
g_4 (1, 0)	$2[p + (1 - p)p_1](1 - p)(1 - p_1)(1 - b_1)$
g_5 (2, 0)	$(1 - p)^2(1 - p_1)^2$
g_6 (1, 1)	$2[p + (1 - p)p_1](1 - p)(1 - p_1)b_1$
g_6 (1, 1):	
g_1 (0, 0)	$[p + (1 - p)p_0][p + (1 - p)p_1]$
g_2 (0, 1)	$[p + (1 - p)p_1](1 - p)(1 - p_0)(1 - h)(1 - b_0)$
g_3 (0, 2)	$[p + (1 - p)p_1](1 - p)(1 - p_0)(1 - h)b_0$
g_4 (1, 0)	$[p + (1 - p)p_0](1 - p)(1 - p_1)(1 - b_1) + [p + (1 - p)p_1](1 - p)(1 - p_0)h$
g_5 (2, 0)	$(1 - p)^2(1 - p_1)(1 - p_0)h$
g_6 (1, 1)	$(1 - p)^2(1 - p_1)(1 - p_0)(1 - h) + [p + (1 - p)p_0](1 - p)(1 - p_1)b_1$

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