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Genetic analysis of risk in clonal populations of forest trees *

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Summary. A major concern arising from the culture of clonally propagated crops of forest trees is risk of catastrophic loss due to an agent or event not anticipated at the time of population establishment. Since danger of such a catastrophe depends to some degree on the genetic variability within clonal mixtures, attention has been focused on the number of clones needed to keep the risk of catastrophic loss below specified levels. In this paper, we describe a genetical analysis of susceptibility to a destructive agent and the effect that frequency of genes for susceptibility have on the number of clones needed to effectively manage this risk. As a part of the analysis, parameters representing the minimum unacceptable mortality rates in plantations (β) and acceptable levels of risk (α) are defined, and their effects on the number of single-pair matings needed for the production of clonal stock are evaluated. Dominance and recessive gene action models for a single two-allele genetic locus are investigated. Probabilities for plantation failure are functions of the gene frequency for the allele conferring susceptibility. These functions converge to zero for allele frequencies less than β but to one for frequencies greater than or equal to β. This convergence is periodic rather than monotonic, since probabilities for plantation failure increase rather than decrease over restricted ranges of increasing numbers of clones. Recessive and dominance gene actions are found to have different effects on the minimum number of clones needed to attain acceptable risk levels. For conditions in which substantial numbers of clones are required, selecting multiple clones per mating is an effective method for reducing the number of matings necessary to achieve acceptable risks.

Key words: Mixtures – Pair-wise matings – Multinomial distribution – Gene action – Catastrophic loss

Introduction

The number of clones to be used in mixtures or to be planted in blocks is an important consideration in the culture of clonally propagated crops of forest trees. Among the important facets of this problem, the effect of numbers of clones on the risk of catastrophic loss has received a great deal of recent attention (Kleinschmitt 1979; Burdon 1982; Libby 1982; Rauter 1982; Huehn 1986). In general, large numbers of clones were thought to be necessary to buffer populations against unanticipated climatic and biotic elements that can cause widespread mortality or reduction in growth. Analyses by Libby (1982) and Huehn (1986) revealed, however, that large numbers of clones can reduce risks to desirable levels only if the probability of choosing susceptible clones is lower than the mortality rate that is considered unacceptable for plantations. When the probability of selecting susceptible clones is low, increasing the number of clones beyond certain numbers only marginally reduces risk. Furthermore, Libby (1982) found that under certain conditions, monoclonal plantations do not result in unacceptable risks and that in some situations risks for these populations are less than for mixtures of two and three clones.

While these analyses yielded much valuable information about the number of components needed in clonal mixtures, genetic control of susceptibility to destructive agents was not considered and the relationship between the frequency of susceptible alleles and the minimum number of clones remained unexplored. As a result, ef-

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fects of genetic factors on the relationship between numbers of clones and risk of catastrophe in clonal populations are incompletely understood. In this paper, we investigate those effects for situations in which susceptibility to an unanticipated destructive event is controlled by a single genetic locus and genotypes are produced by a typical mating system such as is used in some tree breeding programs. We report the effects of: (1) the frequency of susceptible alleles, (2) dominance versus recessive gene action, and (3) sampling due to the mating system and limited number of selections per mating.

Genetic sampling theory

Genetic models

Consider a genetic model in which susceptibility to a pathogen, insect, or destructive physical force is controlled by a single locus with two alleles. In genetic studies of susceptibility to disease in crop plants, loci with recessive and completely dominant gene action have been found (see Christ et al. 1987 for a review). Similarly, in two forest tree species, susceptibilities to particular pathogens have been shown to be affected by a single genetic locus. Both recessive and completely dominant loci were found to be involved (Prakash and Heather 1986; Kinloch et al. 1970). It therefore is reasonable to investigate both types of gene action. We assume that all known sources of risk are being managed, thus our concern is with destructive agents that are unforeseen when populations are established.

We consider a base population from which a restricted number of individuals are drawn for mating to produce progeny from which ortets are selected for cloning. Since the destructive agent is not active in the base population, the locus governing susceptibility is assumed to be selectively neutral in the parent population and genotypes are distributed in Hardy-Weinberg proportions. In the mating system investigated, each individual selected is mated with only one other selected individual. From each such mating, n progenies are chosen as ortets to produce a clonal mixture with equal numbers of ramets per progeny. Since the locus for susceptibility is selectively neutral prior to population establishment, genotype at that locus does not affect the choice of progeny for cloning. We further assume that only individuals with the resistant phenotype survive following a destructive event.

This process for producing clonal mixtures is similar to the Norwegian Forest Research Institute's breeding program for Norway spruce (*Picea abies* L.) (Skroeppa 1982). Individuals are selected for mating and progenies are chosen as ortets for clonal propagation on the basis of growth and adaptive traits. Presumably, therefore, genotypes for a locus controlling susceptibility to an unidentified destructive agent are sampled without bias in the selection process.

Statistical theory

Let A indicate the allele for susceptibility to a destructive agent and a the allele for resistance. If the frequency of A in the base population is represented by p, the frequency of susceptible genotypes in the base population is p^2 for the recessive susceptibility model and $p\,(2-p)$ for the dominance model, because genotypes are distributed in Hardy-Weinberg proportions. The number of susceptible individuals that may result from a mating, however, depends on the genotypes of the parent involved. For a biallelic locus, six unordered pairs of genotypes make up the possible mating types. Since individuals are paired at random for mating, irrespective of genotype, the probability of obtaining a mating of type i can be obtained from the products of the frequencies of the genotypes involved in the mating. These probabilities (q_i) are functions of p, the frequency of the susceptible allele. The six possible mating types, their probabilities of occurrence, and probabilities for producing a susceptible progeny, indicated by s_i, are given in Table 1. If M pairwise matings are made and if n progeny per mating are selected for cloning, the resulting mixture would contain $N=Mn$ clones. Progeny of each such mating will fall into one of the $n+1$ different classes consisting of 0, 1, ..., n susceptible progeny. The probability of obtaining a mating with h susceptible progeny chosen from n progeny is

$$\theta_h = \binom{n}{h} \sum_{i=1}^{6} q_i\, s_i^h\, (1-s_i)^{n-h}. \tag{1}$$

From M matings, the probability of obtaining the set $a_0, a_1, \ldots, a_n$ is given by the multinomial distribution function

$$\Pr(a_0, a_1, \ldots, a_n) = M! \prod_{h=0}^{n} (\theta_h)^{a_h}/a_h!\,, \tag{2}$$

where a_h is the number of matings that produce h susceptible progeny. The θ_h, and thus the probabilities given by Eq. (2), depend on values for p because the q_i are functions of p. They also differ with type of gene action because the s_i profiles are different for the dominance and

Table 1. Mating types, their probability of occurrence (q_i) and probabilities for producing susceptible progeny (s_i)

Mating type	Probability of occurrence	Prob. of susceptible progeny	
		Recessive model	Dominance model
AA × AA	p^4	1	1
AA × Aa	$4p^3(1-p)$	1/2	1
AA × aa	$2p^2(1-p)^2$	0	1
Aa × Aa	$4p^2(1-p)^2$	1/4	3/4
Aa × aa	$4p(1-p)^3$	0	1/2
aa × aa	$(1-p)^4$	0	0

Table 2. Probabilities for obtaining h susceptible individuals (θ_h) from matings producing one, two, and three progeny

Progeny per mating	No. of susceptible individuals	θ_h	
		Recessive model	Dominance model
1	0	$1-p^2$	$(1-p)^2$
	1	p^2	$p(2-p)$
2	0	$1/4(1-p)(4+4p-3p^2-p^3)$	$1/4(1-p)^2(p-2)^2$
	1	$1/2p^2(1-p)(3+p)$	$1/2p(1-p)^2(4-p)$
	2	$1/4p^2(1+p)^2$	$1/4p(4+5p-6p^2+p^3)$
3	0	$1/16(1-p)(16+16p-21p^2-3p^3)$	$1/16(1-p)^2(4-3p)^2$
	1	$3/16p^2(1-p)(9-p)$	$3/16p(1-p)^2(8-5p)$
	2	$3/16p^2(1-p)(3+5p)$	$3/16p(1-p)^2(8+p)$
	3	$1/16p^2(3p+1)^2$	$1/16p(8+35p-30p^2+3p^3)$

recessive models. In our analysis we consider the cases when one, two, or three progeny per mating are selected for cloning. The θ_h expressed as functions of p for these cases are given in Table 2. When $n=1$, Eq. (2) reduces to the probability function for the binomial distribution, with parameter $\theta_1=p^2$ for the recessive model and $\theta_1=p(2-p)$ for the dominance model.

Two additional parameters are needed to evaluate the effect of number of clones in mixtures on risk of plantation failure. We assume that there is a minimum mortality rate such that any plantation with a lower rate can be profitably managed. Therefore, we define β to be the minimum unacceptable mortality rate due to the destructive agent. If the level of mortality in a plantation is greater than or equal to β, the plantation is considered to be a failure. We also assume that plantation managers have a maximum acceptable risk of plantation failure (α). A probability of plantation failure that is less than or equal to α is considered acceptable. The β and α parameters of our model are similar to the q and Q parameters of Huehn's treatment (Huehn 1986).

Plantation failure occurs when a plantation is subjected to a destructive agent and has a clonal composition with a proportion of clones susceptible to the agent that is greater than or equal to the minimum unacceptable mortality rate. The number of susceptible clones in a mixture is given by the linear function

$$y=\sum_{h=0}^{n} h a_h\,, \tag{3}$$

where $y=0, 1, 2, \ldots, N$. Potential for plantation failure (F) occurs when $y\geq\beta N$. Since our interest is in risk of plantation failure if a destructive event occurs, the probability of plantation failure, Pr (F), given a destructive event is an appropriate measure for this risk. We do not use the conditional probability notation since we are only interested in outcomes after a destructive event. Since Pr (F) equals Pr ($y\geq\beta N$), an expression for it must include the probability that a specific number of susceptible individuals represented by z are chosen to be ortets or Pr ($y=z$). This probability is obtained by summing the probabilities for obtaining each set $a_0, a_1, \ldots, a_n$ that produces exactly z susceptible progeny. Each of the probabilities involved in this summation is given by the multinomial probability distribution function presented in Eq. (2). Explicitly, Pr ($y=z$) is

$$\Pr(y=z)=\sum_{a_n=b_n}^{u_n}\cdots\sum_{a_3=b_3}^{u_3}\sum_{a_2=b_2}^{u_2} M!\prod_{h=0}^{n}(\theta_h)^{a_h}/a_h!\,, \tag{4}$$

where the b_i are the lower limits for the a_i and are given by

$$b_i=\begin{cases}\max\left[z-\sum_{j=i+1}^{n}(j-i+2)\,a_j-(i-1)\,M,\,0\right] & \text{for } i=2,\ldots,n-1\\ \max\left[z-(i-1)\,M,\,0\right] & \text{for } i=n\,,\end{cases}$$

the u_i are the upper limits for the a_i and are the largest integers such that

$$u_i\leq\begin{cases}\left[z-\sum_{j=i+1}^{n} j\,a_j\right]\Big/ i & \text{for } i=2,\ldots,n-1\\ z/n & \text{for } i=n\,,\end{cases}$$

a_1 is given by

$$a_1=\begin{cases}z-\sum_{j=2}^{n} j\,a_j & \text{for } n>1\\ z & \text{for } n=1\,,\end{cases}$$

and a_0 is

$$a_0=M-\sum_{j=1}^{n} a_j\,.$$

The collection of events, $E\equiv\{y\geq\beta N\}$, is the union of the mutually exclusive events $E_r\equiv\{y=z$ such that $z\geq\beta N\}$. Therefore, $\Pr(\mathrm{F})=\sum_r P(E_r)$. Since $P(E_r)$ is given by the result in Eq. (4), Pr (F) can also be expressed as

$$\Pr(\mathrm{F})=\sum_{z=k}^{N}\Pr(y=z)\,, \tag{5}$$

where k is the smallest positive integer such that $k\geq\beta N$. It should be noted that when $n=1$, Pr (F) reduces to the cumulative binomial distribution. Since Pr (F), except for the simplest case, is represented by a complicated expres-

sion, these probabilities were evaluated numerically to study the effect of number of clones on risk of plantation failure.

Results

Probability of plantation failure

Probability of plantation failure [Eq. (5)] for a fixed number of clones in a mixture is an increasing function of the allele frequency for susceptibility in both the recessive and dominance models. This is true when clones are derived from both single and multiple progeny per mating. An important parameter to consider in the analysis of these Pr (F) functions is the allele frequency that yields the frequency of susceptible genotypes ($\hat{p}$) in an infinite population, with mortality rate equal to the minimum unacceptable mortality rate, β. As the number of clones increases, the probability of failure Pr (F) converges toward zero if p is below $\hat{p}$, but for allele frequencies at or above $\hat{p}$, Pr (F) converges toward one. By definition, $\hat{p}=\sqrt{\beta}$ for recessive susceptibility and $\hat{p}=1-\sqrt{1-\beta}$ for the dominance model. Convergence of Pr (F) for both the recessive and dominance models is illustrated in Fig. 1 for $\beta=0.5$. In this figure, curves are shown for the Pr (F) functions of odd-numbered mixtures. For both gene action models, Pr (F) at any $p<\hat{p}$ declines toward zero with increasing numbers of clones in mixtures. Conversely, for any $p\geq\hat{p}$, Pr (F) moves toward a value of one as mixture size increases. In these illustrations $\hat{p}=0.707$ for the recessive model and $\hat{p}=0.293$ for the dominance model.

Interestingly, however, convergence of the Pr (F) functions with respect to the number of clones is not monotonic. While there is a general effect of decreasing Pr (F) with increasing N when $p<\hat{p}$, Pr (F) can actually increase over certain intervals of increasing N. A type of periodic behavior results in which there is a regular increase in Pr (F) with increasing N in each period, followed by an abrupt drop to the Pr (F) value at the N that begins the next period. Thus, if the period is of length 10, $\hat{p}=0.316$ and for $N=1$ to 10 and $N=11$ to 20, Pr (F) increases for all p; however, for $p<\hat{p}$ Pr (F) at $N=11$ is less than Pr (F) at $N=10$ and Pr (F) at $N=20$ is less than at $N=10$ (Fig. 2). The entire sequence of Pr (F) values with increasing N when $p<\hat{p}$ can be described in terms of the length of the period of increase and the number of such periods. Results consistent with this behavior were first reported by Libby (1982), who described a case in which monoclonal plantings had lower probabilities of failure than mixtures of two and three clones.

Because of this type of periodic pattern of convergence, the minimum number of clones required for Pr (F) $\leq\alpha$ always exists at the beginning of a period. The minimum number of clones such that all higher N have

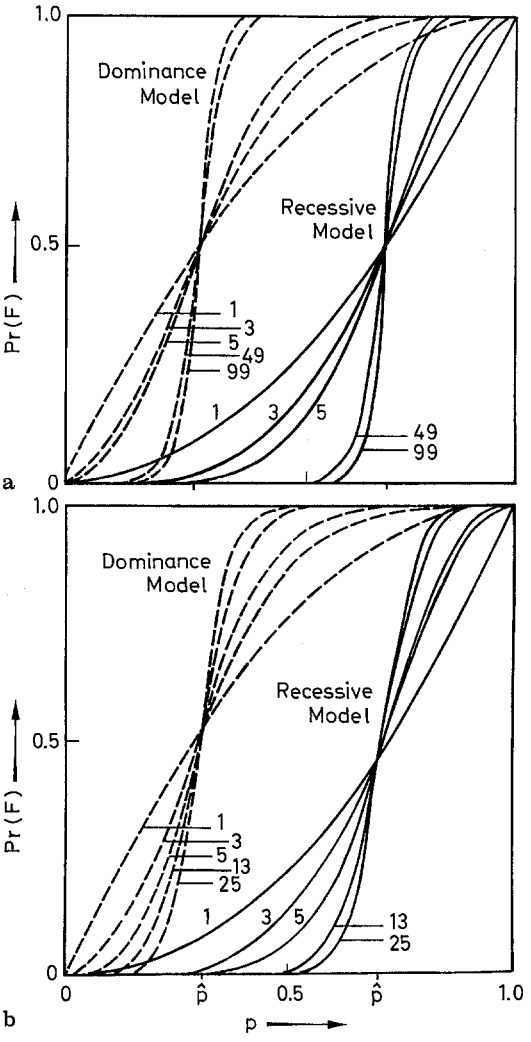

Fig. 1 a and b. Probability of plantation failure [Pr (F)] plotted against frequency of the susceptible allele (p) for increasing numbers of matings. *Solid curves* indicate Pr (F) for the recessive model. *Dotted curves* indicate Pr (F) for the dominance model. Curve numbers indicate number of matings; **a** curves for $\beta=0.5$ when one progeny per mating is selected and **b** curves for $\beta=0.5$ when three progeny per mating are selected

Pr (F) $\leq\alpha$, however, always occurs at the end of a period. Furthermore, period length increases with β^{-1}. Although period length fluctuates at some values of β, it is invariant for integer values of β^{-1} and, for these cases, the period length (ϱ) is given by $\varrho=\beta^{-1}$. For example when $\beta=1/2$, $\varrho=2$; for $\beta=1/3$, $\varrho=3$; for $\beta=0.2$, $\varrho=5$; and for $\beta=0.1$, $\varrho=10$.

Differences between the dominance and recessive models in probabilities of failure result from differences in the frequency of susceptible individuals in the base population. Such differences in Pr (F) functions for the two models are illustrated in Fig. 1 for $\beta=0.5$. In Fig. 3, curves representing the relationship between β and $\hat{p}$ for the two models are shown. In both models, allele frequen-

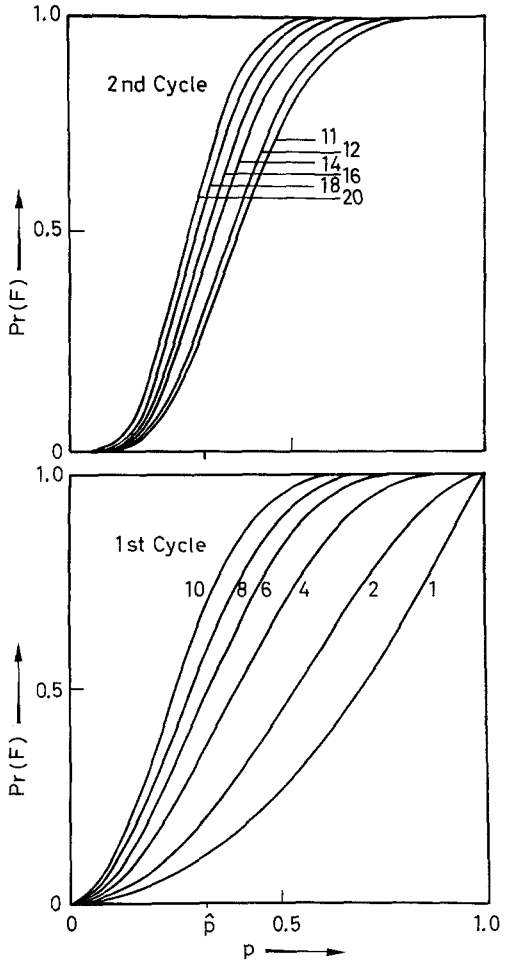

Fig. 2. Probability of plantation failure Pr (F) plotted against frequency of the susceptible allele (p) for two periods in the recessive susceptibility model whe $\beta=0.1$ and one progeny per mating is selected. Curves for mixtures in the second period are shown above curves for mixtures in the first period. Curve numbers indicate number of matings

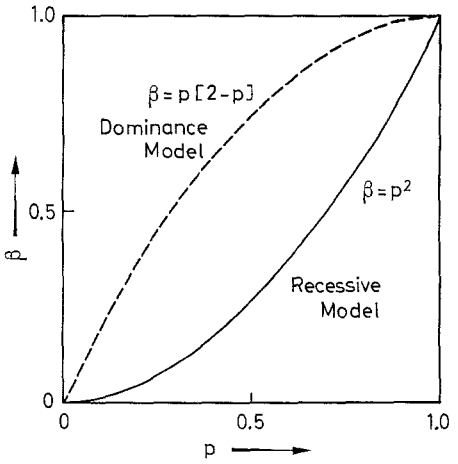

Fig. 3. Boundary curves for regions of convergence for probabilities of plantation failure [Pr (F)] when one progeny per mating is selected. In the region above a curve, Pr (F) values approach zero as the number of clones in mixtures become large. Below and along the curves, Pr (F) values approach one as the number of clones in mixtures becomes large

cies at or below the frequency that produces the designated maximum acceptable risk when a single clone is selected yield acceptable probabilities of plantation failure regardless of the value of β. For higher allele frequencies, however, acceptable values for Pr (F) only result at those allele frequency and β combinations that are above the appropriate curve in Fig. 3.

Except for extreme α or high β levels, it is not possible to reduce risks to acceptable levels by increasing the number of clones in populations having moderate and high allele frequencies when susceptibility is dominant. Even at moderately low allele frequencies, moderate α and β levels are required to achieve acceptable risks. For moderate β and α levels ($0.4 \leq \beta \leq 0.6$, $0.1 \leq \alpha \leq 0.2$), there are only short spans of allele frequencies for which the risk can be reduced to acceptable levels by increasing the number of clones. At the higher frequencies in these intervals, satisfactory risks can be achieved by using large numbers of clones, as is illustrated for $\beta=0.5$ in Fig. 1. There is considerably greater latitude, however, for achieving acceptable risks with the recessive susceptibility model.

Selection of a single progeny per mating

Reasonably low risk of catastrophic loss can be obtained with monoclonal cultures for both gene action models when allele frequencies for susceptible alleles are low. At high frequencies for the susceptible allele, it is not possible to achieve low or even moderate values for Pr (F) with any N except at very high levels of β (Fig. 3). For intermediate allele frequencies, however, reasonable values for Pr (F) can be obtained at intermediate and high levels of β with multiclonal mixtures.

We now discuss results for cases in which a single progeny from each mating is chosen for cloning and confine our attention to the recessive susceptibility model, since the dominance model has a restricted number of conditions for achieving low to moderate risk levels. The number of clones in this context then also indicates the number of matings. To display the joint effects of allele frequency (p) and minimum unacceptable mortality rate (β) on the minimum number of clones required for $\Pr(F) \leq \alpha$, we partition the β, p plane into sections. This is easily done when only one progeny is selected per mating because then the Pr (F) functions are cumulative binomial distribution functions. The upper value for the allele frequency that defines the partition for a particular range of β values and number of clones can be obtained for a specific α level by equating the Pr (F) function for

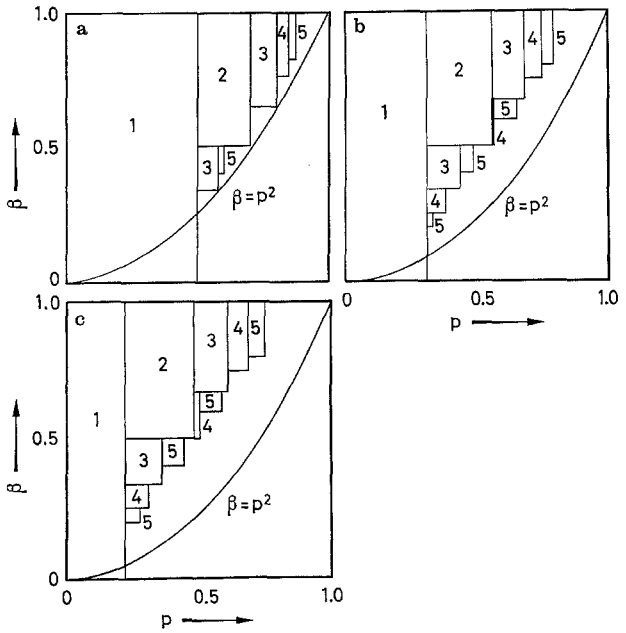

Fig. 4a–c. Regions of β, p plane where the specified minimum numbers of clones are required to reduce probability of plantation failure [Pr (F)] below fixed levels of acceptable risk (α). **a** $\alpha=0.25$, **b** $\alpha=0.10$, and **c** $\alpha=0.05$

Table 3. Max-min N for the recessive susceptibility model when $\alpha=0.1$ and a single progeny is selected per mating

Allele frequency	β values			
	0.1	0.25	0.4	0.5
0.05	1	1	1	1
0.10	1	1	1	1
0.15	11	1	1	1
0.20	31	5	1	1
0.25	91	5	3	3
0.30	>100	9	3	3
0.35	0	17	6	3
0.40	0	37	6	5
0.45	0	>100	11	7
0.50	0	0	21	9
0.55	0	0	46	13
0.60	0	0	>100	27
0.65	0	0	0	79
0.70	0	0	0	>100

these conditions to the value for α and solving for p. Since these partitions are for minimum numbers of clones, the lower allele frequency for a monoclone partition is $p=0$. For a multiclonal partition, the lower frequency corresponds to the p value at the point that the partition intersects that for a mixture with a smaller number of clones. For the recessive model, minimum numbers of clones for three α levels are presented in Fig. 4 for mixtures of up to five clones.

The portion of the β, p plane above the curve, $\beta=p^2$, can be completely partitioned if mixtures with enough clones are considered. The number of clones in the largest mixture required to cover this area depends on the value of α. The area on and below the curve and to the right of the one-clone block contains β, p combinations for which the specified α cannot be attained regardless of the number of clones employed. Increasing the number of clones for these combinations results in an increase in Pr (F). It is apparent that the minimum number of clonal components necessary to achieve low to moderate risk levels is five or less for a large portion of the β, p plane where such risk levels are attainable. Larger numbers of clones are required in the area directly above the $\beta=p^2$ curve to reduce the risk to practical levels. This area increases as α declines. For β, p combinations in this region, acceptable probabilities of plantation failure can only be obtained by using large numbers of matings for the production of ortets. There is a practical limit, however, to the area in this region for which acceptable probabilities of plantation failure can be realized, since for low α values, large mixtures are necessary to cover the β, p combinations that correspond to points near the $\beta=p^2$ boundary curve. In the present context, this implies a large number of pairwise matings.

Since convergence of the Pr (F) functions is periodic rather than monotonic, multiple minima may exist for numbers of clones that produce probabilities of plantation failure below a fixed α value. For some purposes, it might be appropriate to consider the maximum value of these minima for determining an appropriate number of clones for mixtures rather than the smallest minima. Such a maximum of a minimum number (max-min N) for a fixed α is the smallest number of clones in a mixture for which $\Pr(F)\leq\alpha$ but also for which all mixtures with larger number of clones have $\Pr(F)\leq\alpha$. Because of this property, max-min N serves as a useful benchmark number for indicating that the desired risk level is assured if mixtures with that or a larger number of clones are used. In this respect it differs from the minimum number of clones criterion but is similar to the necessary number of components in mixtures defined by Huehn (1986). Max-min N for $\alpha=0.1$ and $\beta=0.1$, 0.25, 0.4, and 0.5 are presented in Table 3. The range of allele frequencies for which moderate to large numbers of clones are useful is narrow, just as it is for minimum N. On the basis of these results, approximately 30–35 matings are sufficient to provide progeny for use as clonal ortets over a wide range of allele frequencies (p) and minimum unacceptable mortality rates (β).

Selection of multiple progenies per mating

Probabilities of plantation failure for selection of two and three progeny per mating were compared to those ob-

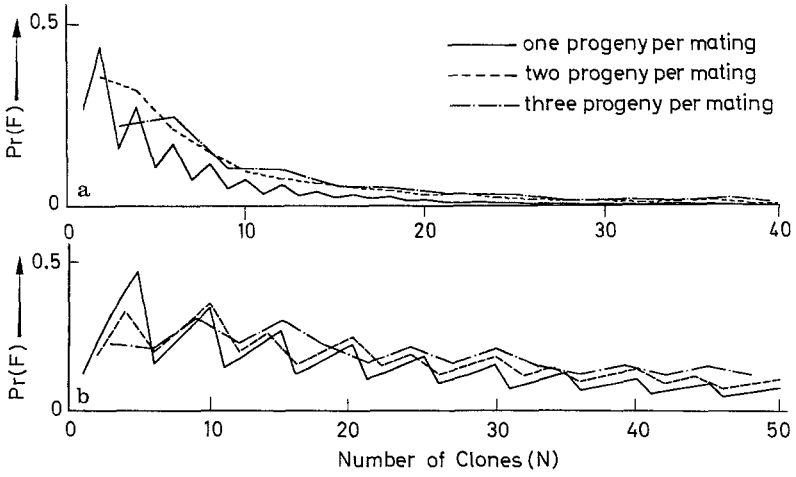

Fig. 5a and b. Probabilities of plantation failure [Pr (F)] for the recessive model plotted against number of clones. **a** $\beta = 0.5$ and $p = 0.5$, and **b** $\beta = 0.2$ and $p = 0.35$

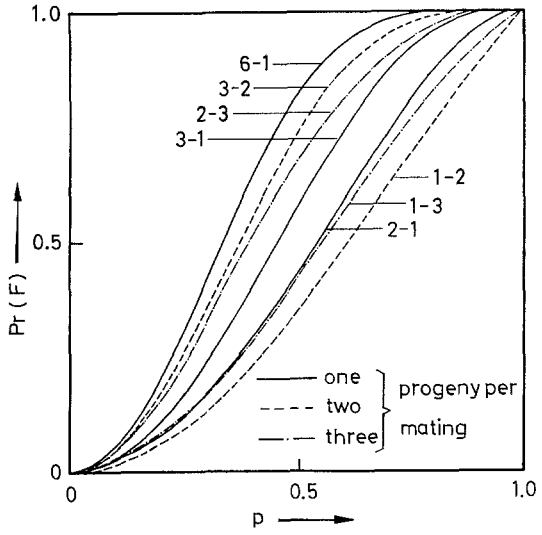

Fig. 6. Probabilities of plantation failure [Pr (F)] for the recessive model when selection involves different numbers of progeny per mating at $\beta = 0.1$. The first number of the curve designations indicates number of matings, the second number indicates the number of progeny per mating

tained for single progeny selection. Convergence of Pr (F) functions for multiple progenies per mating is periodic, just as it is for the single progeny per mating selections. The periodic behavior, however, depends on the total number of clones N, rather than on number of matings M. Pr (F) values for the different numbers of progeny selections, therefore, were compared for a fixed number of clones. Values for multiple numbers of progeny are lower than those for single progeny selections in mixtures of the first Pr (F) period (Figs. 5 and 6). The reverse pattern occurs in subsequent periods (Fig. 5). As expected, differences in Pr (F) values for different numbers of progeny per mating decrease as the number of clones is increased. Such a decrease is especially evident in Fig. 5a.

Selecting multiple progeny per mating offers little advantage over choosing a single progeny per mating for those β, p combinations that yield acceptable risks with small numbers of clones. Selecting multiple progeny can offer advantages, however, for β combinations that require moderate numbers of clones to reach desired risk levels. In these situations, comparable risks to those achieved by single progeny selections can be attained with substantial reductions in the number of matings and only a modest increase in the number of clones. As previously discussed, however, at moderate α levels, the area containing β, p combinations for which this is true is not large.

Discussion

Mode of gene action for susceptibility to an unforeseen catastrophe has a substantial impact on the numbers of clones to include in mixtures, in order to reduce risk of plantation failure. When susceptibility is controlled by a locus with recessive gene action, acceptable risks can be achieved for an array of conditions. For a number of minimum unacceptable plantation mortality rates (β), only moderate risks of plantation failure result for a wide range of allele frequencies when mixtures contain a small number of clones. A more conservative strategy with larger multiclonal mixtures is called for if mixtures are to be grown in conditions for which β values are variable or subject to change, or in circumstances for which β is unknown. Likewise, if the maximum risk that is acceptable (α) is reduced, larger mixtures are needed to adequately cover the range of possible allele frequencies.

If susceptibility is controlled by a dominant gene, options for reducing risks are limited. Increasing the number of clones does not lower risks to acceptable levels if the susceptible allele occurs at middle and high frequencies. For low frequencies, acceptable risks can be ob-

tained with mixtures of from one to three clones. Thus, for catastrophic events in which susceptibility is controlled by dominance gene action at a single locus, monoclones or two- and three-clone mixtures do not, in general, produce appreciably greater risks than larger multiclonal mixtures.

For conditions in which moderate or large multiclonal mixtures are necessary, selection of multiple progeny per mating is attractive. A large number of pairwise matings is required for these conditions if a single individual is selected from the progeny of each mating. If multiple progeny per mating can be selected for cloning, the number of matings can be reduced, with only a modest increase in number of clones for equivalent risks. In some circumstances, the use of multiple ortets per family may be necessary to provide sufficient numbers of plantlets to meet planting demands. For this situation, then, the number of matings required to produce ortets so that risk of plantation failure is acceptable will be somewhat less than the number of matings required when a single progeny is selected from each mating.

Finally, we should point out that our model is only a first approximation to behavior of destructive agents in nature. We assume, e.g., that all components of a mixture are equally exposed to the destructive agent and that all susceptible individuals die as result of that exposure. We do not consider the effect of number of susceptible individuals on the growth and success of a destructive agent and have not incorporated in our model the epidemiology of the destructive agent. Introduction of these effects into the analysis of risk for mixtures has been undertaken by Huehn (1986). Incorporation of such effects into the analysis of risk for models with genetic control of susceptibility should be further enlightening.

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