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ON NUMBERS OF CLONES NEEDED FOR MANAGING RISKS IN CLONAL FORESTRY

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ABSTRACT

An important question in clonal forestry concerns the number of clones needed in plantations to protect against catastrophic failure while at the same time achieving the uniform stands, high yields, and ease of management associated with this management system. This paper looks at how the required number of clones needed to achieve a predetermined maximum acceptable level of risk changes as underlying system parameters – level of loss acceptable to the plantation manager; number or severity of pest attacks; level of clonal resistance to attack; and gene frequencies associated with 'susceptible' alleles – increase or decrease. In general, the number of clones needed decreases as the intensity of pest attack increases, and increases if any of the other quantities increases. An explanation of these trends is offered in terms of risk-prone vs. risk-averse behavior, and implications for governmental regulations and forest management are discussed.

Key words: clonal forestry, risk, numbers of clones, risk prone and risk averse strategies

INTRODUCTION

For some forest species it is now possible to plant large tracts with propagules of a few highly productive clones. While such plantings have potential for high yield and are more easily managed than mixed stands, there is a risk that some or all of the clones chosen may be susceptible to attack by an insect or pathogen unforeseen as a problem at the time of planting. Concern about the possibility of extensive population failures with large scale adoption of clonal culture arises because a number of severe pest outbreaks have occurred when genetic diversity has been greatly restricted in production populations. The devastating effect of the 1970 southern corn leaf blight epidemic on maize grown in the United States is one example from agriculture where extreme genetic uniformity resulted in an undesirable outcome. At the time of the 1970 epidemic, the preponderance of commercial maize grown in the United States contained a single cytoplasmic clone, with 85% of the crop carrying male sterile cytoplasm (cms-T) derived from a single progenitor (LEVINGS 1990; ULLSTRUP 1972). This cytoplasmic uniformity contributed to the evolution and spread of a new race of pathogen that is extremely virulent on plants with cms-T cytoplasm.

Similar events have occurred occasionally in clonal forestry. Examples in Europe include the outbreak of *Venturia populina* in poplar plantations in Italy during the 1930's, and the devastation caused in the 1970's by the spread of *Marssonina brunea* through poplar stands following widespread monoclonal planting of clone I-214 (ZSUFFA *et al.* 1993; HEYBROEK 1978). In Australia, an outbreak of leaf rusts severely damaged poplar clonal plantations during 1972 and 1973, and caused a major disruption in the effort to establish poplar clonal culture on that continent (PALMBERG 1978).

First attempts to control the risk associated with clonal plantations were legislative, as members of the European Community, particularly Sweden and the Federal Republic of Germany, mandated minimum numbers of clones to be used, the numbers ranging from 20 to over 100 depending on the species and other considerations (HEDSTRÖM & KRUTZSCH 1982; MUHS 1982, 1993). Subsequent proposals for regulation of clonal materials have been considered in Belgium, Canada, Denmark, and New Zealand (MUHS 1993).

Implicit in these regulations is the assumption that using more clones will lead to a smaller chance of stand failure. It turns out, however, that the situation is more complex. Studies by LIBBY (1982) and HÜHN (1986) using mathematical models of risk suggest that in some circumstances a larger number of clones actually introduces greater risk than a smaller number. BISHIR & ROBERDS (1995, 1997) generalize these models and present examples showing that risk can decrease, remain almost constant, or surprisingly, increase substantially as the number of clones increases.

In a recent study, BISHIR & ROBERDS (1997) present theoretical arguments which suggest that in general the level of risk is unlikely to change significantly after the number of clones used exceeds about 30 or 40. Use of very large numbers thus appears not only unattractive commercially, but unnecessary. However, there remains the question of when and why it sometimes is better to use a moderate number of clones rather than many.

Three methods of resolving this conundrum are presented in this paper. Each leads to the conclusion that, in general, a larger number of clones is appropriate when the risk of plantation failure is small relative to the level of risk one is willing to accept, while a smaller number of clones can be better when the reverse situation obtains. The second and third approaches suggest why these trends occur.

ANALYSIS USING COMPUTER SIMULATION

To gain insight into the problem, we simulated a simplified form of the model for analysis of risk in clonal plantations set out in BISHIR & ROBERDS (1995). Because the criteria used in our model may not be those considered important in government regulation or in the management of a particular plantation, we make no claim that the particular numbers of clones deemed 'optimal' in the model simulation are in fact numbers that should be used in clonal plantings. Rather, our goal is one of observing the trends in the numbers obtained, and seeking a general explanation of these trends.

Briefly, the model used postulates a base population that has been generated by random mating and selection, and from which individuals are chosen for phenotypic traits such as form, growth rate, fruit production, etc., to serve as ortets for production of genetically identical individuals by vegetative propagation. The collection of ramets (individual plants) derived from a single ortet constitutes a clone. A desired number of ramets are grown in field plantings. The resulting plantation is subject to infestation by insects or to attack by pathogens, both of which we shall refer to as pests. No pests are present in the base population. Genes that control susceptibility to pests are assumed to segregate independently from those that influence the traits for which the clones are chosen.

To simplify computations, we use a single locus two allele (*A* and *a*) model in which allele *a* is recessive for susceptibility to pest attack. Whether the stand remains economically viable at time of harvest depends on three random quantities: the number, X, of clones of susceptible genotype *aa* among Q clones chosen for planting, the yearly sequence D_1, D_2, D_3, ... of severities of pest attack on the stand, and the responses to attack of the individual ramets in the stand (for simplicity, each remains economically viable, or not). If p is the frequency of the susceptible allele *a*, then X will be a binomial random variable with parameters Q and p^2. To further reduce computational difficulties, we assume the D's are independent and take only two possible values: 1, if a pest attack occurs and 0, otherwise, with respective probabilities λ and $1 - \lambda$. Thus, if harvest is anticipated after T growing seasons, and if attacks can occur but once per season, the number Y of attacks also has a binomial distribution, with parameters T and λ. Finally, we assume response to pest attack depends only on the present, and not on the cumulative history of attack, with probability v_1 that a ramet of genotype *aa* remains viable following an attack, and probability v_2 that a ramet of genotype *Aa* or *AA* successfully weathers an attack.

Following LIBBY (1982), ROBERDS *et al.* (1990), and BISHIR & ROBERDS (1995), we introduce a number β, $0 < \beta < 1$, such that a plantation is considered economically viable if, at harvest time, the proportion of originally planted ramets that have died or have suffered pest damage beyond a commercially acceptable level is below β. Beta thus is analogous to a maximal acceptable proportion of loss (MAL – see LIBBY 1982). If S denotes the proportion of ramets that remain viable, failure of the stand occurs when $S \leq 1-\beta$. Then the risk, or probability of failure associated with the plantation, is given by

$$R = P(S \leq 1 - \beta) \qquad [1]$$

Ideally, the desired number of clones is the smallest number for which the risk falls below a maximum level α acceptable to the plantation manager. Unfortunately, it is sometimes impossible to reach such a level, no matter how many clones are chosen. Thus, we adopt the approach of HÜHN (1986) and choose the smallest number of clones that produces a risk R either (a) smaller than α, or (b) within $\delta = 0.02$ of the risk-value associated with use of an infinite number of clones. We shall denote this number of clones as Q^*, and refer to it as the number *required* in order to meet the criteria indicated above.

The quantities α and β, along with Q^*, p, v_1, v_2, and the product λT, are the parameters involved in our computations. [Since T is usually fairly large and λ is not close to 1.0, the binomial distribution for Y can be approximated closely by a Poisson distribution with a single parameter equal to the product λT.]

Table 1 contains some typical results regarding the required number of clones. Outcomes are shown for two of the more than 400 combinations of parameter values we investigated. The body of the table lists

required numbers of clones corresponding to genotypic survival probabilities $v_1 = 0.1$, $v_2 = 0.95$, and a desired maximum risk level $\alpha = 0.05$. In part (A) of the table, the acceptable damage level $\beta = 0.33$, while $\beta = 0.67$ in part (B). In each portion there are six rows, corresponding to values of the product λT. As we move across a particular row the value of the gene frequency p increases and a prototype pattern emerges. The required number of clones, Q^*, equals 1 for very small p-values; then there is an increase in Q^*, first gradual, then steep, until a point is reached at which Q^* abruptly drops back to 1.0 and remains at that level for all larger p-values. Tabular values of 500 indicate only that the required number of clones exceeds 250, the largest finite Q^* value considered in the computations. In part (B) of the table, values in the second and third rows exhibit two rise-and-fall sequences instead of one. Our numerical investigations suggest this is an artifact of the simplified model we used, rather than a feature of more realistic models. We emphasize again that the Q^* values in the table are presented only to illustrate the trends we observed. They may or may not approximate the actual numbers of clones appropriate to a real plantation or those based on criteria deemed important in government regulation.

In Table 2, it is v_2 that changes, v_1 and β being fixed throughout this table. Here, the increase from $v_2 = .75$ in part (A) to $v_2 = .98$ in part (B) elicits the same kinds of changes produced in Table 1 by increase in β.

A PICTORIAL VIEW

A useful way to view the overall implications of our model is to plot risk R as a function of p, the frequency of allele a. Curves resulting from such plots are presented in ROBERDS & BISHIR (1997), for a variety of harvest times T and numbers of clones Q. Figure 1 illustrates the pattern typically observed. All parameters except p and Q – viz., α, β, λ, T, v_1 , and v_2 – are held fixed. Two curves are shown, one corresponding to $Q = 1$, the other to $Q =$ infinity. Both curves rise as the frequency of allele a increases. This will always be the case since an increase in p raises the probability of susceptible aa genotypes among the chosen clones, causing the likelihood of plantation failure to increase. (We assume $v_1 < v_2$.) The curves coincide at $p = 0$ and $p = 1$ as in those cases all clones will have the same probability, v_1 at $p = 0$ and v_2 at $p = 1$, of successfully withstanding a pest attack.

Figure 1 contains the most common pattern obtained in our computations, in which the curve for $Q =$ 1 initially rises more rapidly, but is overtaken at a point

Table 1. Required numbers of clones, Q^*, corresponding to different values of p, the frequency of susceptible allele a, and λT, the expected number of pest attacks over the lifetime of the plantation. In part (A), the maximum acceptable proportion of trees lost before harvest is $\beta = 0.33$, while $\beta = 0.67$ in part (B). In both parts, the probabilities of ramet survival following a pest attack are $v_1 = 0.1$ and $v_2 = 0.95$. A tabular value of 500 indicates that more than 250 clones are needed to achieve the α and/or δ criteria described in the text.

A)

p / λT	0.0	.05	.10	.15	.20	.25	.30	.35	.40	.45	.50	.55	.60	.65	.70	.75	.80	.85	.90	.95	1.0
0.5	1	1	1	1	1	1	1	1	10	20	100	500	1	1	1	1	1	1	1	1	1
1	1	1	1	1	1	1	5	10	20	80	500	1	1	1	1	1	1	1	1	1	1
2	1	1	1	1	1	10	20	40	150	500	1	1	1	1	1	1	1	1	1	1	1
5	1	1	1	1	500	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
10	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
20	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1

B)

p / λT	0.0	.05	.10	.15	.20	.25	.30	.35	.40	.45	.50	.55	.60	.65	.70	.75	.80	.85	.90	.95	1.0
0.5	1	1	1	1	1	1	1	1	5	5	5	5	5	5	10	20	30	500	1	1	1
1	1	1	1	1	1	1	5	5	5	5	5	5	5	10	20	60	5	500	1	1	1
2	1	1	1	1	1	5	5	5	5	5	5	5	10	20	40	500	1	5	1	1	1
5	1	1	1	1	1	5	5	5	5	5	10	20	30	60	500	500	1	1	1	1	1
10	1	1	1	1	1	5	5	10	10	20	40	100	80	1	1	1	1	1	1	1	1
20	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1

Table 2. Required numbers of clones, Q^*, corresponding to different values of p, the frequency of susceptible allele a, and λT, the expected number of pest attacks over the lifetime of the plantation. In part (A), the probability a resistant genotype survives a pest attack is $v_2 = 0.75$, while $v_2 = 0.98$ in part (B). In both parts, the maximum acceptable proportion of trees lost before harvest is $\beta = 0.5$, and the probability a susceptible genotype survives a pest attack is $v_1 = 0.1$. A tabular value of 500 indicates that more than 250 clones are needed to achieve the α and/or δ criteria described in the text.

A)

λT \ p	0	0.05	0.1	0.15	0.2	0.25	0.3	0.35	0.4	0.45	0.5	0.55	0.6	0.65	0.7	0.75	0.8	0.85	0.9	0.95	1.0
0.5	1	1	1	1	1	1	1	1	1	1	1	80	500	1	1	1	1	1	1	1	1
1	1	1	1	1	10	40	250	1	1	1	1	1	1	1	1	1	1	1	1	1	1
2	1	1	1	1	10	80	500	1	1	1	1	1	1	1	1	1	1	1	1	1	1
5	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
10	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1
20	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1	1

B)

λT \ p	0	0.05	0.1	0.15	0.2	0.25	0.3	0.35	0.4	0.45	0.5	0.55	0.6	0.65	0.7	0.75	0.8	0.85	0.9	0.95	1.0
0.5	1	1	1	1	1	1	1	1	5	5	5	10	20	30	50	1	1	1	1	1	1
1	1	1	1	1	1	1	5	5	5	5	10	20	30	80	30	1	1	1	1	1	1
2	1	1	1	1	1	5	5	5	5	10	20	20	40	250	1	1	1	1	1	1	1
5	1	1	1	1	1	5	5	5	5	10	20	30	125	500	1	1	1	1	1	1	1
10	1	1	1	1	1	5	5	10	10	20	40	250	500	1	1	1	1	1	1	1	1
20	1	1	1	1	1	10	30	60	100	1	1	1	1	1	1	1	1	1	1	1	1

p = c, after which the curve for Q = infinity remains higher. Points a and b are the values of p at which the respective curves achieve a height of α, the hoped-for bound on the probability of plantation failure. In the figure, $a < b < c$. Though less common, other patterns do occur. The crossing point $p = c$ can come before the curves reach level α, in which case the order of the points is $c < b < a$. Occasionally, values of the fixed parameters are such that the curves start above α, in which case we assign $a = b = 0$. And some parameter combinations produce more than one crossing point. In these cases, however, the curves do not separate widely between crossings (in our computations, never more than 0.05 in the vertical direction) and a single crossing is, for practical purposes, the generic outcome.

We also assume that curves corresponding to $Q = 2$, 3, ..., lie between the two curves shown. This is sometimes not the case for small numbers of clones, as fluctuations typically occur due to the discrete nature of the probability distributions of x and y. For practical purposes, however, these too can be ignored.

With these disclaimers, we can determine required numbers of clones associated with broad ranges of p, as indicated below the horizontal axis in Figure 1. We always choose the smallest number of clones that enables us to meet the desired criteria. For instance, only one clone is required when p lies between 0 and a,

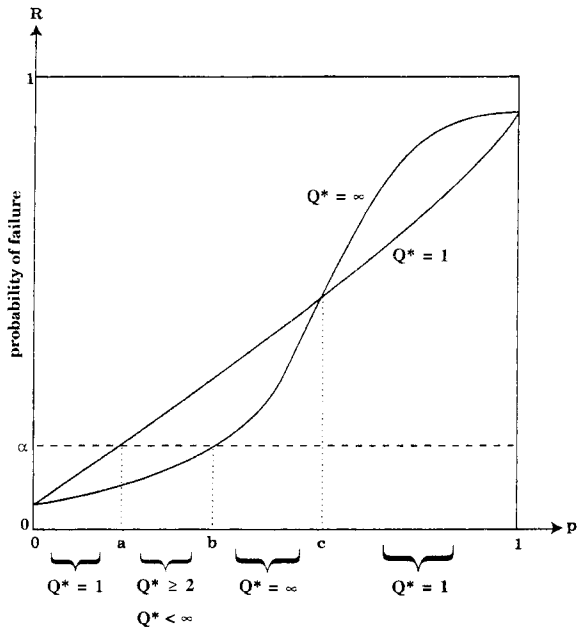

Figure 1. Typical risk curves corresponding to use of a single clone ($Q = 1$) or use of an 'infinite' number ($Q = \infty$). In the figure, the risk R of plantation failure – equation (1) – is plotted as a function of p, the frequency of susceptible allele a. Minimum numbers of clones needed to meet the risk criteria imposed in the text are indicated by Q^*.

as here the curve for $Q = 1$ lies below α, thus falling within the desired bound. When the value of p is reater than b, the desired α-level cannot be attained. Nevertheless, only one clone is required when p exceeds c, for then the failure curve corresponding to $Q = 1$ is the lowest of all the curves. In contrast, between b and c the curve corresponding to Q = infinity is lowest. The interval from b to c is usually narrow, the curve for Q = infinity being quite steep there (see ROBERDS & BISHIR 1997). The most complex portion is the interval from a to b, in which level α can be achieved with a finite number of clones but the required Q^* is larger than 1, and further computations must be made to determine it precisely. Only in the interval from a to c is the assumption underlying the European Community mandates, that using more clones will provide greater safety, consistent with our results.

ANALYSIS BASED ON PROBABILITY THEORY

Our overall numerical results, only a small portion of which appear in Tables 1 and 2, indicate that increase in λ or T, and thus in λT, tends to decrease the needed number of clones, while increase in any of v_1, v_2, and β produces opposite effects. What is the common thread in these results? To help answer this, we first consider another question – How can a smaller number of clones ever be better than a larger number? Coin tossing provides a simple analogy in a setting free of the complications associated with our clone model.

Suppose we can toss a fair coin any number, N, of times we choose, and win a prize if the proportion of heads is less than 0.7. How many tosses should we make? The answer is "As many as possible." The proportion of heads is approximately normally distributed, with an expected value of 0.5 and variance equal to $1/4N$. Thus the density is centered at ½, and narrows as N grows larger. The probability of obtaining a proportion of heads smaller than 0.7 approaches 1.0 as N increases.

Now suppose the rules change so we win the prize only if the proportion of heads obtained is less than 0.3. Tossing more times increases the likelihood of a proportion near 0.5 and thus lessens our chance of winning. In fact, the largest probability of winning is ½, obtained by tossing only once.

To relate this scenario to the clonal setting, substitute ramets for coin tosses. The target value, 0.7 or 0.3, represents β, the threshold value used to determine stand failure, while the expected proportion of heads, ½, is replaced by $E(S)$, the expected proportion of ramets having no value at harvest – see the discussion leading to Equation (1). If the expected result is a successful stand, that is, if $E(S) < \beta$, then the more clones the better. This would tend to be the case when the probabilities of ramet survival in the face of pest attack, v_1 and v_2, are high, when β, the threshold value used to indicate stand failure, is large, or when λT, the expected number of pest attacks, is small. On the other hand if v_1, v_2, or β is small, or λ or T is large, so λT is large, then it is more likely that $E(S) \geq \beta$. In this case, the expected result is failure and, as in the coin toss, we want to increase the variance among possible outcomes so as to increase our chance of being away from the mean. We do this by choosing a smaller number of clones.

Of course, the simple coin tossing model is not completely analogous to our more complex setting. We choose clones, rather than ramets. Even with an infinite number of clones we cannot reduce the variance to zero because of the variation associated with the number of pest attacks. And the parameter α in our model further clouds the picture. Still, while these features soften the 'one or many' dichotomy seen in coin tossing, the broad conclusions are similar and all are consistent with the simulation results described above.

LIBBY (1982) expresses these relationships in terms connected to the clonal process, using Maximum Acceptable Loss (MAL), equivalent to our β, and Risk to a Random Genotype (RRG), similar to our $E(S)$. As a rule of thumb, Libby suggests we should use a large number of clones if RRG < MAL , while a small number, perhaps only one, is best when MAL < RRG.

RISK-PRONE AND RISK-AVERSE STRATEGIES: AN ANALOGY FROM ECOLOGY

Libby's rule is closely related to concepts that form the basis of 'risk-prone' and 'risk-averse' strategies used in ecological theory (MANGEL & CLARK 1988; REAL & CARACO 1986; STEPHENS & KREBS 1986). When a 'safe' strategy – one with a small variance – is likely to result in failure (that is, when the expected result is failure as, e.g., when the coin toss target was 0.3), it is better to use a 'risky' strategy – one with large variance; for us, this means fewer clones – that has at least some chance of success. On the other hand, when success is likely it is better to choose a risk-averse, or 'safe', strategy; in the clonal forestry context, this means using a large number of clones.

From our model computations, Libby's rule appears to be accurate only when there is no 'environmental' variation (no variation in number of pest attacks). The more general risk-prone, risk-averse paradigm, however, is consistent with all our numerical results and, in particular, with the trends noted in our computations above. A plantation is more likely to fail if survival

probabilities (v_1 or v_2) decrease, if the expected number of pest attacks, λT, increases, or if β decreases. The theory then advocates a more risky strategy associated with use of a smaller number of clones. The paradigm is also consistent with the abrupt change from a large number of clones to the choice of only one clone that we noted in Tables 1 and 2 as allele frequency p increases across a row. A similar change occurs in the coin tossing example as the target proportion decreases through the value 0.5.

Of particular interest from these results is the prediction that the number of clones chosen should decrease as harvest time, T, increases, as this runs counter to conventional wisdom (*e.g.*, LINDGREN 1993; KLEINSCHMIT *et al.* 1993) which holds that as T increases, the plantation is potentially exposed to a larger array of pests and, therefore, the greater genetic diversity inherent in using more clones offers higher risk protection. Since our model does not take multiple pests into account, the question of how much these trends tend to counter each other is still open and further study is needed.

DISCUSSION

The question of the number of clones needed in reforestation is important both commercially and environmentally. The results of our investigations suggest that with regard to risk considerations the answer is many-faceted. Even with an extremely simple underlying model, the complex variety of particular cases we simulated led to 'required' numbers ranging from 1 to over 100 clones. In general, however, as suggested on theoretical grounds by BISHIR & ROBERDS (1997), situations requiring more than 40 clones are not prevalent. In the extensive computations we performed, only 4 percent of the required numbers of clones fell in this range. While situations requiring many clones do occur, in our model they are associated with fairly narrow ranges of parameter values. In practice, it is unlikely that parameter values could be determined accurately enough to know whether we are in such a region. It appears, then, that legal mandates requiring large numbers of clones only occasionally have the intended effect of reducing risk. Since some of our results indicate that such a requirement can actually increase risk, we feel the issue of how to formulate legal restrictions deserves careful review.

While the details of these results are varied, all the general patterns observed are consistent with the risk-prone, risk-averse paradigm cited in the preceding section. As noted there, this theory advocates a risk-prone strategy, one with large variance, when chance of failure is high, while a risk-averse response, one having small variance, is appropriate when failure is unlikely. In the context of risk analysis in clonal forestry, this implies use of many clones when this will assure a low risk of failure, but only a few clones when failure is the 'expected' outcome. Since the probability of plantation failure rises when either λ or T increases, and drops if v_1, v_2, or β increases, the predictions of this theory are thus consistent with each of the trends described in our computational results.

A seeming inconsistency in Tables 1 and 2 is that even though increase in allele frequency p leads to increase in probability of failure (and thus, presumably, to use of fewer clones) the 'desired' number of clones is listed as 1 when p is small. However, this is an artifact of our criteria for choosing the required number of clones. When v_1 is small, as it was in the two tables, the α-criterion (choose the smallest number of clones that makes the probability of failure less than alpha) comes into play when p is close to zero, and the Hühn-criterion (choose the smallest number of clones that produces a risk R either (a) smaller than α, or (b) within 0.02 of the R-value associated with use of an infinite number of clones) applies until p reaches the point beyond which the risky choice of a single clone is best, at least in our model. Without these requirements, an infinite number of clones would be best when p is small. Further, the multi-pest threat noted at the end of the preceding section suggests that, on balance, the best number of clones probably is never extremely small. These considerations, together with the theoretical conclusion of BISHIR & ROBERDS (1997) that using a very large numbers of clones rarely offers much more protection against risk than a moderate number, have led in British Columbia, Canada, to regulations suggesting use of 10 to 30 clones, depending on circumstances and goals (Alvin Yanchuk, personal communication). Further refinements in laws and regulations can be expected as we gain practical and theoretical experience.

One of the corollary benefits expected in some clonal forestry ventures is a reduction in harvest age. As growth rate is enhanced through breeding and management intensification, desired tree sizes are reached at younger ages and a reduction in harvest age is possible. An expected concomitant reduction in numbers of clones needed to manage risks, however, may not be realized if the highly intensive management practices employed create conditions that are conducive to increased frequency of attacks (increase in λ) by a particular pest, or to susceptibility to a wider variety of pests. Our analysis demonstrates that it is the product λT that must be evaluated when assessing the effects of changes in λ or T on required numbers of clones needed in risk management. Effects associated with reductions

in T might be offset by increases in λ that result from more intensive management practices.

Our discussion has been presented in terms of a simple genetic mechanism for resistence to pest attack in which viability of individuals following an attack is controlled by a single genetic locus having two alleles, the allele associated with high viability expressing dominance over the allele for low viability. While the results described here pertain to this special genetic model, they also reflect behavior that results from a more complex system of inheritance. In some species, individual tree viability following attack by certain pests may be regulated by multiple loci, with each locus possessing two alleles, one conferring pest resistance, the other susceptibility. If such loci assort independently, and only individuals that are homozygous for the susceptible alleles at every locus suffer reduced viability, the effect on individual viabilities, and thus plantation survival rates, is no different than that observed with our single-locus dominance inheritance model. In the context of the epistatic model just described, the parameter p in our analysis represents the frequency of the haplotype that causes high susceptibility. Thus, the single locus dominance model is a particular form of the more general epistatic susceptibility model and as a result our analysis and results are perhaps more broadly applicable than is at first apparent.

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NOTE

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