POPULATION SIZE AND DESIGN OF BREEDING PROGRAMMES

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SUMMARY

The choice of population size for animal breeding programmes depends on cost and management factors and on predictions of response to selection. In the short term these predictions can be based on parameters such as variances, covariances and effects of inbreeding which are estimable in the base population, but in the longer term less accessible measures are required, such as the joint distribution of effects and frequencies of both initially present and new mutant genes influencing the trait.

With dominance, inbreeding effects increase linearly with t/N, where t is generation number and N is effective population size. With the additive model of infinitessimal gene effects, variance and response decline at first in proportion to $(t/N)^2 V_{AO}$ and increase through mutation in proportion to $t^2 V_m$, where V_{AO} and V_m are the initial genetic variance and new variance per generation from mutation, respectively. Eventually response from mutation increases in proportion to NV_m. The larger the effect of genes on the trait, the earlier mutation influences mean and variance of response.

Population size effects over the range N = 10 to 160 are generally trivial for 5 generations, but can become substantial after 10 generations. The breeder's time horizon has a major influence on choice of population size.

INTRODUCTION

When designing breeding programmes we are considering how present actions are likely to affect future selection responses, performance and economic returns from the programme. In order to reach optimal solutions we need to know the relationships between changes in the input and output variables, only some of which are readily accessible. For example, adequate estimates of variance and heritability of major production traits can often be obtained, so it is possible to predict the relationship between selection intensity and response or the extra benefits of incorporating family information in an index for a few generations of selection, while genetic parameters change little. Any limitations in accuracies of the predictions can, in principle, be overcome by taking more of the same kinds of measurements, for example covariances among sibs or other relatives. Such predictions of short term responses to selection of quantitative genetics rely on the usual multivariate normal models and are not particularly sensitive to the underlying genetic determination of the traits.

In contrast, in discussing the role of population size in design of breeding programmes we have to deal with relatively long term aspects and adequate predictions can not be based solely on measures such as covariances among relatives. There are some simple exceptions, such as the relation between population size and selection intensity for fixed proportion selected, and, with a few more assumptions, the relation between performance and inbreeding level for traits not under selection. But when we consider the

relation between population size and long term response, we need information on distributions of gene effects and frequencies which are not readily accessible, nor likely to be. Therefore we are faced with making models of quantitative traits, and assessing their relevance using experimental and such other data as are available. It follows that any recommendations and decisions about choice of population size in breeding programmes are open to debate. It is not my aim here to provide specific solutions, but to review the bases on which they should be made.

SHORT TERM INFLUENCES - MEAN RESPONSE

Structures

There are important non-genetic influences to be mentioned first. The cost of any breeding programme rises in relation to its size and these costs are encountered from the outset regardless of the long-term benefits from increase in long-term response. Although fixed overheads lead to economies of scale, there are also diseconomies, notably in effective management. Many successful breeding programmes originated from the ideas, industry and control of a single person - such benefits are likely to be lost as the populations expand.

The relation between population size and selection intensity ($\bar{i}$) for fixed proportion selected can be computed from tables of order statistics. The further reduction in $\bar{i}$ as a consequence of family structure is usually forgotten, however, but the relevant results have been reviewed recently (Hill, 1985a). Overall the effects are unlikely to be important unless selection is made within contemporary groups which are small and comprise only a few families, such as typically occurs in pig performance testing programmes with a closed nucleus.

The size of the nucleus population may also influence the number of generations of multiplication from nucleus to commercial stock or the extent of selection possible during the multiplication. With a large nucleus the genetic lag between the best and what is marketed is reduced (Bichard, 1971), thereby affecting the level rather than rate of improvement.

Inbreeding depression

With the usual dominance model of inbreeding depression, there is a linear relationship between inbreeding coefficient (F) and performance in unselected populations; and, because $F = t/(2N)$ approximately for small t/N, where t is generation number and N is effective population size, inbreeding depression increases initially in proportion to t/N. If D is the inbreeding depression per unit inbreeding for some trait, the reduction in its performance is FD or $tD/2N$, approximately.

Providing gene effects are not large and the time scale is short, the joint action of inbreeding and selection can be computed as the sum of the changes from each and assumed to be linear in generation number. The response in a single trait from selection with accuracy r over t generations is $R_t = tirh\sigma$, approximately. The net response is then, following similar analyses by Nordskog and Hardiman (1980):

$$R_t(net) = t[irh\sigma - D/(2N)] = tirh\sigma[1 - (D/\sigma)(Ni)^{-1}(2rh)^{-1}] .$$

This result can also be obtained by summing effects over loci, providing each has sufficiently small selective value that gene frequency changes are small. The effects of population size and selection are seen to enter through the product term Ni, usual in such models of their joint effects (Robertson, 1960). The formulae can easily be extended to the multi-trait case. With selection on an index with weights b, the vector of responses is $b'G/\sigma_I$, where σ_I is the standard deviation of the index and G is the genetic covariance matrix. If the effects of inbreeding on each trait are given by the vector D the net economic response is

$$R_t(\text{net}) = ti[b'G/\sigma_I - D'/(2Ni)]a \quad .$$

where a is the vector of economic weights. The optimum index, $b = P^{-1}Ga$, is not affected by the degree of inbreeding depression on any trait, whether or not it is included in the index, because the model is linear.

These results are in terms of a non-overlapping generation case. When generations overlap, inbreeding increases initially at a non-linear rate (Johnson, 1977) and a precise statement of net response must account for both the cumulative selection (Hill, 1974) and the inbreeding of specific cohorts. In steady state, the increase in inbreeding per year equals $1/(2NL)$, where L is the generation interval, and the response per year is proportional to i/L, so the foregoing equations apply without change to responses per generation or, if divided by L, to responses per year.

It is easiest to get some feel for these results by considering individual traits, taking two extreme examples in terms of extent of inbreeding depression. For example, for litter size at weaning in pigs the total effect, due to inbreeding of the litter and the dam is about 0.57 pigs per 10% inbreeding (Hill and Webb, 1982). Thus D = 5.7 and with σ= 2.3, D/σ= 2.5. For mass selection on single litter size, r = h/2, and with h^2 = 0.1, $R_t(\text{net}) = 0.115ti[1 - 25/(Ni)]$. It is clear that, with selection intensities typically between 1 and 2, inbreeding effects will be negligible only if N greatly exceeds 20. At the other extreme, taking a figure of 0.20%/1% (Pirchner, 1985) for the effects of inbreeding on fat depth, mass selection on both sexes (r = h), a heritability of 0.4 and coefficient of variation of 10%, then D/σ= 2 and $R_t(\text{net}) = 0.4ti\sigma[1-2.5/(Ni)]$.

For traits of reproduction the inbreeding effects are also countered in part by natural selection. As this does not act on any single trait, but on fitness as a whole, the effects of natural selection on each trait can not be predicted with any certainty. It is clear, however, that if the selection programme is designed such that there is little variation in family size, e.g. by testing a fixed number per litter, the balancing effects of natural selection are substantially reduced.

When the commercial product is a breed or line cross, these inbreeding considerations matter only at the level of the parental or grandparental pure lines, where reduced reproductive rate may increase costs and decrease selection intensity.

Effects of changes in variance

With increasing generations, the expected variance and thus expected response changes as a consequence of drift. The extent of this depends on the mode of gene action: with additivity and no selection, the additive genetic

variance, V_A, declines as $V_{A0}(1-1/2N)^t = V_{A0}(1-F)$; with dominance it may increase if recessive genes are initially at low frequency (Robertson, 1952). As a reference point it is useful to consider the model of additive gene action with genes having sufficiently small effects that changes in gene frequency as a result of selection can be ignored (Robertson, 1960) - the so-called "infinitessimal model" (Bulmer, 1980). The initial variance then declines in proportion to 1-1/2N and thus, if F is small, in proportion to t/N and the response declines accordingly. The cumulative response is a sum of terms proportional to 1, 1-1/(2N), 1-2/(2N), ..., approximately, to give

$$R_t = (iV_{A0}/\sigma)[t-t(t-1)/(4N)],$$

plus higher order terms. Because the variance also declines as a consequence of negative linkage disequilibrium caused by selection (Bulmer, 1980), the total reduction in the initial variance is likely to be greater than this, but the change due to small population size rather less.

These calculations ignore any input of variation from mutation which, though likely to be small initially, accumulates (Clayton and Robertson, 1955; Frankham, 1980; Lynch and Hill, 1986). Let V_m be the increment in additive genetic variance each generation from mutation, again assuming the genes are of infinitessimally small effect. After t generations, the total variance is, from Clayton and Robertson (1955),

$$V_{At} = [(1-1/2N)^t]V_{A0} + 2N[1-(1-1/2N)^t]V_m$$
$$= 2NV_m + (1-1/2N)^t(V_{A0} - 2NV_m).$$

In early generations, the cumulative response is thus approximately

$$R_t = i\{tV_{A0} + [t(t-1)/2][V_m - V_{A0}/(2N)]\}/\sigma.$$

Information on input of variance from mutation is limited to laboratory animals and maize. Estimates of V_m for bristle number in Drosophila are centred around $0.001\sigma^2$, i.e. a new heritability of 0.1%, while M. Lynch (unpublished) finds that values of V_m for other species are larger, perhaps as high as $0.0001\sigma^2$ x the generation interval in days. Thus for Drosophila bristle number, assuming a heritability of 0.25, the new variance approximately equals the loss per generation of original variability in a population of effective size 125 and for species with longer generation interval the critical population size may be much smaller.

Although a useful reference point, the infinitessimal model can be no better than an ever poorer approximation with increasing generation number: it requires that values of Ns, where s is selective value, are less than 1 or so. The contribution to heritability of an additive locus with frequency q and difference a between the homozygotes in effect on the trait is $q(1-q)a^2/(2\sigma^2) < a^2/(8\sigma^2)$, so the total heritability is $h^2 < nE(a^2/8\sigma^2)$, for n loci. Thus, for $Ns = Nia/\sigma < 1$ with a typical value of $Ni = 100$, it is necessary that $a/\sigma < 0.01$ at all loci, so if genes have equal effect and $h^2 = 0.25$ the infinitessimal model requires $n > 20000$. This seems an unlikely scenario, so we consider more general models subsequently.

SHORT TERM INFLUENCES - VARIATION IN RESPONSE

The effects of finite population on variation in response to selection

have been discussed extensively (e.g. Hill, 1980; Nicholas, 1980). Two sources of variation in response can be identified: the first applies to both selected and unselected populations and derives from the effect of genetic sampling on mean performance; the second applies only to selected populations and derives from the effect of sampling on genetic variance and thus on subsequent response.

For additive genes the drift in mean in unselected populations is simply $2FV_{A\emptyset}$, from Wright's theory, and is approximately $tV_{A\emptyset}/N$ if t/N is small. For non-additive genes the drift in mean depends on the distribution of frequencies and degrees of dominance (Robertson, 1952), but for small t/N the variance is approximately $tV_{A\emptyset}/N$ for any dominant or epistatic model. This can be simply illustrated by expressing the mean as a Taylor series expansion in gene frequencies, and noting that the first derivatives of mean with respect to gene frequency are twice the average effects of the genes (Kojima, 1959). The relation between drift in gene frequency and in mean follows immediately.

These results apply to unselected populations and, without knowledge of the distribution of gene effects and frequency, we have little choice but to use them as approximations in the case of selection. For the infinitessimal model of all genes of very small effect and with additive gene action, a more precise analysis is possible. As a consequence of selection, the variation is reduced to $[1 - i(i-x)h^2/2]V_{A\emptyset}$ after one generation, and not by a great deal more subsequently if loci are unlinked (Bulmer, 1980), and the subsequent drift variance of the mean is reduced accordingly, i.e. by $i(i-x)h^2V_{A\emptyset}/(2N)$. The variation in variance, however, rapidly builds up and, if most pairs of loci are unlinked, asymptotes very quickly at $\mathrm{Var}(V_A) \doteq 2V_A^2/(3N)$ (Avery and Hill, 1979, correcting their 1977 result) and the consequent variance in response is $2i^2h^2V_A/3N$. Although rather larger, this term is of the same order but opposite sign to the reduction in variance in mean from selection, so the effects partly cancel. This suggests, and simulations of Robertson (1980) support the view, that the simple formulae for drift in unselected populations hold, at least for short periods, more generally. The obvious exception is where there are some genes of very large effect, although analysis of models (see the next section) with distributions of effects and frequencies have, however, shown the formulae to apply reasonably well for at least 5 generations.

The foregoing analysis has dealt solely with the case of drift variance; to this has to be added a term due to error of estimation of genetic mean from phenotypic mean, which does not accumulate over generations. This term depends on maternal environmental and within-family variances (Hill, 1980).

These results on variance of response have been reviewed because they play some role in design of breeding programmes, although more importantly in the design of selection experiments and of control populations to be used to monitor response in improvement programmes. Two relevant aspects are: firstly, the issue raised by Nicholas (1980) of whether a programme would be "successful", i.e. whether the response would exceed some specified amount, typically, the relevant quantity is the ratio $SE(R_t)/E(R_t)$; secondly, there is the possibility of selecting between lines, utilizing the variability among them. As discussed by Madalena and Hill (1972), for a short time the best subpopulation is likely to be superior to the mean performance of a single large population, but in the long term there are not benefits from this subdivision, in that subdivision and crossing leads to the same limit (for

additive genes) if no between-line selection is practised, and to a lower limit if some sublines are rejected.

LONG TERM INFLUENCES

As Robertson (1960) pointed out, selection limits are a function of the product Nir, where r is accuracy and long term responses a function of this product and time expressed as t/N. For given numbers recorded, he showed that for mass selection Ni was maximised when one-half the population is selected, and increases in accuracy by incorporating family information might also lead to a reduction in long term response through a reduction in effective population size. There is therefore an obvious conflict between short and long term responses to selection, which is exacerbated when the effects of mutation are included for then the long term rate of response is approximately proportional to Nir.

We have attempted recently to define multi-locus models in terms of the joint distribution of gene effects and frequency rather than by specifying any particular set of values of either (Hill and Rasbash, 1986). The analysis has, so far, been restricted to two alleles and additive gene action without linkage. The last of these assumptions is not critical for species with many chromosomes, either for response from either initial variation (Robertson, 1970a) assuming linkage equilibrium or from new mutations (Keightley and Hill, 1983). The distribution of gene effects of previously segregating and mutant genes has been taken as a gamma distribution, which enables a wide range of possible "shapes", from equality of gene effects to very extreme leptokurtic form. Three examples of gene frequency distribution have been used to describe the existing variation: all frequencies 0.5, uniform over range 0-1, and inversely proportional to q(1-q), i.e. "U-shaped", such as derives from a neutral mutation finite population model.

The analysis shows that, for these examples where the mean gene frequency is one-half, the distribution of initial frequencies or gene effects, given the mean gene effect, does not have a marked influence on the mean or variance of response, except that (i) in the U-shaped case there are always further small increases in the limit and its variance for increases in population size, no matter how large it is, because there are so many initially rare, but favourable genes, and (ii) with initial frequencies of 0.5 the variance of response may decline after several generations as all favourable genes reach fixation.

The joint effects on response of variation initially present and deriving from mutation are illustrated in Figures 1 and 2, using results modified from Hill and Rasbash (in preparation) for specified population sizes and an initial uniform distribution. The critical parameters are the ratio of variation from mutation per generation and initially present, V_m/V_{A0}, the increment in mean response or its variance being proportional to this quantity, and the mean selective value, scaled as E(s) where E(s) = E(ia/σ) is the mean selective value of favourable genes. These results show that neither population size nor mutation nor size of gene effect has significant influence for at least 5 generations on either the mean or the variance of response. By 10 generations, the population size effects are beginning to be noticed, but those of mutation are only significant for the case of very large gene effects. Subsequently the pattern becomes more complicated: for the case of very small gene effects (E(s) = 0.0125) population size has a marked influence on the response from initial variation, the limits approaching $2NR_1$, as

pointed out by Robertson (1960); but mutation has rather less influence for the 50 generation period shown, basically because the mutants have low initial frequency and selective value and take a long time to produce response. Ultimately the rate of response from the mutants is not dependent on E(s). The variance of response is proportional to the mean response when E(s) is low, again as described by Robertson (1960), regardless of the source of the variation. When gene effects are larger, population size has less influence on response from initial variation, and a very large influence on that from mutation. Further, when variation derives from a few genes of large effect by mutation, it is clearly very much a hit-or-miss process as to whether the relevant mutants appear and get fixed. Thus the variation in response can be enormous, that from mutation swamping that from initial variation within very few generations, if population size is large. (Variances for N = 160 and E(s) = 0.2 are very large, but are not shown because the numerical analysis was not sufficiently accurate).

In summary, we see from these examples that population size has an important role: if gene effects are small on average, then it influences the response from initial variation, if the gene effects are large then it increases the response from mutation after 20 or fewer generations. The longer the time horizon, the more important is population size and mutation in these models.

Among the more important assumptions in this analysis is that mutation effects are symmetric for the trait in question, i.e. that favourable and unfavourable mutants are equally likely, and the distribution does not change with time. Asymmetry of effects is readily taken account of, in that the long term response is a function not of V_m but of the variance contributed by mutants of favourable effect. Let $E(a^2)$ be the mean squared effect of all mutants and $E^+(a^2)$ the mean squared effect of those for which a is positive. Then we replace V_m by $2V_m E^+(a^2)/E(a^2)$, which, with symmetry, reduces simply to ^mV_m. (see Hill, 1982 for details). A second assumption is that of additivity of gene action: put simply, the results apply with some modification of constants provided the mutants have an effect in the heterozygote, and if they are recessive, the mutants are unlikely to contribute to response. A third assumption is that of mass selection; with family selection there is, relative to predictions solely in terms of accuracy of selection, some disadvantage in weighting family means positively, basically because the mutant originally appears only in one member of the family (Hill, 1985b).

In a major respect these analyses are simplistic in that no account is taken of the opposing forces of natural selection. If this acts in a classical stabilising selection fashion, in that fitness declines as a quadratic function of the deviation of the population mean from its norm, natural selection effects will become stronger until a plateau is reached (James, 1962; Z.-B. Zeng and Hill, in preparation). If the stabilizing selection is sufficiently strong, this plateau will not depend at all on population size, particularly if variation is renewed by mutation. Alternatively, if fitness differences are expressed through the direct effect of the genes affecting the trait under selection on reproduction and other aspects contributing to fitness, selection response will still depend on population size, and the rate of occurrence and fixation of new mutants which are favoured by the "index" of desired and fitness related traits will still be proportional to population size.

DISCUSSION

A critical factor in any discussion of population size is the time horizon of the breeder: if he is concerned with surviving for only a few years it is clear that he should select as intensely as possible and not be concerned either with maintenance of variation or inbreeding depression. As this horizon increases, so does the ideal population size. How is this to be rationalized?

One approach, proposed by James (1970), is to discount expected returns from improvement in later years. Assuming knowledge of the cost of the breeding programme per individual scored and the size of the market, and using the infinitessimal model of decay of variation, he was able to compute optimal size of the programme. With large animals having a four or so year generation interval and an annual discount rate of 5%, for example, the discount rate per generation is approximately 20%. At that level the breeder is unlikely to be concerned much beyond 6 generations or 24 years and there is little case for maintaining large populations. With poultry or pigs having annual generations, the case for maintaining large populations is stronger. The problem is that such an argument used for large animals ignores any social responsibility to one's descendents. An alternative approach, used by Robertson (1970b) was to compute the optimal series of population sizes and selection intensities to maximise response to a fixed time, a relevant criterion in a selection experiment, less obviously so in a breeding programme.

These analyses ignored the role of new mutation, where although population size has no effect on rates of response in early generation, in the long term the rate is proportional to size, albeit after making some simplistic assumptions. Some of these have been mentioned, but we also need to know how variation is maintained in current populations and to what extent mutation/population size or mutation/natural selection balances are relevant. Certainly one can take an optimistic view, illustrated by the results of the Illinois corn experiment: keep selecting and the population will keep responding (Dudley, 1977). Many experiments with laboratory animals have shown limits, or at least perceived limits, but many have been run with rather small population sizes. It is difficult to disentangle the response due to initial and new mutational variance in analysis of selection experiments, but in those long term experiments on mice reviewed by Eisen (1980), there was a linear rather than diminishing returns relationship between total advance and population size. Providing breeders are prepared to assume a long time horizon they need to consider whether new variation is to come from: mutation in highly selected, competitive, populations offer more hope than "germ plasm resources" which are simply unselected populations. But perhaps, looking only a few years hence, we will obtain new variation by molecular tricks of one form or another, either by transposable element mutagenesis (Mackay, 1984) or insertion of genes from other populations or species.

ACKNOWLEDGEMENTS

I am grateful to Jonathan Rasbash for a great deal of assistance and to the Agricultural and Food Research Council for financial support.

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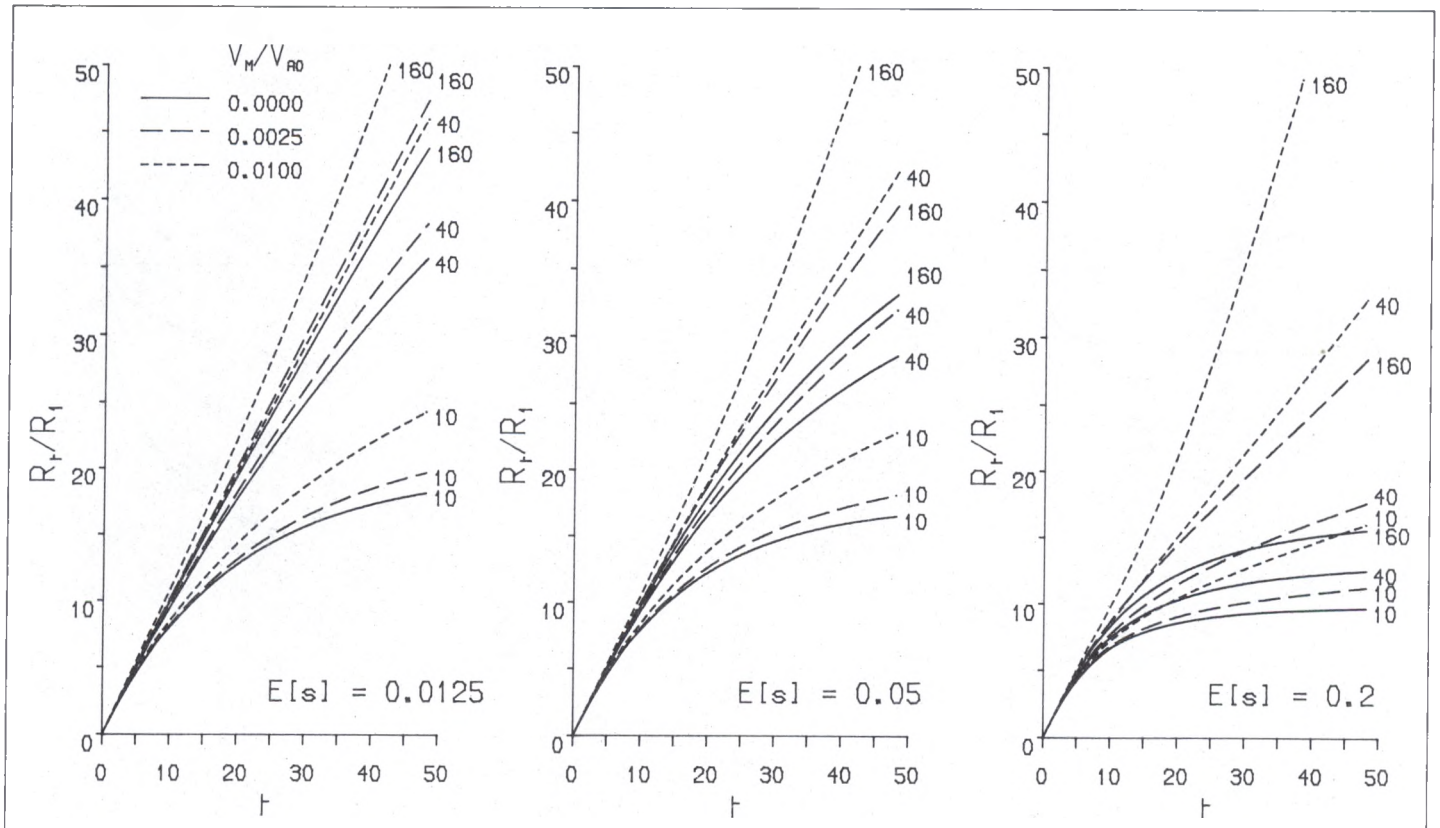

FIG 1.
Ratio of expected response at generation t to that from generation 1 for different values of N.

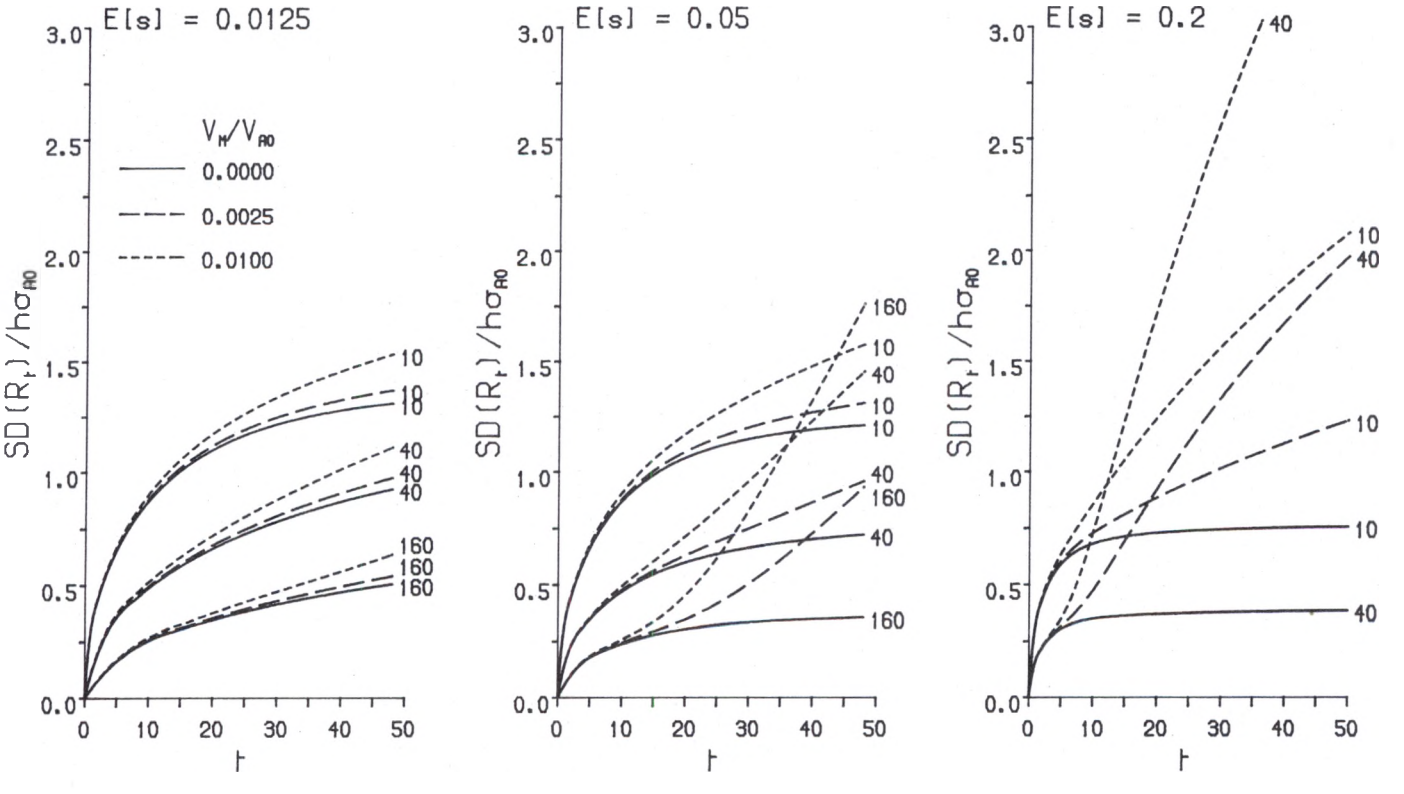

FIG 2.
Ratio of standard deviation of expected response at generation t to initial genetic standard deviation for different values of N.