POPULATION BOTTLENECKS AND NONEQUILIBRIUM MODELS IN POPULATION GENETICS. II. NUMBER OF ALLELES IN A SMALL POPULATION THAT WAS FORMED BY A RECENT BOTTLENECK

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Manuscript received April 22, 1985
Accepted June 7, 1985

ABSTRACT

A model is presented in which a large population in mutation/drift equilibrium undergoes a severe restriction in size and subsequently remains at the small size. The rate of loss of genetic variability has been studied. Allelic loss occurs more rapidly than loss of genic heterozygosity. Rare alleles are lost especially rapidly. The result is a transient deficiency in the total number of alleles observed in samples taken from the reduced population when compared with the number expected in a sample from a steady-state population having the same observed heterozygosity. Alternatively, the population can be considered to possess excess gene diversity if the number of alleles is used as the statistical estimator of mutation rate. The deficit in allele number arises principally from a lack of those alleles that are expected to appear only once or twice in the sample. The magnitude of the allelic deficiency is less, however, than the excess that an earlier study predicted to follow a rapid population expansion. This suggests that populations that have undergone a single bottleneck event, followed by rapid population growth, should have an apparent excess number of alleles, given the observed level of genic heterozygosity and provided that the bottleneck has not occurred very recently. Conversely, such populations will be deficient for observed heterozygosity if allele number is used as the sufficient statistic for the estimation of $4N_fv$. Populations that have undergone very recent restrictions in size should show the opposite tendencies.

THE fate of genes in a population that experiences a sudden reduction in size has become an increasingly important topic in population genetics, molecular evolution and conservation biology. When populations become small, they lose genetic variability, which may make the population susceptible to extinction because of lack of adaptive flexibility (BEARDMORE 1983). The effects of different conservation management strategies on genetic variation in populations of threatened animals and plants are just beginning to be worked out (SCHONEWALD-COX *et al.* 1983; HOLDEN and WILLIAMS 1984). Severe population restrictions also provide theoretical questions of direct interest to the evolutionary geneticist. Population bottlenecks may be the cause of some dis-

crepancies that have been observed between theory and observations on the patterns of protein polymorphism in natural populations (NEI, 1980).

NEI, MARUYAMA and CHAKRABORTY (1975) pioneered the theoretical study of bottleneck effects. They modeled an extreme bottleneck in a population that was in equilibrium between mutation and random genetic drift and then measured the fraction of the population's genetic variability that was lost following the bottleneck. Although explicit formulas could be derived to study the gene diversity (genic heterozygosity) of the population, formulas for allele number and distribution were not readily obtained. Some studies of allele number were accomplished by computer simulation; those indicated that, following an extreme bottleneck, most segregating alleles can be lost, particularly if those alleles were at low frequency before the bottleneck.

In two preceding papers (MARUYAMA and FUERST 1983, 1984), we developed methods to study a population that had, in a single step, greatly increased its size from a small number of founders. This theory enabled us to determine the transient behavior of the number of alleles as this number increased in response to the increased population size. In this paper, we consider the converse situation: the population is reduced in one step from a large size to a small one. We have adapted our previous numerical methods and have utilized those developed by WATTERSON (1984) to estimate the change in the number of alleles following a sudden reduction in population size.

Our studies focus on a small time period, of the order of N or $2N$ generations, where N may be 50–100, or even less; therefore, we are discussing very short real time, especially when compared with the time scale considered in our earlier papers. This comparison is necessary because, as we detailed in the first paper of this series (MARUYAMA and FUERST 1984), we are attempting to approximate the complicated processes that accompany a population bottleneck by studying models that constitute different portions of the overall cycle of population size crash-flush. Our first paper considered changes in allele numbers in a population that has attained a large size following initial monomorphism. Those changes take long periods of time and involve the interaction of mutation and drift. In this paper, we are interested in the early phases of the bottleneck cycle, the time immediately following a crash in population size. Allelic changes caused by a reduction in population size occur rapidly because they are mediated only by the sampling effects of small population size and are not dependent on mutational input.

The results presented here are particularly relevant to situations encountered in conservation biology. The captive breeding of an endangered species of animals is frequently initiated using a small number of founder individuals. Species management often dictates that the population size will never grow very large, because of economic constraints and limitations of available space in breeding facilities. Management of these small populations is likely to be necessary for a limited number (perhaps tens) of geneerations, or maybe for longer. The conservation of germplasm in various economically important crop species of plants also involves the breeding of small numbers of individuals of each of the different strains being conserved, again for an indeterminate num-

ber of future generations. Our methods, and the results presented below, should be useful for predicting the patterns of allelic loss for these situations.

THE MODEL OF POPULATION RESTRICTION

The first paper of this series (MARUYAMA and FUERST 1984) presented a list of various models that can be used to obtain analytically various statistics describing the changes in allele number attributable to population size changes or bottlenecks. In this paper, we present results from the model of the initial portion of the cycle of population change. This cycle is completed by the model of population expansion given in the earlier paper.

Consider a locus with a large number of existing states. Assume that each mutant at the locus is new, every existing allele is subject to mutation at a constant rate, v, each generation and all extant alleles are selectively neutral. Further assume that, although finite, the population size is large enough to be approximated by a diffusion process. In the model, the population undergoes a sudden decrease in size and remains small following the change. The relevant time period of the model is short, and as a consequence, the fate of alleles can be determined almost entirely by following those alleles that exist in the population before the bottleneck, with mutational input of new alleles being effectively negligible. It should be noted that, even though mutation is a negligible force during the processes that are being studied, both our methods and those of WATTERSON (1984), which we use, explicitly include mutational input.

NUMBER OF ALLELES FOLLOWING THE BOTTLENECK

The fate of genetic variability measured by the level of genic heterozygosity can be easily calculated for this model. The change in the number of alleles in nonequilibrium populations is more difficult to calculate; only recently was the problem analytically solved by GRIFFITHS (1979 a, b; 1980) and by WATTERSON (1984). NEI and LI (1976) studied the change in the frequency spectrum. Griffiths' formulas are expressed as series expansions by orthogonal polynomials on the Dirichlet distribution. Although the analyses using Griffiths' formulas are exact, calculation of allele numbers can be fairly complicated. It involves handling terms in large factorials, especially when the sample size is large. Watterson's method provides simpler analytical formulas, which can be used to calculate allele number. In MARUYAMA and FUERST (1984), we also developed a numerical method that can be used for calculating the number of alleles and the age of these alleles in a sample of genes taken from an evolving population. The method is based on numerical integration of the Kolmogorov backward equation, expressed in the form of a difference equation. Essentially this same method can be used to compute the number of alleles in a small finite population derived from a larger steady-state population. The details of the derivations will not be presented, because no new theoretical concepts are involved; the reader is directed to our earlier papers.

We have compared the results obtained using the analytical method of WATTERSON (1984) and using the numerical integration method of MARUYAMA and

TABLE 1

Comparison of the numerical methods adapted from MARUYAMA *and* FUERST *(1984) with the method of* WATTERSON *(1984) for the determination of the total number of alleles and the numbers in two rare allele classes*

	$4N_0v = 0.1$			$4N_0v = 1$			$4N_0v = 10$		
Time	Total	Single	Double	Total	Single	Double	Total	Single	Double
0.025									
(MP)	1.24	0.090	0.059	2.69	0.789	0.478	6.04	3.727	1.433
(W)	1.25	0.092	0.059	2.78	0.789	0.481	6.18	3.814	1.452
0.05									
(MP)	1.22	0.078	0.056	2.54	0.652	0.441	5.31	2.826	1.534
(W)	1.23	0.079	0.056	2.57	0.660	0.445	5.44	2.892	1.581
0.10									
(MP)	1.20	0.062	0.048	2.29	0.476	0.365	4.29	1.786	1.087
(W)	1.21	0.063	0.049	2.32	0.484	0.371	4.41	1.828	1.115
0.25									
(MP)	1.14	0.041	0.035	1.85	0.248	0.216	2.80	0.684	0.539
(W)	1.15	0.041	0.035	1.87	0.253	0.223	2.89	0.704	0.563
0.50									
(MP)	1.08	0.031	0.027	1.48	0.123	0.111	1.86	0.252	0.223
(W)	1.10	0.028	0.023	1.49	0.125	0.116	1.94	0.264	0.239

(MP) = method of Maruyama and Fuerst; (W) = method of Watterson. Sample size $M = 10$; $4N_0v$ = value before population reduction; $4N_rv = 0.01$ for all cases. Time is measured in units of $2N$ generations, where N is the population size following the bottleneck.

FUERST (1984) for a wide range of parameters, and we find the results to be in close agreement (see Table 1). The number of alleles that appear in a sample of genes will be a function of the mutation rate (v), the original population size (N_0), the reduced population size (N_r), the sample size (M), and the time since the reduction (t). If N_rv is much smaller than one, new mutations are essentially negligible for the purposes of the present paper. Hereafter, calculations assume a fixed value of $4N_rv = 0.01$, unless stated otherwise. Some examples of the comparisons between the two computational methods for the model of population restriction are given in Table 1 for the calculation of the total number of alleles and for the number of alleles present either once or twice in a sample. The calculations assume that the population starts with a defined value of $4N_0v$ and that population size is reduced to yield a $4N_rv$ value after the bottleneck of 0.01. It is clear that the numerical integration method developed in our earlier paper yields results comparable to the analytical method of WATTERSON (1984). In the studies to be presented below, we have used both methods of calculation.

The average number of alleles expected to appear in samples taken at various times following the reduction in population size was determined for different values of $4N_0v$ in the original equilibrium population. Some examples are presented in Figure 1. As is well known, the number of alleles declines rather rapidly after the population size reduction. The rate of decrease de-

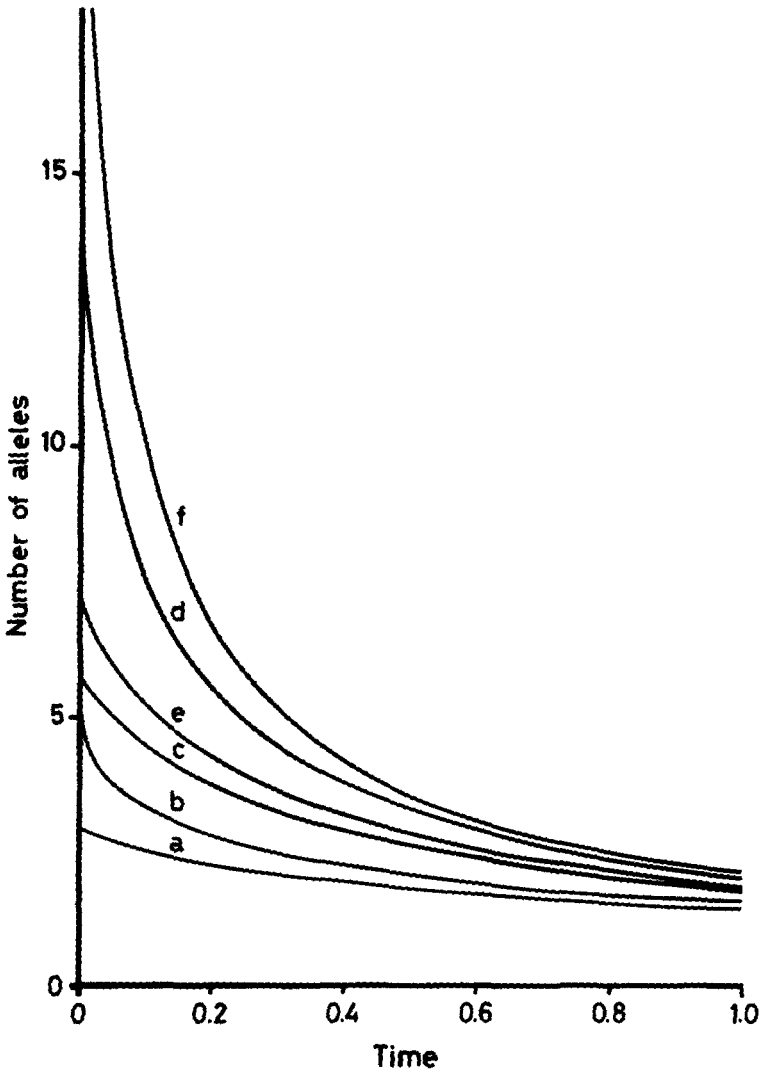

FIGURE 1.—Number of alleles to appear in a sample of genes taken from a bottlenecked population. Time in units of $2N$ generations. (a) $4N_e v = 1$, $M = 10$; (b) $4N_e v = 1$, $M = 100$; (c) $4N_e v = 5$, $M = 10$; (d) $4N_e v = 5$, $M = 100$; (e) $4N_e v = 10$, $M = 10$; (f) $4N_e v = 10$, $M = 100$.

pends on $4N_e v$; the decrease is more rapid for larger values of $4N_e v$. This decrease in allele number thus differs from the decrease in heterozygosity, which is always proportional to the reciprocal of the population size, $1/N_e$, but is independent of the initial level of variation. The loss of alleles due to random genetic drift is, in contrast to heterozygosity, proportional to the square of the number of existing alleles. More precisely, if n is the number of alleles, the rate of change from n to $n - 1$ is proportional to $n(n - 1)/2N$ (KIMURA 1955; FELSENSTEIN 1971; BURROWS and COCKERHAM 1974). This explains the results seen in Figure 1, where allelic loss is much more rapid from populations that

have larger initial $4N_o v$ values. Larger $4N_o v$ values imply greater allelic diversity before the bottleneck. Figure 1 indicates that, for many biologically meaningful cases, most alleles that were present in the original population will be lost in the first N generations. This means almost complete loss of allelic diversity, because newly arising mutations can essentially be ignored in the short time periods being considered after the reduction in population size.

Figure 1 also indicates that sample size has only a minor effect on the number of alleles observed in samples taken from the reduced population at various times following the bottleneck. This seems to be a consequence of changes in the allele frequency spectrum in the reduced population that is the source of the samples being taken. As the population goes from a large size to a small one, many alleles with low or moderate frequency are being lost (also shown in Figure 2). Small samples taken from the population following reduction will contain almost all of the allelic variation that remains in the population. Increasing the sample size will therefore increase the number of sampled alleles only slightly.

The methods developed in our earlier papers and those presented by WATTERSON (1984) also enable us to obtain details about allele number. These include such statistics as the number of alleles that are present only once in a sample or that each present twice, and so on. In our earlier papers we termed these classes singly or doubly present alleles. This terminology permits us to study the behavior of rare alleles. Statistics of rare alleles have often proved to be important in making population inferences about size and mutation rate and also in discriminating among rival evolutionary hypotheses (NEI 1977; NEEL and ROTHMAN 1978; OHTA 1976).

In a steady-state population, the number of singly present alleles is given by $4NvM/(4Nv + M - 1) = 4Nv/(1 + 4Nv/M - 1/M)$, adapted from WATTERSON (1974). For a sufficiently large M, this number is almost equal to the value of $4Nv$, a population parameter, and is thus almost independent of the sample size. Hence, for each fixed sample size, the number of singly present alleles in the equilibrium population increases almost linearly with $4Nv$.

The situation is drastically altered, however, when the population is suddenly reduced in size. As shown in Figure 2, the numbers of singly present alleles in a sample can be greatly different, depending on the sample size and the time that has elapsed since the population reduction. These results show that, following the bottleneck, the number of singly present alleles decreases more rapidly over time when comparisons involve large sample sizes, and the decrease is more pronounced when the reduced population is derived from a larger and more variable population.

Even though the number of rare alleles taken from a steady-state population increases with increasing sample size, the number of singly or doubly present alleles—our measure of rare alleles—in a large sample from a nonequilibrium reduced population can actually be much smaller than that observed in a small sample. This can be seen in Figure 2 by comparing graphs (c) and (d) or (e) and (f). The depression in the number of singly present alleles is most conspicuous immediately following the reduction in population size. This is a

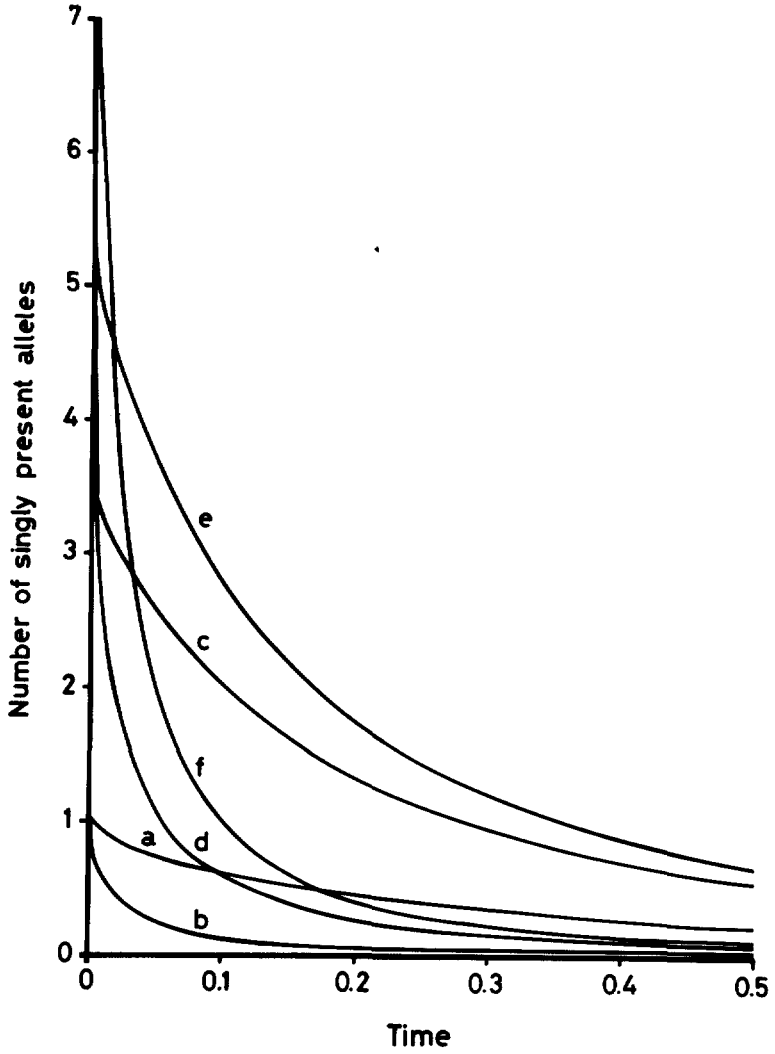

FIGURE 2.—Number of allele each represented only once in the sample. Parameters for (a) to (f) are the same as in Figure 1.

consequence of the rapid loss by drift of low-frequency alleles that existed in the prereduction population. Larger populations have more of these alleles to lose. Additionally, the lower numbers of singly present alleles seen in the larger postbottleneck samples, when compared to smaller samples, reflect the almost complete lack of low-frequency alleles in the postreduction population. An allele having frequency 0.1 in the population may easily appear as a singly present allele in a small sample (if it appears at all), but will probably appear in several copies in a larger sample. Because few alleles with low frequency exist in the population following reduction, it is less likely that the sample will contain alleles with low sample frequency. The behavior of rare alleles following the population reduction thus appears to be complicated. It is certainly

worth noting, however, that the number of rare alleles may actually decrease as the sample size becomes large, a situation that does not occur when the population size is steady or expanding.

TRANSIENT DEFICIENCY OF ALLELES

When a large steady-state population is reduced to a small size, it begins to lose the genetic variability that has been built up by mutation. Variability measured by the level of genic diversity (expected heterozygosity) will fall at a rate of $1/2N$ per generation; allelic diversity, however, declines at a much faster rate, as noted above. This can lead to difficulties when one studies natural populations. We may falsely assume that a population exists in a steady state with a small population size. If we compare the observed level of heterozygosity with the number of alleles, we will find that the observed number of alleles is lower than that predicted for an equilibrium population with a corresponding level of heterozygosity. In most situations where data on electrophoretic or nucleic acid variability has been gathered from natural populations, we do not know the history of the population and cannot determine whether the population under study is in a steady-state condition or not. In fact, it may be most appropriate to assume that natural populations are hardly ever in an equilibrium state. Because of this shortcoming in our knowledge of natural populations, it is extremely important to determine the theoretical relationship between the observed gene diversity and the observed number of alleles in small nonequilibrium populations.

As in our previous paper (MARUYAMA and FUERST 1984), we are interested in the difference between the expected number of alleles in a sample taken from a transitional population and the number expected in the sample based on the (false) assumption that we are sampling an equilibrium population. This difference interests us because many workers have compared the average number of alleles and an expected number based on the average heterozygosity for natural populations that have been studied using protein electrophoresis. The equilibrium number of alleles in our studies is generated by determining the equilibrium $4Nv$ value that corresponds to the heterozygosity observed in the transitional population. In the first paper of this series, we found that, when the population expands from a small initial size, this difference was positive, indicating an excess number of alleles. We also found that the excess disappeared fairly rapidly, usually by about $0.5N$ generations. Note, however, that N can be large in this case.

The difference between the expected and the observed total number of alleles found in a small population following a restriction in size is presented in Figure 3. Irrespective of the values of the parameters considered, when a population has contracted in size, there is a transient deficiency in the number of alleles present in the population as compared with that expected in an equilibrium population that has an equivalent heterozygosity. The magnitude of this deficiency depends on an interaction between the size of the sample and the value of $4N_ov$ just before the population reduction. The maximum deficiency that is observed for each case occurs very shortly after the popula-

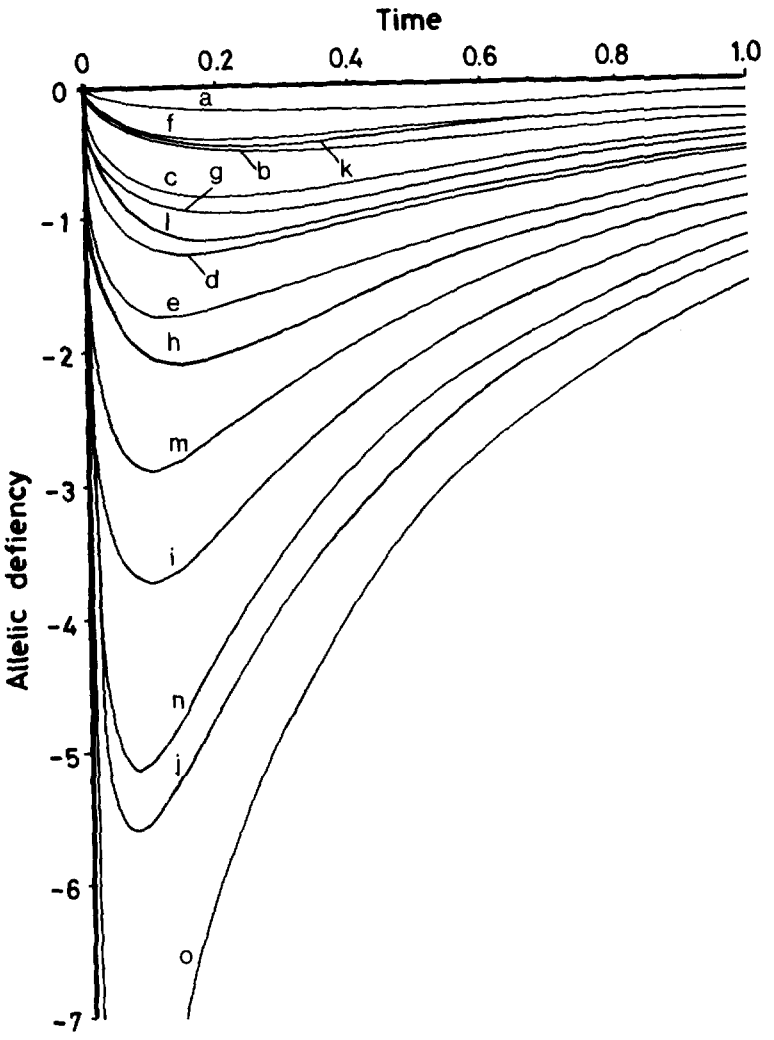

FIGURE 3.—Deficiency of alleles, based on the average number calculated by method of MARUYAMA and FUERST (1984) and on the (false) assumption that the population is in steady state at the observed level of genic diversity. $4N_e v = 1$ for (a) to (e); $4N_e v = 5$ for (f) to (j); $4N_e v = 10$ for (k) to (o). M = 10 for (a), (f) and (k); M = 20 for (b), (g) and (l); M = 50 for (e), (k) and (m); M = 100 for (d), (i) and (n); M = 200 for (e), (j) and (o).

tion restriction, and the deficiency diminishes as population gene diversity declines to ultimately catch up with lowered allele numbers.

There are only minor differences between different starting populations if sample sizes are small, even between populations that differ in $4N_e v$ by one order of magnitude; compare (a) *vs.* (k) in Figure 3. Larger samples, however, show a strong effect of the level of gene diversity present in the initial population. These differences can be attributed in great part to the rare alleles that are lost rapidly in small populations. When the original population has a large number of rare alleles, because it possessed a large $4N_e v$, the deficiency of

alleles will be very large, as seen for (o) in Figure 3. Here, there are many rare alleles lost in the first few generations, while the initial high heterozygosity is declining at a relatively slow pace. However, if the number of alleles is small in the original population, such as for (e) in Figure 3, the rates of loss for alleles and for gene diversity are approximately equal, and the deficiency is small even with larger samples.

A further aspect of allele loss is the relationship between the observed and expected (equilibrium) number of alleles in certain rare allele classes. Rare alleles have played a role in several attempts to discriminate between rival models of molecular evolution (OHTA 1976, NEI 1980). We have focused on those alleles that occur most infrequently in a sample, examining the number of alleles that are present only once in a sample of genes or that each present twice. Results for singly present alleles are shown in Figure 4. The expected allele number was again derived by assuming that the observed gene diversity was obtained from an equilibrium population. As with total allele number, singly present alleles show consistent deficiencies for all combinations of $4N_o v$ and M. The effects of sample size and initial level of variation are very similar to those described above for total alleles. Comparison with the results given in Figure 3 indicate that, particularly when the total deficiency is large, singly present alleles constitute the allelic class most sensitive to the population reduction. This one class of alleles is responsible for the major portion of the total allele deficiency. For instance, if we compare graph (n) in the Figures 3 and 4, we can see that singly present alleles make up approximately 4.1 of a total 5.1 allele deficit, when $M = 200$ and $4N_o v = 10$. Examining all the data, we can conclude that 80% or more of the total deficiency of alleles in a sample taken from a population soon after a bottleneck would be due to alleles that are expected to appear only once in the sample. Examination of our data indicates that, of the residual deficiency, about three-fourths, or another 15% of the total deficiency, can be attributed to the absence of alleles that would occur only twice in a sample. Consequently, a deficiency of rare alleles should be a characteristic of any population that has recently undergone a severe population reduction and has not had sufficient time to regain variation through growth and mutation.

An alternative way of looking at the data would express the relationship between allele numbers and gene diversity in terms of the expected gene diversity given a particular observed number of alleles, rather than the expected number of alleles given an observed heterozygosity. The former approach has been advocated by WATTERSON (1977). The data presented here would certainly suggest that there would be a relative excess in heterozygosity for a given observed number of alleles in a population that had recently experienced a restriction in size. The problem has been given more consideration in a recent study by WATTERSON (1985).

DISCUSSION

The analyses presented above and in the preceding paper of this series are important for both biological and theoretical reasons. The two models account

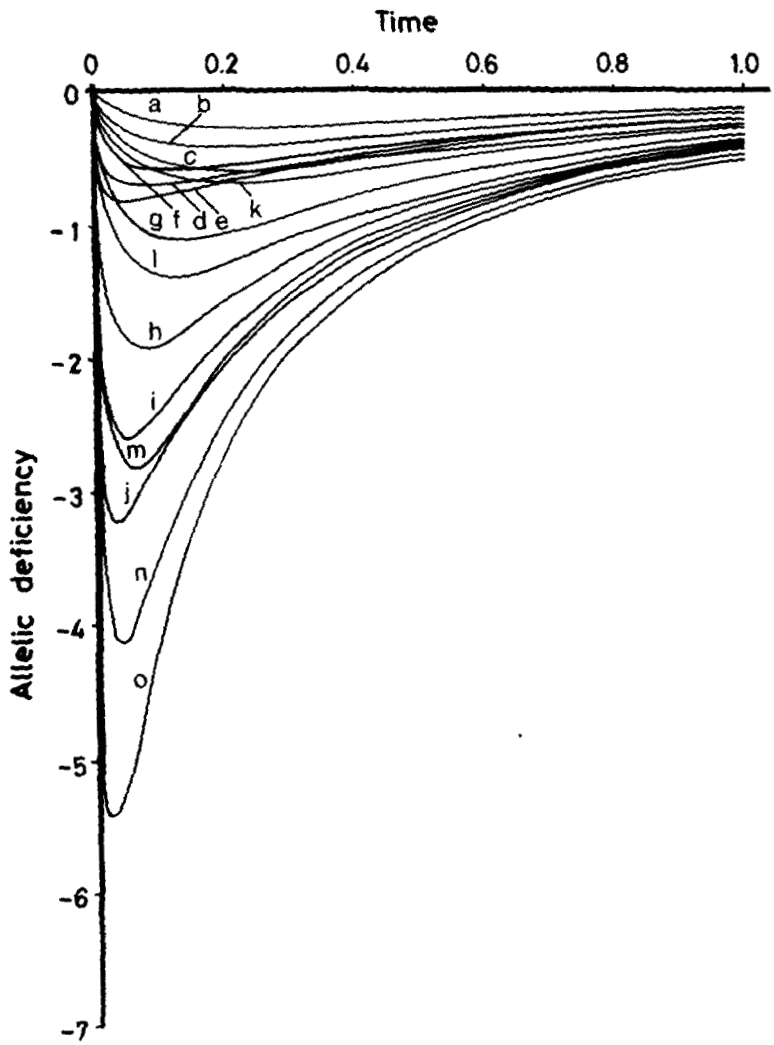

FIGURE 4.—Deficiency of alleles each represented only once in a sample, based on the two assumptions stated in Figure 3. Graphs (a) to (o) have the same parameter values assigned for (a) to (o) of Figure 3.

for the major stages of the size cycle of a population that experienced a bottleneck in population size and subsequently reestablished the original, or at least a large, size. Since we find that expansion and contraction of population size have different effects on allele number, comparison of the two papers allows us to weigh the relative importance of the different parts of a single population cycle.

In our earlier study we found that allele numbers showed significant excesses when the population rapidly expanded from a homoallelic state. In this paper we have examined the converse situation, population size contraction, and find allelic deficiencies. When the two parts of a complete population size cycle are

compared, the loss of alleles following population contraction, illustrated in Figures 1 and 2, seems to be relatively less important, in terms of absolute numbers of alleles in a sample of genes taken from a population, than is the increase in the number of alleles that follows the rapid expansion of population size studied in our earlier paper. Comparison of Figure 1 with the equivalent figure in MARUYAMA and FUERST (1984) shows that the time course for the decrease of alleles during population contraction will be much shorter than the time course taken by increases in allele number accompanying population expansion. Although the two processes of allele number change are antagonistic, the process of increase will probably be the more important. Unpublished results from computer simulations of a single bottleneck followed by gradual population expansion also indicate that an excess should occur, although it may not be as great as we suggested in our earlier paper.

In our analyses, we have emphasized some statistics that are often calculated for survey data on natural populations. Natural populations might not be in a steady state, but the calculation of these test statistics usually assumes a theoretical population at equilibrium between mutation and random drift, and having an average gene diversity equivalent to that seen in the natural population. To assess the validity of such tests, we have examined carefully how the transient situation affects total allele number. Our results suggest that the number of alleles obtained in a sample of the population will be significantly affected by changes in population size. To make inferential statements based on allele numbers, it is obvious that one needs information about the stability of the population or the stage of a size cycle at which the population has been sampled. For example, NEI (1980) has invoked population bottlenecks to explain increased numbers of alleles in some natural populations, while OHTA (1976) explained allele excess by invoking the presence of slightly deleterious alleles. The results presented in our two papers in this series certainly support Nei's contention that rare alleles will be relatively increased in a population that has recently gone through a bottleneck, but we cannot even conjecture, on the basis of our theoretical studies, how frequently population bottlenecks occur in nature.

The number of rare alleles in a sample has also been used for the estimation of mutation rate (more precisely $4N_e v$) in natural populations (NEI 1977; NEEL and ROTHMAN 1978). Strictly speaking, our results show that it is not possible to estimate the mutation parameter $4N_e v$, using either the gene diversity or the allele number, without knowledge of the time since changes in population size. If the population has undergone a very recent alteration in size, the mutation rate estimate may be inflated because of the initial loss of alleles following a severe contraction in size. Some deflation of the estimate of the mutation rate might occur if the population were in an expansion phase of population size; however, as we have shown here and in the preceding paper in this series, rare alleles approach their altered equilibrium more rapidly than do alleles of intermediate or high frequency. Therefore, if we are to use statistics of allele number to estimate $4N_e v$, rare alleles are likely to introduce the smallest bias. Again, information about the stability of the populations

being studied would seem to be necessary for adequate interpretation of any estimate.

Although allelic deficiencies in a small population may be relatively less important than allelic excesses in an expanding population, the results of this paper do have some implications for two biologically important situations: the short-term evolution of introduced populations, such as pest species of plants or insects, and the preservation of genetic diversity in small managed populations of endangered animals or plants. Our results indicate that an allele deficiency should be seen in populations that are sampled soon after the population bottleneck and before mutational input can overcome the initial loss of alleles to sampling. Just such situations are seen repeatedly when introduced pest populations that can be traced to a small number of founders are examined. The case of various introductions of the Mediterranean fruit fly (*Ceratitis capitata*) seems to fit the theory nicely (HUETTEL *et al.* 1980).

Due to environmental deterioration, there has been much recent concern about the biological conditions necessary for the preservation of species that are in danger of extinction (SOULE and WILCOX 1980; FRANKEL and SOULE 1981; SCHONEWALD-COX *et al.* 1983). Other workers are concerned about the preservation of potentially useful genotypes of domesticated plants that may be lost without special efforts being made to preserve them (HOLDEN and WILLIAMS 1984). Management strategies must include consideration of methods that preserve as much genetic diversity as possible. The loss of alleles predicted by Figures 1 and 2 would be most obvious and important if a population were maintained at a small size, for several generations, without being permitted to expand rapidly. This is just the situation that is likely to become increasingly prevalent for many large vertebrate species in danger of extinction because of habitat destruction. These species are coming under some type of conservation management, whether it be in zoos or in natural parks. The populations have been reduced, often rapidly, to small sizes, and the scarcity of available habitat makes it unlikely that the size of the population will rapidly increase in the foreseeable future.

The population geneticist must provide tools to the species manager that will be helpful when decisions concerning population structure and breeding patterns must be made. A previous attempt to define the rate of loss of alleles in such small contracted populations was made by DENNISTON (1978). He used a simple model of multinomial sampling to investigate the number of alleles remaining in a population of a small fixed size. GALE and LINDSEY (1984) summarized some results from branching processes on specific breeding models useful for the preservation of variation in small plant populations. Our models, and the methods of WATTERSON (1984), constitute more general approaches that can be used for populations of any size to predict the loss of variation and also the allelic classes that will be affected. We have recently adapted the methods used in this present paper to provide detailed information on the pattern of allelic loss during the very early stages (first 20 generations) of the captive management of small populations of endangered species (FUERST and MARUYAMA 1985). We hope that conservation biologists may be able to apply

these methods during the process of decision making that accompanies all attempts to preserve endangered genetic material.

Finally, we feel that the results presented here do not yet provide a definitive picture of the changes in allele number that occur as natural populations undergo changes in size. Further studies, especially with respect to the relative excess or deficiency of alleles and the dynamics of allele number change over time, are being conducted using models in which the population undergoes multiple cycles of change in population size, and in which population growth following a bottleneck is gradual, rather than instantaneous. Preliminary results from these models suggest that our conclusions concerning the overall patterns of allele number change may be altered as we analyze models that more closely approximate a biologically realistic situation.

We thank GEOFF WATTERSON for his assistance and discussion and for pointing out some of his earlier results. We also thank an anonymous reviewer for additional assistance in improving this paper. This study was supported by research grant BSR 8110220 from the United States National Science Foundation and by grant 57120001 from the Japanese Ministry of Education, Science and Culture.

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Communicating editor: M. NEI