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A Genetic Model of Dental Reduction Through the Probable Mutation Effect

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ABSTRACT A simulation approach is used in order to elucidate the nature of the hypothesized "probable mutation effect" as it applies to dental reduction in man. Computer-generated simulations of the accumulation of mutations in a human gene pool show the results of the proposed model under the influence of various parameters, as well as illustrating the nature of such genetic change through time. This approach supports a polygenic model of the probable mutation effect as a viable hypothesis for an explanation of the dental reduction which has occurred in some human populations over the last 40,000 years.

In the evolution of fully modern man from Paleolithic era populations, there has been a steady reduction in the size and morphological complexity of the dentition (Brace, 1967; Brace and Mahler, 1971; Brace and Nagai, 1982; Brose and Wolpoff, 1971; Dahlberg, 1963; Frayer, 1978; LeBlanc and Black, 1974). Rather than suggesting that the reduction of these structures was due to natural selection for smaller teeth, Brace (1967) and others (e.g., Brose and Wolpoff, 1971; Wolpoff, 1969, 1975) have argued that reduction resulted from a relaxation of the selective forces. Though the posterior dentition had been undergoing reduction for some time, natural selection had maintained larger anterior dentition in early human populations. Changes in diet and culture reduced the selective advantage of these teeth. Tools took the place of anterior teeth for many survival functions, while dietary adaptations (Osborne, 1967) and cooperative behavior further lessened the selective differences among dental variations. A reduction in size and complexity of all the teeth followed, possibly due to the probable effect of accumulating selectively neutral mutations in the gene pool.

The "probable mutation effect" was proposed by C. Loring Brace (1963) as a possible mechanism for microevolutionary structural reduction. Supposing that a feature of an organ is no longer of use to an organism, natural selection will not act to maintain that feature. If mutations of genes affecting this feature occur at a constant or stochastically regular rate, they may accumulate through time in the absence of selective pressure. This is basically what Sewall Wright (1931, 1964) termed "mutation pressure." Most mutations are deleterious to the effectiveness of the biochemical chain of events which operate in the ontogenetic development of a given structure, diminishing the capacity of the gene to perform its previous function. Thus as these recurrent mutations are allowed to accumulate in the gene pool, the phenotypic structure itself may eventually be reduced in individuals throughout the population.

Neutrality theory has produced predicted rates of accumulating neutral mutations from single genes (Kimura, 1970). Brues (1968), Holloway (1966), Prout (1964), and Williams (1978) argue that mutation pressure alone would be insufficient to account for the necessary accumulation of mutations in the gene pool. What remains to be seen is whether a polygenic model of the probable mutation effect can account for the observed reduction of a quantitative trait within a limited time frame. If no such genetic model can account for the observed phenotypic reduction, then similar selective forces must be imposed on the model for each of the populations which have experienced structural reduction of the anterior dentition. Here I propose a genetic model of the probable mu-

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tation effect and use mathematical simulations to explore its characteristics. Though the model is not a precise representation of actual evolutionary events, I believe that it can, in principle, account for the structural reduction of human anterior dentition in the absence of natural selection.

ASSUMPTIONS OF THE MODEL

Evolutionary changes of quantitative traits such as tooth size are difficult to model with any precision, especially due to the complex interactions of the genetic and environmental factors which influence individual phenotypes (see Chakraborty and Nei, 1982; Relethford and Lees, 1982). One must assume that the changes seen are primarily genetic in origin and are not solely attributable to environmental differences. There is much evidence to justify the assumption that human tooth size is under considerable genetic control. Though there is a wide range of heritability estimates in various populations (as one would expect), the heritability of tooth size is consistently high, with environmental and developmental components also being of importance (Alvesalo and Tigerstedt, 1974; Garn, et al., 1965; Kolakowski and Bailit, 1981; Townsend and Brown, 1978).

Of the many models possible, the model which has been chosen to simulate the probable mutation effect is both deterministic and continuous. Since births and deaths occur continuously and not at discrete intervals equal to the length of a generation, the continuous model is more appropriate. Changes in gene frequency can then be mathematically described by differential equations (Ewens, 1979: 48).

The deterministic processes of evolution listed by Wright (1955) are recurrent mutation and natural selection, and these are the phenomena of concern to us here. In small populations, however, there are stochastic processes such as genetic drift which can alter allele frequencies. The magnitude of the effect of these processes is inversely related to population size. Although stochastic processes would seem to be important in small Paleolithic populations, they are not appropriate for this model for several reasons. First of all, though we may be dealing with small populations in which genetic drift may have some effect, there is no way to know what the population size was or how fast it was growing. Thus any estimate of population size would add potential error and complexity to the simulation. Furthermore, it can be argued that population size has little or no effect on the phenomena with which we are dealing, i.e., recurrent mutations of selectively neutral alleles. Assuming that the mutant alleles are functional yet selectively equivalent to the wild-type allele, it can be mathematically shown that the rate of substitution of these mutant alleles is equal to the rate at which they arise by mutation, and is independent of the population size. The probability, U, of the ultimate fixation of a selectively neutral mutant is:

$$U = 1/2N$$

where N is the effective population size. The rate K of mutant allele substitutions is

$$K = 2NMU,$$

where M is the mutation rate. Substituting the value of U,

$$K = 2NM(1/2N)$$
$$= M$$

(Kimura 1979:276).

Thus I will assume that any possible effects of genetic drift and gene flow were negligible through time and I will use a deterministic model.

Evidence from the fossil record suggests that anterior dental reduction has occurred over the last 40,000 years (Brace, 1967: 812). This then is the amount of time simulated in order to observe the behavior of the probable mutation effect. An average generation length in this simulation is set at 18 years.

Natural mutation rates of known deleterious mutants in humans range from 2×10^{-4} to 1×10^{-6} per generation (Dobzhansky, et al., 1977: 68). A sample of mutation rates within this range, specifically rates from 2×10^{-4} to 2×10^{-5} at intervals of 2×10^{-5}, will be simulated and held constant through time. The reverse mutation rate, however, is a matter of much debate. Because mutations to the alleles which result in reduction are those most likely to occur, the reverse mutation rate should be significantly lower. Furthermore, as I will demonstrate below, the reverse mutation rate has very little effect within the time period with which we are dealing.

In order to illustrate the effects of these parameters, it is worthwhile to imagine the simplest case. As Brace (1964: 453) puts it, "If a gene can be postulated which specifi-

cally affects only one character, and the ecological or adaptive situation becomes altered, reducing or suspending the selective advantage conferred by the possession of the character involved, then the Probable Mutation Effect will occur when mutations at that particular locus arise." When dealing with a single gene locus, the change in the frequency of q per generation (G) is defined by:

$$dq = Mp - Vq,$$

(Wright, 1931)

where p is the frequency of the "normal" or "wild-type" allele, q is the frequency of the mutant allele(s) such that $q = 1 - p$, M is the mutation rate of p to q, and V is the reverse mutation rate. A differential equation representing continuous change through time is simply:

$$dq/dG = M(1 - q) - Vq$$

(Note: The units can be changed from generations to years by dividing the right side of the equation by the length of the generation, e.g., 18 years.) This system will reach equilibrium when

$$q = M/(V + M)$$

Calculations made of genetic changes in the absence of the pressures from natural selection with a mutation rate (M) of 1×10^{-6} and a reverse mutation rate (V) of 1×10^{-7}, q will reach a value of 0.022 after 40,000 years, while p remains relatively high at 0.978. It can easily be shown that the reverse mutation rate has very little effect at this point. With M and V being equal at 1×10^{-6}, the frequencies are effectively the same as above (with a difference of only 0.0002 after 40,000 years). The reason for this is simply that the frequency of the mutant alleles is not high enough to yield many reverse mutations. It is only when the frequency of the mutant forms gets to be much higher that the reverse mutation rate will become an important factor, resulting in the asymptotic slowdown of change in allele frequencies (Brues, 1968: 437). Consequently, a conservative estimate of V will be held constant at one tenth of M throughout these simulations.

Dental reduction cannot be accounted for within 40,000 years with only a single gene locus, as is evident from the above example (see also Williams, 1978); neither the necessary change in allele frequencies nor the characteristic variability can be accounted for without a polygenic system. Thus we must postulate a number of loci at which the probable mutations will result in dental reduction. The aforementioned genetic studies consistently suggest that tooth size is a polygenic trait. Potter et al. (1976) have suggested that four independent genetic factors could account for maxillary tooth dimensions, while the mandibular dimensions may be influenced by seven independent genetic factors, though the precise nature of this polygenic system is not known. For purposes of exploring the effects of differing numbers of loci, the simulations will involve five to 25 independent, autosomal loci.

Even though Holloway (1966:7) has suggested that the probable mutation effect must apply to the accumulation of recessive alleles, a close look at modes of genic action suggest that there are other possibilities. The mutant alleles may be "recessive" with respect to the presence of the structure, yet codominant with respect to tooth size. Even with a partial dominance effect of the wild-type allele over the mutant allele, the result of size reduction can still occur, though to a lesser degree. In this model I will assume that the mutant alleles are "leaky genes," or hypomorphs, which contribute to less efficient production of phenotypic tooth size with no dominance effect. It is important to note that the mutant alleles are only neutral in the sense of selection among alternative alleles; they are not necessarily neutral in the sense of being nonfunctional genes. There will be an infinite number of possible allelic states, and the assumption is made that all allelic effects are additive with no dominance and no epistasis. This corresponds to the "discrete allelic-state model" (Chakraborty and Nei, 1982:305).

None of the mutations in this polygenic system may have pleiotropic effects which would significantly alter relative fitness because this could subject the gene frequencies to selective pressures. The gene could, for example, have pleiotropic effects on other teeth or facial form. There is also likely to be a distribution of effect of the mutant allelic forms, with some causing more reduction in tooth size than others. Consequently, there are a variety of possibilities for how many mutant alleles would be required in an individual to cause significant phenotypic reduction. To demonstrate the effects of these

variations, the probability of genetically caused reduction (R) was calculated according to a number of mutant alleles (n) potentially necessary for such reduction. For example, with n = 2, only those individuals having two or more loci with mutant forms will be counted as experiencing reduction in tooth size. Assuming random mating, with "N" being the number of loci involved, the probability (R) of reduction in tooth size for an individual is calculated by:

$$R = 1 - Q_0 - Q_1 \cdots - Q_{n-1},$$

where Q_n is the probability of carrying n mutant alleles over N loci on either chromosome, as calculated by:

$$Q_n = \frac{2N!}{n!\,(2N-n)!} \cdot q^n \cdot p^{2N-n}$$

On the other end of the distribution, mutant forms can arise which would truly be deleterious in terms of natural selection. Because these alleles could arise from either the wild-type or the mutant alleles, they will be left out of this model. With natural selection potentially at work, it is necessary to clarify that the probable mutation effect refers only to the accumulation of mutant alleles that are selectively neutral with respect to the wild-type alleles; selective forces are not totally eliminated in a global sense, but have been relaxed and are negligible in the maintenance of these alternative allelic forms.

The initial values for p and q are a final matter of importance in this model. There is no reason to believe that they must be started at 1.0 and 0.0 respectively, especially when we are dealing with recurrent mutations at many loci. A number of mutant alleles could accumulate which would not have much effect on fitness within a population due to their low frequencies. Thus a "mutational load" can be modeled in order to find an approximate starting frequency. In order to model a conservative mutational load, I will assume that natural selection acts against an accumulation of only two or more mutant alleles before the time period when selective forces were relaxed. The selection coefficient(s) for each mutant allele is negatively related to the probability of two or more accumulated mutants over all loci (i.e., s = −R), such that:

$$dq/dG = M(1-q) - V_q + sq(1-q)/(1-s(1-q)$$

(Wright, 1931).

For example, if the probability of having two or more mutant alleles (R) is 0.01, the selection coefficient for each mutant allele is −0.01.

SIMULATION TECHNIQUES

A series of models were set up on a computer using the Continuous System Modeling Program (CSMP), a simulation package designed for continuous, deterministic models. A data set was constructed from the output of the program including the values of p and q, as well as the probabilities of an individual having a given number of mutant alleles (Q_n) among a specific number of loci (N). These probabilities may be interpreted as relative frequencies of the population with the respective genetic makeup. The first 20,000 years of simulated time represents a time period when natural selection is working against the mutants, allowing sufficient time for the establishment of a stable mutational load. The selection coefficient is then set equal to zero for the next 40,000 years of simulated time. The accuracy of the results generated by the simulation program were verified by sample comparisons with calculations of mathematical solutions done on a hand calculator. Further calculations and analyses were performed using the Statistical Analysis System.

RESULTS

Listings of the simulated results are found in Table 1. The most conservative parameters simulated, with only five potential loci and a mutation rate of 2×10^{-5}, result in a predicted phenotypic reduction in a maximum of 41% of the population, depending on how many mutant alleles were required for reduction. Furthermore, 59% of the population carried no mutations at any of the five loci. Without an estimate of the amount of reduction in size resulting from the mutant allelic forms, it is impossible to translate these figures into a prediction of the actual percentage of change in tooth size through time. Though these results would predict some reduction in mean tooth size, they may not account for the amount of reduction seen in modern populations, where reduced dentition is more widely distributed throughout the population.

Results of simulations under the least conservative parameters, with 25 loci and a mutation rate of 2×10^{-4}, show virtually everybody in the population carrying mutant alleles. In fact, the most common individual (11.7% of this population) would carry 18 mu-

tant alleles over all relevant loci, and nobody would carry fewer than three mutant alleles. This demonstrates that even with the required number of mutant alleles (n) being relatively high, the probable mutation effect of phenotypic structural reduction would still be seen throughout most of the population. With the value of q at each locus being 0.355, many of these mutant alleles would be in the homozygous state. This could well be more genetic cause than is required for the phenotypic reduction seen today in tooth size, but as illustrated in Table 2, there is a wide range of parameters which approach this magnitude of reduction throughout the population after a simulated 40,000-year time span of relaxed selection. Table 2 summarizes the range of parameters simulated here that would result in 90% or more of the population's having genetically caused reduction in tooth size.

Each series of simulations demonstrates some basic principles. In summary it can be said that with high mutation rates, less loci need to be postulated in order to account for the probable mutation effect within a 40,000-year time period. Likewise, with a greater number of loci at which mutations affecting a structure are allowed to accumulate, there is a greater probability of carrying mutant alleles and consequently a greater probability of phenotypic structural reduction. Further variation, however, is imposed by the number of mutant alleles necessary to cause such reduction. This parameter has a more profound influence when the number of potential loci is lower, but as mentioned above, can be extended higher with similar results when the number of potential loci is greater.

The behavior of this system through time is illustrated in Figure 1a and b. Note that with increased mutation rates, the mutational loads are also greater and are reached more rapidly. With more loci, there would be a smaller mutational load at each locus, but a higher probability of phenotypic reduction throughout the population. In all cases, the phenotypic expression of the genetic load is a very small portion of the eventual reduction, but even under strong selection there is some genetic variability maintained by mutation pressure.

DISCUSSION

The accumulation of selectively neutral mutations at loci affecting a structural trait is predicted by this model of population genetics, as postulated by Brace. The significance of these results is that the probable mutation effect of structural reduction can manifest itself in a population within a limited time frame. This is in direct contradiction to the findings of Williams (1978). Williams uses a single locus model, assuming that such a model would maximize structural reduction because each locus of a polygenic system would only have a small effect in reducing the phenotype. It is, however, small reductions which have taken place, and the simulation shows that this can occur more quickly with a greater number of loci. The greater number of loci would also result in a greater distribution of reduced phenotypes throughout a population. As a consequence, no one gene has to reach fixation, as assumed by Williams, and actually can stay at fairly low frequencies.

A consideration of population size and structure is of great importance from the perspective of population genetics. Genetic drift occurring in small populations has been excluded from the model for mathematical reasons, but there are also instructive heuristic reasons. It is known that the probability and time required for a selectively neutral mutant to reach fixation are inversely proportionate to the population size (Kimura, 1970). Thus with many small human populations instead of one infinitely large population, it is likely that mutant alleles at the loci of concern would reach fixation in some of these populations. Once an allele is fixed in a population, the only way it can be lost is by further mutation or by gene flow. Thus subdividing a population is one way to insure that some of these mutations are not lost. If they are lost, chances are that it will be by genetic drift because natural selection is often very ineffective in small populations (Wright, 1932). As human populations increased in size and gene flow became more extensive, which certainly has occurred since the Paleolithic, the alleles could spread and interpopulation variability would decrease. However, due to the random nature of mutation and genetic drift, not all populations can be expected to conform to the general trend of reduction. Thus the interpopulation variability observed by Bailit and Friedlaender (1966) or even the apparent tooth size increase observed by Scott (1979) would actually be expected in some cases under a model of relaxed selection.

Results of the simulations support the contentions of Brace (1963) and Wolpoff (1969) that the probable mutation effect model predicts increased variability within a large population; the accumulation of a variety of

TABLE 1. The frequencies of the mutant alleles (q), the probability of carrying no mutant alleles (Q_0), and the probability of reduction (R) under conditions of various mutation rates (M) and numbers of loci (N).

M	Loci	q	Q_0	R				Mean	Std
				n = 1	n = 2	n = 3	n = 4	n	dev
2×10^{-5}									
	5	0.051	0.594	0.406	0.089	0.012	0.001	0.51	0.69
	10	0.048	0.374	0.626	0.249	0.069	0.014	0.96	0.96
	15	0.047	0.237	0.763	0.414	0.165	0.050	1.41	1.16
	20	0.046	0.150	0.850	0.558	0.282	0.113	1.85	1.33
	25	0.046	0.095	0.905	0.675	0.405	0.196	2.29	1.47
4×10^{-5}									
	5	0.094	0.374	0.626	0.239	0.060	0.010	0.94	0.92
	10	0.090	0.151	0.849	0.550	0.268	0.100	1.81	1.28
	15	0.089	0.061	0.939	0.760	0.507	0.276	2.67	1.56
	20	0.088	0.025	0.975	0.879	0.698	0.475	3.53	1.79
	25	0.088	0.010	0.990	0.941	0.826	0.650	4.38	1.99
6×10^{-5}									
	5	0.134	0.238	0.762	0.395	0.140	0.034	1.34	1.08
	10	0.130	0.061	0.939	0.755	0.493	0.258	2.60	1.50
	15	0.129	0.016	0.984	0.913	0.761	0.551	3.86	1.83
	20	0.128	0.004	0.996	0.971	0.901	0.770	5.11	2.11
	25	0.127	0.001	0.999	0.991	0.962	0.895	6.37	2.36
8×10^{-5}									
	5	0.172	0.152	0.848	0.533	0.239	0.077	1.72	1.19
	10	0.168	0.025	0.975	0.873	0.677	0.440	3.36	1.67
	15	0.166	0.004	0.996	0.970	0.897	0.760	4.99	2.03
	20	0.166	0.001	0.999	0.994	0.972	0.916	6.62	2.34
	25	0.165	0.000	1.000	0.999	0.993	0.975	8.25	2.61
1.0×10^{-4}									
	5	0.208	0.097	0.903	0.647	0.346	0.135	2.08	1.28
	10	0.204	0.010	0.990	0.936	0.806	0.605	4.08	1.80
	15	0.202	0.001	0.999	0.990	0.959	0.883	6.07	2.20
	20	0.201	0.000	1.000	0.999	0.993	0.973	8.05	2.53
	25	0.201	0.000	1.000	1.000	0.999	0.995	10.06	2.84

1.2×10^{-4}								
	5	0.242	0.063	0.937	0.737	0.450	0.206	2.42	1.36
	10	0.238	0.004	0.996	0.968	0.888	0.736	4.76	1.90
	15	0.236	0.000	1.000	0.997	0.984	0.947	7.09	2.32
	20	0.236	0.000	1.000	1.000	0.998	0.992	9.43	2.69
	25	0.235	0.000	1.000	1.000	1.000	0.999	11.75	2.99
1.4×10^{-4}								
	5	0.274	0.040	0.960	0.806	0.546	0.284	2.75	1.41
	10	0.270	0.002	0.998	0.985	0.937	0.831	5.40	1.99
	15	0.269	0.000	1.000	0.999	0.994	0.977	8.05	2.42
	20	0.268	0.000	1.000	1.000	1.000	0.998	10.72	2.80
	25	0.267	0.000	1.000	1.000	1.000	1.000	13.38	3.14
1.6×10^{-4}								
	5	0.305	0.026	0.974	0.859	0.631	0.364	3.05	1.46
	10	0.301	0.001	0.999	0.993	0.965	0.895	6.02	2.05
	15	0.300	0.000	1.000	1.000	0.998	0.991	8.98	2.49
	20	0.299	0.000	1.000	1.000	1.000	0.999	11.95	2.90
	25	0.298	0.000	1.000	1.000	1.000	1.000	14.91	3.23
1.8×10^{-4}								
	5	0.334	0.017	0.983	0.897	0.703	0.444	3.34	1.49
	10	0.330	0.000	1.000	0.996	0.981	0.936	6.62	2.11
	15	0.329	0.000	1.000	1.000	0.999	0.996	9.85	2.56
	20	0.328	0.000	1.000	1.000	1.000	1.000	13.13	2.96
	25	0.328	0.000	1.000	1.000	1.000	1.000	16.36	3.31
2.0×10^{-4}								
	5	0.362	0.011	0.989	0.926	0.764	0.519	3.62	1.52
	10	0.358	0.000	1.000	0.998	0.990	0.962	7.17	2.15
	15	0.357	0.000	1.000	1.000	1.000	0.998	10.70	2.61
	20	0.356	0.000	1.000	1.000	1.000	1.000	14.24	3.02
	25	0.355	0.000	1.000	1.000	1.000	1.000	17.78	3.36

The probability of reduction (R) is tabulated according to a sample range of the number of mutant alleles (n) required for phenotypic reduction. The mean and standard deviation of the number of mutant alleles (n) expected to be carried by an individual is also included (population size = 1000).

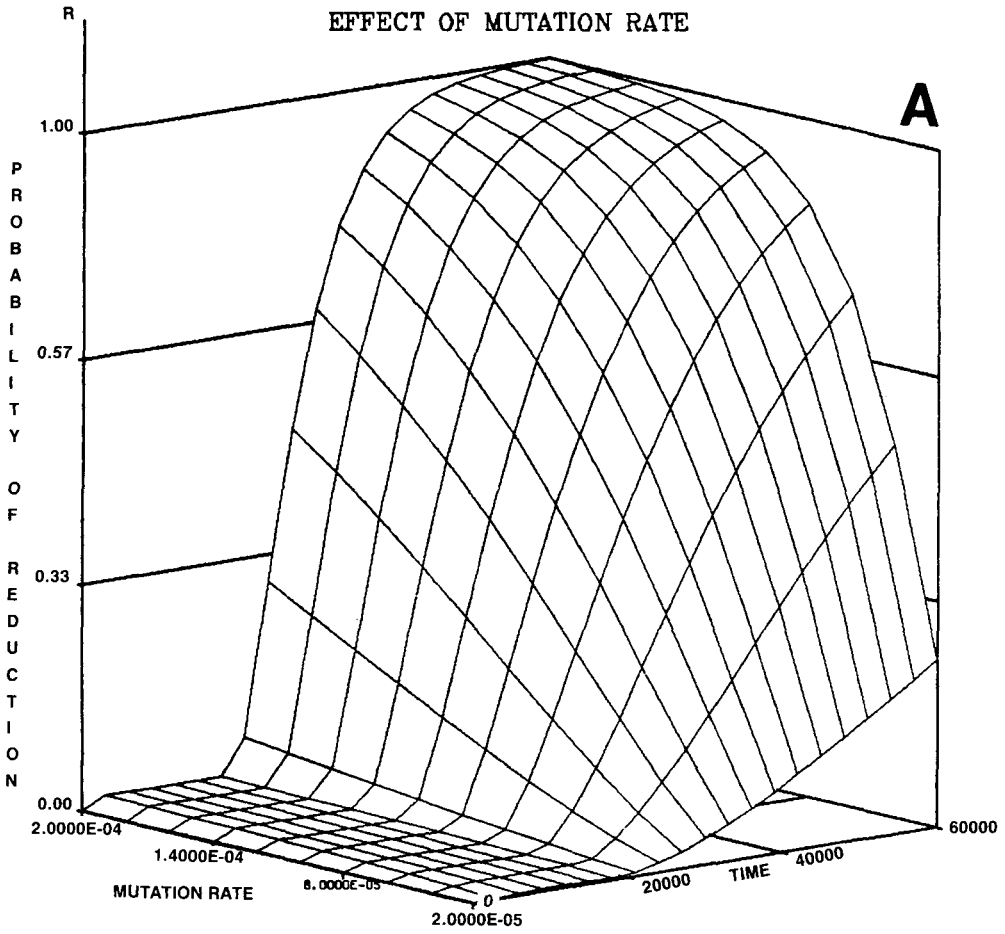

Fig. 1A. The effect of the mutation rate on predicted phenotypic reduction (R) through the simulated time period. The number of loci (N) is held constant at 10, and the required number of mutant alleles (n) is held constant at 2.

allelic forms at alternate loci would yield many genetic polymorphisms in the population. As suggested above, subdividing the population would result in increased variability within the total population. The resultant phenotypic variability within each of the subpopulations would depend on population size, average heterozygosity, and the number of alleles in the gene pool (Chakraborty and Nei, 1982: 310). Furthermore, the variability would depend on the nature of the environment for a given population or subpopulation (Doyle and Johnston, 1977). Consequently, a comparison of tooth size variability among successive fossil "populations" as attempted by Frayer (1978), or between fossil and modern populations, would not necessarily be contradictory to the validity of the model.

Dental asymmetry has been proposed as an indicator of population variability in tooth size. Suarez (1974) has suggested that the problem of finding comparable breeding populations in the fossil record can then be sidestepped by looking at bilateral asymmetry of

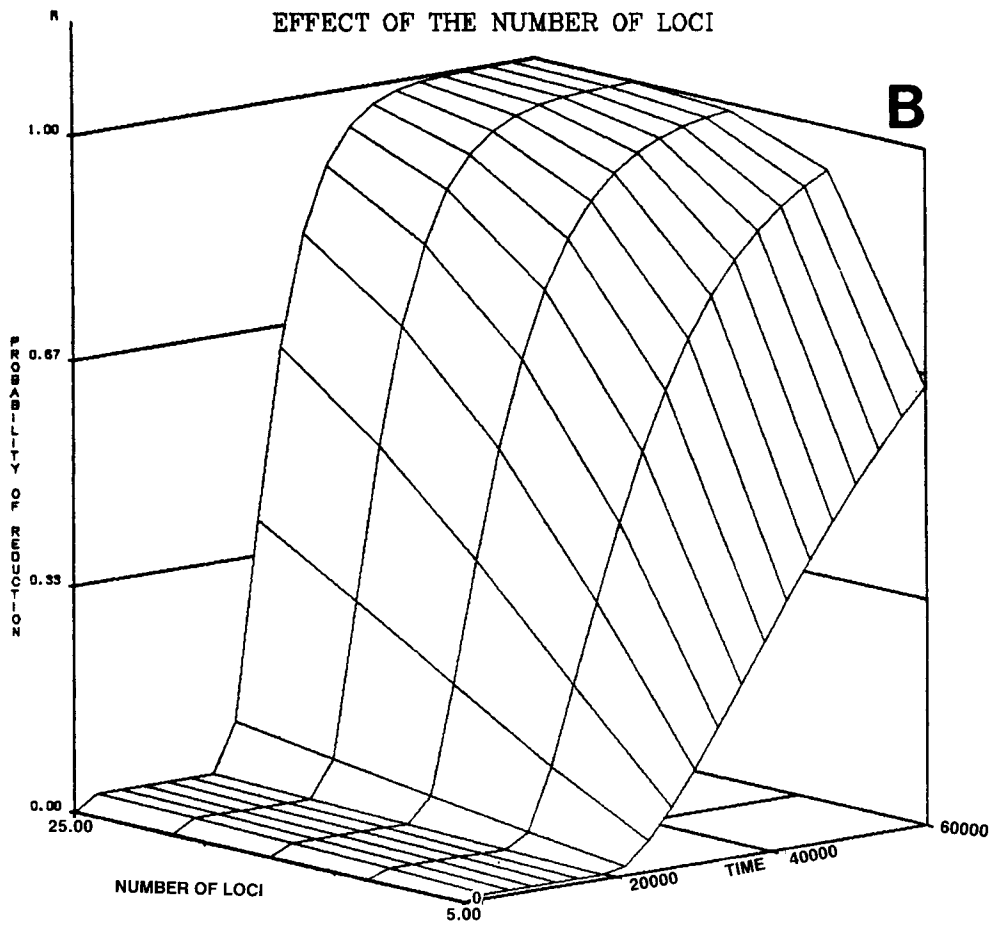

Fig. 1B. The effect of the number of loci (N) on predicted phenotypic reduction (R) through the simulated time period. The mutation rate (M) is held constant at 1×10^{-4}, and the required number of mutant alleles (n) is held constant at 2.

TABLE 2. The range of mutation rates (M) and numbers of loci (N) resulting in a predicted phenotypic reduction throughout 90% or more of the simulated population[1]

Mutation rate	Maximum value of n for each no. of potential loci				
	5	10	15	20	25
0.00020	2	4	7	10	13
0.00018	1	4	7	9	12
0.00016	1	3	6	8	11
0.00014	1	3	5	7	9
0.00012	1	2	4	6	8
0.00010	1	2	3	5	6
0.00008		1	2	4	5
0.00006		1	2	3	3
0.00004			1	1	2
0.00002					1

[1]The value printed on the table is the *maximum* number of mutant alleles (n) required for such phenotypic reduction.

individual tooth size through time, but a further problem is encountered. Studies have shown that the heritability of tooth size asymmetry is very low or negligible in populations where tooth size is highly heritable (Bailit et al., 1970; Townsend and Brown, 1980). This is not to say that such asymmetry is not inherited, but if it is to be correlated with tooth size variability in general then it should have a comparable heritability to tooth size. Consequently, tooth size asymmetry cannot be used reliably as a measure of tooth size variability within a population (see also Doyle and Johnston, 1977).

Another aspect which could alter the predictions of this model is genetic architecture. I have assumed that the loci involved were

autosomal and unlinked. Garn et al. (1965), however, have suggested that a component of tooth size determination may involve an X-linked gene. This would only slightly alter the predictions of the model, and could actually favor greater structural reduction by having a greater effect in males. Likewise, the tight linkage of two loci affecting the same trait would have little or no effect in the absence of selection. Only if a locus of concern was tightly linked to a locus undergoing selection in a population where there is initial linkage disequilibrium between these loci, will linkage have any effect. This could then speed up either the fixation or the loss of a neutral mutation, depending on the nature of the disequilibrium; modeling of this effect would thus be purely hypothetical and restricted to certain types of populations.

The only major aspect of population genetics left for consideration in this model is that of natural selection. There are many models both with and without natural selection which can account for the maintenance of polymorphisms (Livingstone, 1980), but it is difficult to choose the correct model. Natural selection could have continued through time at low levels or may have been reinvoked at various points in time. This could alter the numeric predictions of the model in degree, but does not change the model in principle. On the other hand, the selective forces operating to maintain large anterior teeth could have been reduced long before 40,000 years ago, with the phenotypic effects being unnoticeable in the fossil record until sufficient reduction had occurred. In this case, the more conservative estimates of mutation rates or numbers of loci could be considered to be more acceptable; conversely, low mutation rates and few loci would actually delay a noticeable change in the fossil record (see Figs. 1a and b).

It is the possibility of selective forces acting for the reduction in tooth size which is of the most concern to the critics of the probable mutation effect. There have been many arguments for natural selection as an explanation for aspects of tooth size reduction (see Brues 1966, 1968; Carlson and Van Gerven, 1977; Frayer, 1978; Greene, 1970, 1972; Holloway, 1966, 1967; Prout, 1964; Sofaer et al., 1971). Many possible reasons for this reduction have been suggested, most of which attribute the reduction to selection for hypothetical pleiotropic effects of the genes, as originally suggested by Wright (1964). Any such selectionist model for the structural reduction of human anterior dentition must assume a sufficiently large population size, mutant alleles which result in reduction, and a polygenic system which affects the trait. These are the same assumptions as the model simulated here, yet the further assumption of natural selection acting either on the trait itself or on another trait affected by the same genetic system must also be imposed upon the model. Clearly the probable mutation effect is a simpler model in comparison to the selectionist alternative, yet it adequately explains the observed structural reduction and variation in tooth size under a broad range of conditions.

CONCLUSIONS

Observations of simulated genetic changes through time do not conclusively associate the probable mutation effect with the actual microevolutionary forces which have led to structural reduction of the human dentition. I have only suggested a possible model and a reasonable range of parameters by which the probable mutation effect may manifest itself in the evolution of modern populations. What these parameters actually were certainly varied greatly and cannot be known at this point, though current advances in developmental and population genetics may further restrict the possibilities.

Notions of the causes of structural reduction must be shown to be consistent with evidence from both paleoanthropology and genetics. Within a wide range of parameters and a limited time frame, the model proposed here adequately predicts the observed structural reduction. This, however, does not free us from looking for other possible causes of structural reduction. The model cannot be accepted until we more fully understand the functional relationships of the dental apparatus, as well as the underlying genetic factors which affect the dental structures. On the other hand, the discovery of functional differences associated with reduced tooth size is necessary but not sufficient reason for natural selection to be imposed upon the model. It is up to the selectionist to demonstrate that any such functional differences could be advantageous within a given environment and would be subject to natural selection, given the constraints of a population's size and structure. Thus with our current state of knowledge, the probable mutation effect hy-

pothesis cannot be rejected and stands as a viable alternative to natural selection as an explanation of human dental reduction.

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