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The Consequences of Phenotypic Plasticity in Cyclically Varying Environments: A Genetic Algorithm Study

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By "phenotypic plasticity" we refer to the capacity of a genotype to exhibit different phenotypes, whether in the same or in different environments. We have previously demonstrated that phenotypic plasticity can improve the degree of adaptation achieved via natural selection (Behera & Nanjundiah, 1995). That result was obtained from a genetic algorithm model of haploid genotypes (idealized as one-dimensional strings of genes) evolving in a fixed environment. Here, the dynamics of evolution is examined under conditions of a cyclically varying environment. We find that the rate of evolution, as well as the extent of adaptation (as measured by mean population fitness) is lowered because of environmental cycling. The decrease in adaptation caused by a varying environment can, however, be partly or wholly compensated by an increase in the degree of plasticity that a genotype is capable of. Also, the reduction of population fitness caused by a variable environment can be partially offset by decreasing the total number of genetic loci. We conjecture that an increase in genome size may have been among the factors responsible for the evolution of phenotypic plasticity.

Introduction

The extent to which phenotypes are subject to environmental modification is commonly termed phenotypic plasticity (Gause, 1947; Bradshaw; 1965). Implicit in this definition is the idea that the same genotype can exhibit different (adaptive) phenotypes in different environments. By enlarging the range of selective environments within which a genotype can function effectively, phenotypic plasticity can play an important role in evolution (Via & Lande, 1985; West-Eberhard, 1989).

We have shown previously (Behera & Nanjundiah, 1995) that the definition of phenotypic plasticity can usefully be extended to accommodate the possibility that different phenotypes result from identical genotypes even in the same environment. This would imply that there is a stochastic element to development. It has been argued that in respect of a certain class of demands made on the phenotype, the presence of stochasticity can cause nearly optimal adaptation to be attained more rapidly than in its absence. Hinton & Nowlan (1987) showed in a computational study that under specified conditions phenotypic plasticity can modify evolutionary pathways and accelerate the course of evolution. A more general result is that up to a certain optimal level, plasticity slows down the rate of evolutionary change but improves adaptation; beyond this optimum both the rate of evolution and degree of adaptation decrease (Behera & Nanjundiah, 1995). That result was derived after examining the consequences of plasticity in a fixed environment. Now we relax this assumption and look at the implications of plasticity in a changing environment. We idealize the situation and assume that the environment cycles between two extremes every few generations. By "extremes" we mean that an optimally adapted phenotype in one environment has the lowest possible fitness in the other and vice-versa.

The dynamics of evolution in a variable environment is an intricate problem and bears on

fundamental issues such as genetic assimilation (Waddington, 1961), the evolution of sex (Hamilton *et al*., 1981) and host–parasite interactions (Seger, 1988). Our interest is restricted to asking, under conditions in which the environment varies cyclically, (a) in what manner is genetic equilibrium (fixation) reached? (b) how are the rate of evolution and equilibrium population fitness affected by the possibility of phenotypic plasticity? and (c) how does the degree of adaptation reached at fixation correlate with the level of plasticity?

Model

The model is essentially identical to the one analysed previously (Behera & Nanjundiah, 1995), which in turn was based on that of Hinton & Nowlan (1987). We consider haploid genotypes represented by linear strings of 0s, 1s and *X*s where each symbol stands for one of the possible three alleles at a genetic locus. The total length of the string, meaning the total number of loci, is N. A 0 or a 1 means that the allele at that locus has a constitutive expression and contributes in a predetermined fashion (as described below) to the overall phenotype. An X on the other hand means that the expression of the gene at that locus is facultative; X can act either like a 1 or a 0 allele in terms of its contribution to the phenotype. Which of the two alternatives it adopts is determined by a ''coin-tossing'' procedure, in other words by making use of a random number generator. A complete series of coin tosses performed in one generation on a single genotype is referred to as a *trial*. Operated on each genotype in every generation, each trial produces a set of uniformly distributed (pseudo) random numbers between 0 and 1. By carrying out ''coin-tossing'' independently for each X in a genotype, we decide whether that X mimics a 0 or a 1. The *a priori* probabilities of the 0, 1 and X alleles (i.e. the probabilities used to create the starting population of genotypes) are denoted by p_0, p_1 and p_X respectively, with $p_0+p_1+p_X=1$.

At each ''toss'' every X has a 50% probability of becoming functionally equivalent to a 1 (and with the same probability, of becoming equivalent to a 0). Equivalence refers to the phenotypic effect of the X and applies just for that generation. Given N loci and an *a priori* probability p_X of an X allele, the mean number of *X*s in any genotype is Np_X. The maximum number of coin-tosses for each genotype is restricted to 2^{Np_X}. Coin-tossing is stopped after a particular trial if all the *X*s in a genotype become 1s, otherwise it continues until the 2^{Np_X} trials are exhausted. (Notice that after the coin-tossing procedure is completed, every genotype is indistinguishable, in terms of how it relates to the environment, from some genotype made up solely of 0s and 1s.) Finally, each genotype is assigned a fitness that depends both on its attained phenotype and on the number of tosses of the coin needed to reach that phenotype.

The fitness of a genotype has two components. To simplify the description we assume for the moment that the environment is fixed and that the optimal phenotype in this environment corresponds to a string of 1s.

(a) ''degree of matching'' model for fitness

The first component pertains to the attained phenotypic state without regard to how it is attained. It depends only on the degree of matching between the phenotype (after completion of coin-tossing) and a specified target phenotype which, as indicated above, is a string of 1s.

This component of fitness is defined as

$$W_D=\frac{1}{N}\sum_{i=1}^{N} d_i \qquad (1)$$

where i stands for a genetic locus and $d_i=1$ if the ith locus has a 1 allele and $d_i=0$ otherwise.

(b) ''combined'' model for fitness

For assigning the second component of the fitness we take note of the manner in which a phenotype is attained; this highlights the plastic aspect of the phenotype. The assumption is that the larger the number of coin-tossing trials undertaken in the course of attempting to reach the target, the smaller the value of fitness. This component of fitness is defined as

$$W_P=n/(2^{Np_X}) \qquad (2)$$

where n is the number of trials that still remain after the genotype has attained the target and Np_X is the expected number of X alleles in a genotype. $W_P=0$ when $n=0$ (no tosses left) and $W_P=1$ when $n=2^{Np_X}$ (coin-tossing is not necessary, because the genotype consists of a string of 1s and has the maximum possible fitness right from the start). On the other hand, if a genotype has a constitutively determined phenotype of less than maximum fitness, meaning a phenotype made up of 0s and 1s only, W_P is assigned its minimum value of zero. The total fitness function W_T is calculated as the average of W_P and W_D:

$$W_T=(W_P+W_D)/2. \qquad (3)$$

It is possible to interpret the elements that enter into W_T along the following lines. W_D is that portion of total fitness which is based partly on the genetically specified component of the phenotype and partly on the degree to which the phenotype matches the target after phenotypic transformation of Xs to 1s and 0s; W_P represents that portion which is dependent on the consequences of coin-tossing for physiological adaptation. Depending on how long it lasts (i.e. how many trials it takes), coin-tossing and the concommitant search for the target extracts a cost which is measured in terms of fitness.

The functional form we have chosen for the plastic component of fitness W_P ensures that both W_D and W_P range between 0 and 1. In our earlier study, dealing with the implications of phenotypic plasticity in a fixed environment (Behera & Nanjundiah, 1995), W_P—there called W_{HN}—varied between $1/N$ and 1 instead of 0 and 1. For the values of N used by us the difference is not significant. Also, over and above the "cost" implicit in the definition of W_P, previously we had included an extra cost, paid in terms of a reduction in fitness, associated with coin-tossing. (That was justified on the grounds that the detrimental effects on fitness would increase disproportionately with the number of coin-tossing trials.) Also, the two fitness components in the determination of the total fitness function W_T were assigned different weightages. Based on that experience, we do not expect any qualitative insight to emerge from the incorporation of these additional features into the present model, and therefore have not included them.

Environmental variation is modelled by assigning different target genotypes, or "target states", one for each environment. For the sake of simplicity these are taken to be exact opposites: a string of 1s, i.e. $\{1, 1, \ldots, 1\}$ and a string of 0s i.e. $\{0, 0, \ldots, 0\}$, respectively. The definition of fitness when $\{0, 0, \ldots, 0\}$ is the target state mirrors exactly the definition with respect to $\{1, 1, \ldots, 1\}$ as the target state. As a consequence, if the fitness of a phenotype increases with respect to one environment, it automatically decreases with respect to the other. The environment is changed periodically with a prespecified period which we call a "cycle length". See Fig. 1 for an illustration of how fitnesses are calculated.

When phenotypic plasticity is absent, we define fitness as in the "Degree of matching" model [eqn 1] except that in this situation genotypes are assumed to be made of 0s and 1s only. The presence of phenotypic plasticity is modelled by using the "combined" model [eqns 2–3] for fitness; in this case genotypes contain 0s, 1s and Xs. Evolution is simulated by a version of the genetic algorithm proposed by Holland (1975). An initial set of 1035 genotypes is generated by selecting 0, 1 and X alleles

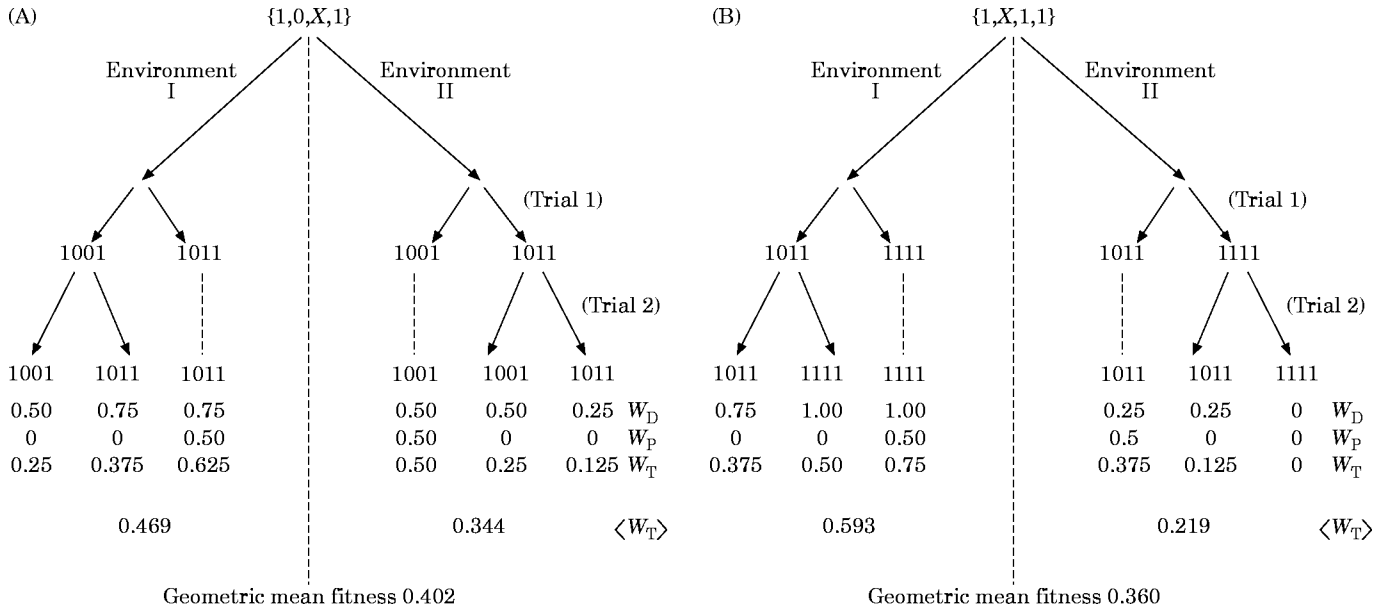

FIG. 1. Illustration of a typical calculation of fitnesses after fixation has been attained under conditions of a cycling environment. Target phenotypes vary between those corresponding to genotypes $\{1, 1, \ldots, 1\}$ and $\{0, 0, \ldots, 0\}$. For simplicity's sake we consider genotypes with $N=4$ loci and $p_X(0)=0.25$. The upper limit to the number of coin-tossing trials per genotype is $2^{Np_X}=2$. At fixation, we assume that the genotype is either $\{1, 0, X, 1\}$ in (a) or $\{1, X, 1, 1\}$ in (B). In both situations it is possible that the full quota of trials has gone through. On the other hand, the phenotype that is theoretically the closest possible to the target may be attained after the first toss (dashed lines).

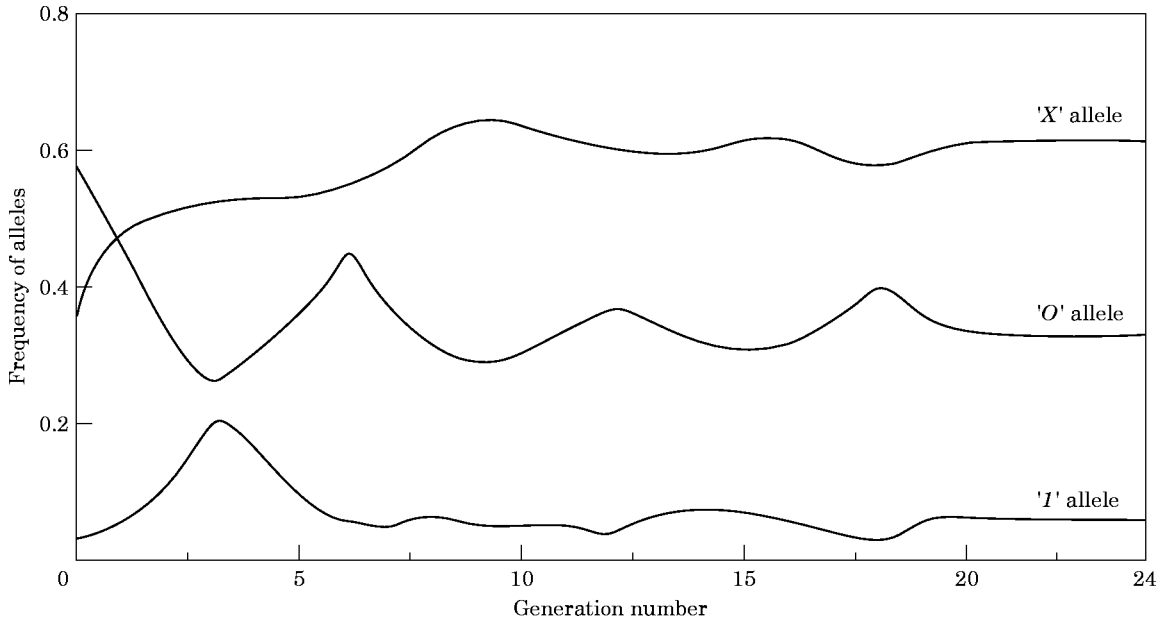

FIG. 2. Allele frequency versus generation number in a cyclically varying environment ("Combined" model; $p_X(0) = 0.39$, $p_1(0) = 0.03$, cycle length = 3). Generation number 0 corresponds to the beginning of the simulation and an environment in which $\{1, 1, \ldots, 1\}$ is the target sequence. The target alternates between $(1, 1, \ldots, 1\}$ and $\{0, 0, \ldots, 0\}$ every three generations.

at random with probabilities p_0, p_1 and p_x respectively. The number of genetic loci is 18 or, in some simulations, 36.

The only elements of the model that remain to be described are mating and recombination. Mating is at random and is assumed to be followed by a single obligatory crossover between parental genomes. One offspring is generated at each mating by choosing a crossover point at random and copying all alleles from the first parent up to the crossover point, and from the second parent beyond the crossover point. After fitnesses are computed, genotype frequencies are weighted in proportion to their relative numbers and arranged in descending order of fitness. Truncation selection is performed by restricting the number of individuals that go on to mate to 46. Random mating generates 1035 ($= {}^{46}C_2$) individuals for the next generation. We continue the analysis for as many generations as necessary until fixation is reached.

Results

The number of generations required for fixation, and the mean fitness of the population during the course of evolution until fixation is reached, are computed for different cycle lengths. As might be predicted, because of probabilitistic nature of the system the outcome varies from one simulation to the next even when initial conditions are held fixed. An average result is estimated by repeating each simulation six times with the same set of initial conditions. In what follows, fitnesses always refer to the target sequence appropriate to that environment. As a measure of long-term average fitness, we compute the geometric mean of the fitnesses in the two environments (see Discussion). The initial environment in all our simulations corresponds to the target sequence $\{1, 1, \ldots, 1\}$. By and large the qualitative outcomes of the "Degree of matching" [eqn 1] and "Combined" [eqns 2–3] models are similar, and sometimes we illustrate a result of one model and at other times of the other. We draw attention to those cases in which the predictions of the two models differ in essential respects.

RATE OF EVOLUTION

Fixation is always reached, irrespective of the cycle length, and the final genotype is usually made up of 0, 1 and X alleles. Figure 2 depicts the results of a typical simulation for the evolution of allelic frequencies in the "Combined" model with $p_X(0) = 0.39$ and cycle length = 3. Allelic frequencies fluctuate until fixation is reached at the 20th generation. At that stage, the situation is as follows with respect to initial values: the frequency of the 1 allele is marginally higher, that of the 0 allele significantly lower, and that of the X allele significantly higher than at the start. The number of generations required for fixation decreases as a

function of increasing cycle length, though it should be noted that the correlation is significant only in the case of the "Combined" model (Table 1; compare a, b with c, d). Put differently, the rate of evolution increases with a slowing down in the frequency of environmental cycling. Also, the time taken to attain fixation increases—equivalently, the rate of evolution decreases—and displays large variations as the number of loci is increased. This is shown in the data as a special case of the "Degree of matching" model (Table 1 and b). Similarly, the rate of evolution decreases as a function of the level of plasticity (Table 1 and d). Table 2 shows that allelic frequencies at fixation vary in a manner that depends on initial conditions. Going through Tables 2A–2C, it is evident that the higher the value of $p_1(0)$, meaning the greater the starting level of adaptation, the higher the value of $p_1(f)$ and the geometric mean fitness at fixation. As the cycle length approaches the time needed to attain fixation in a constant environment with $\{1, 1, \ldots, 1\}$ as the sole target state, the frequency of the 1 allele at fixation tends to increase while that of the 0 allele tends to decrease. Under these conditions the 0 allele can be lost, in which case the final genotype is made of 1s and Xs alone.

MEAN FITNESS

Figure 3 shows the change of population mean fitness in the "Combined" model as a function of generation number. As in the case of Fig. 2, the conditions are $p_X(0) = 0.39$ and cycle length = 3. Note that here the fitness has been computed separately with respect to each of the two different environments. The mean fitness fluctuates and, after 20 generations, displays a regular periodicity on account of fixation.

Because we are dealing with a cyclically varying population mean fitness at fixation, the appropriate measure of long-term average population fitness is the geometric mean of fitnesses in the two environments. Accordingly, we have computed the geometric mean fitness (GMF) under different conditions after fixation has been achieved (Table 2). The trend is highly significant: GMF increases with increasing cycle lengths, that is, decreases as the frequency of environmental change increases [Figs 4A–4B]. In the "Degree of matching" model, as the number of genetic loci is increased the GMF at a particular cycle length is reduced (compare a and b in Fig. 4). The same result holds in the "Combined" model (data not shown). In the "Combined" model, with a given cycle

TABLE 1

Fixation time versus cycle length

	"Degree of matching" model		"Combined" model	
	(a)	(b)	(c)	(d)
No. of loci N	18	36	18	18
Initial conditions:				
p_1	0.03	0.03	0.03	0.03
p_X	—	—	0.16	0.50
Cycle length	Number of generations for fixation (mean ± S.D.)			
1	7.33 ± 1.37	11.23 ± 3.20	32.83 ± 13.40	46.52 ± 12.50
2	5.83 ± 0.90	10.48 ± 2.86	24.50 ± 6.60	42.90 ± 10.20
3	6.67 ± 0.75	8.35 ± 2.12	22.17 ± 7.71	39.20 ± 16.70
4	5.87 ± 1.25	9.42 ± 3.10	—	—
5	6.67 ± 1.25	9.86 ± 1.98	23.17 ± 6.15	38.70 ± 13.50
10	—	—	19.17 ± 2.67	30.80 ± 7.77
15	—	—	20.67 ± 5.22	32.20 ± 8.10
∞	5.62 ± 0.75	8.68 ± 1.69	19.67 ± 2.13	31.26 ± 6.33
r	−0.63	−0.60	−0.86	−0.89
P	0.178	0.208	0.014	0.007

The number of generations required for fixation (G) as a function of the period (T, with a generation as the unit) with which the environment varies as it cycles between two extremes (mean ± S.D. from 6 simulations with the same initial conditions). Cycle length ∞ stands for a fixed environment with $\{1, 1, \ldots, 1\}$ as the target scale. r is the Spearman rank-order correlation coefficient of G with T and P is the probability that a value of $r \leqslant$ (i.e. $\geqslant$ in absolute terms) that found could occur by chance. The case of a fixed environment, in which $\{1, 1, \ldots, 1\}$ is the constant target, is included by taking a nominal value of $T = 100$.

TABLE 2(A)
Mean population fitness versus cycle length

Cycle length	Equilibrium fitness {1}	Equilibrium fitness {0}	Geometric mean fitness	$p_1(f)$	$p_X(f)$
1	0.161	0.348	0.241	0.051	0.463
2	0.172	0.345	0.243	0.052	0.417
3	0.205	0.311	0.252	0.061	0.602
5	0.205	0.316	0.254	0.062	0.555
10	0.294	0.233	0.261	0.277	0.611
15	0.328	0.198	0.255	0.444	0.556
∞	0.360	—	0.360	0.502	0.453
r			0.96		
P			<0.001		

(B)

Cycle length	Equilibrium fitness {1}	Equilibrium fitness {0}	Geometric mean fitness	$p_1(f)$	$p_X(f)$
1	0.202	0.309	0.250	0.052	0.663
2	0.235	0.276	0.255	0.111	0.665
3	0.275	0.242	0.258	0.223	0.669
5	0.333	0.223	0.272	0.164	0.618
10	0.422	0.163	0.262	0.385	0.610
15	0.458	0.161	0.271	0.613	0.387
∞	0.482	—	0.482	0.661	0.333
r			0.89		
P			0.007		

(C)

Cycle length	Equilibrium fitness {1}	Equilibrium fitness {0}	Geometric mean fitness	$p_1(f)$	$p_X(f)$
1	0.291	0.221	0.253	0.271	0.613
2	0.321	0.192	0.248	0.382	0.615
3	0.360	0.161	0.241	0.444	0.546
5	0.385	0.159	0.247	0.662	0.338
10	0.478	0.150	0.268	0.664	0.336
15	0.492	0.144	0.266	0.720	0.280
∞	0.502	—	0.502	0.822	0.178
r			0.96		
P			<0.001		

The dependence of mean population fitness at fixation in a cyclically varying environment, as a function of cycle length. Fitness {1} and fitness {0} refer to initial population mean fitnesses calculated in fixed environments with targets {1, 1, . . . , 1} and {0, 0, . . . , 0} respectively. "Equilibrium fitness" refers to fitness calculated with respect to one or the other target after fixation has been attained. Cycle length ∞ stands for a fixed environment with {1, 1, . . . , 1} as the target state. $p_I(0)$, $p_X(0)$ are initial frequencies; $p_I(f)$ and $p_X(f)$ are final frequencies. r is the Spearman rank-order correlation coefficient of GMF with cycle length and P is the probability that the value of r could be $\geqslant$ than shown purely by chance. Initial conditions: $p_X(0)=0.39$ in all cases; (A) $p_I(0)=0.03$, Fitness {1} = 0.177, Fitness {0} = 0.832, Geometric mean fitness (GMF) = 0.384; (B) $p_I(0)=0.10$, Fitness {1} = 0.206, Fitness {0} = 0.713, GMF = 0.383; (C) $p_I(0)=0.30$, Fitness {1} = 0.290, Fitness {0} = 0.598, GMF = 0.416

length of environmental change, the GMF increases with plasticity (Fig. 4B). Figure 5 shows the increase in GMF as a function of plasticity for various cycle lengths. It can be seen that GMF at one cycle length and plasticity can be equal to GMF for a smaller cycle length (i.e. higher frequency of environmental change) but only at a higher level of plasticity.

Discussion

An idealized relationship of genotype to phenotype would be one in which, irrespective of environmental conditions, a unique phenotype is determined by each genotype. Experimental studies in genetics showed quite early that the real situation was far more

complex, not least because environments vary in space, are inconstant in time and can influence the course of development significantly. The notion of a unique phenotype came to be replaced by that of phenotypic plasticity. A genotype is said to be capable of displaying a plastic phenotype if the phenotype varies from one environment to another (Gause, 1947; Bradshaw, 1965). Intimately linked with the concept of plasticity is that of a norm of reaction, defined as the set of environment-phenotype relationships for a given genotype (Suzuki *et al*., 1981).

There is a small but significant body of theoretical work concerned with the role of genotype–environment interaction in the evolution of phenotypic plasticity and reaction norms. Via & Lande (1985) demonstrated that if evolution was not constrained by genetic correlations between the states of a character expressed in two different environments, a population would eventually attain the optimum mean phenotype in each environment even under conditions of panmixis. Gomulkiewicz & Kirkpatrick (1992) used a quantitative genetics model to examine the consequences of spatial and temporal environmental variation for the evolution of reaction norms; they reached the interesting conclusion that even when the reaction norm was the same, evolutionary trajectories could differ between the two forms of variation.

As stated in the Introduction, the sense in which we use the term "phenotypic plasticity" extends the range of situations to which it can be applied. In our scheme, the stochastic behaviour of the X allele makes it possible for the same genotype to exhibit different phenotypes in different individuals even when environmental conditions are held constant. This is reminiscent of developmental noise, by which is meant the ability of random events in development to cause unpredictable variations in phenotype. The analogy is only partly correct;

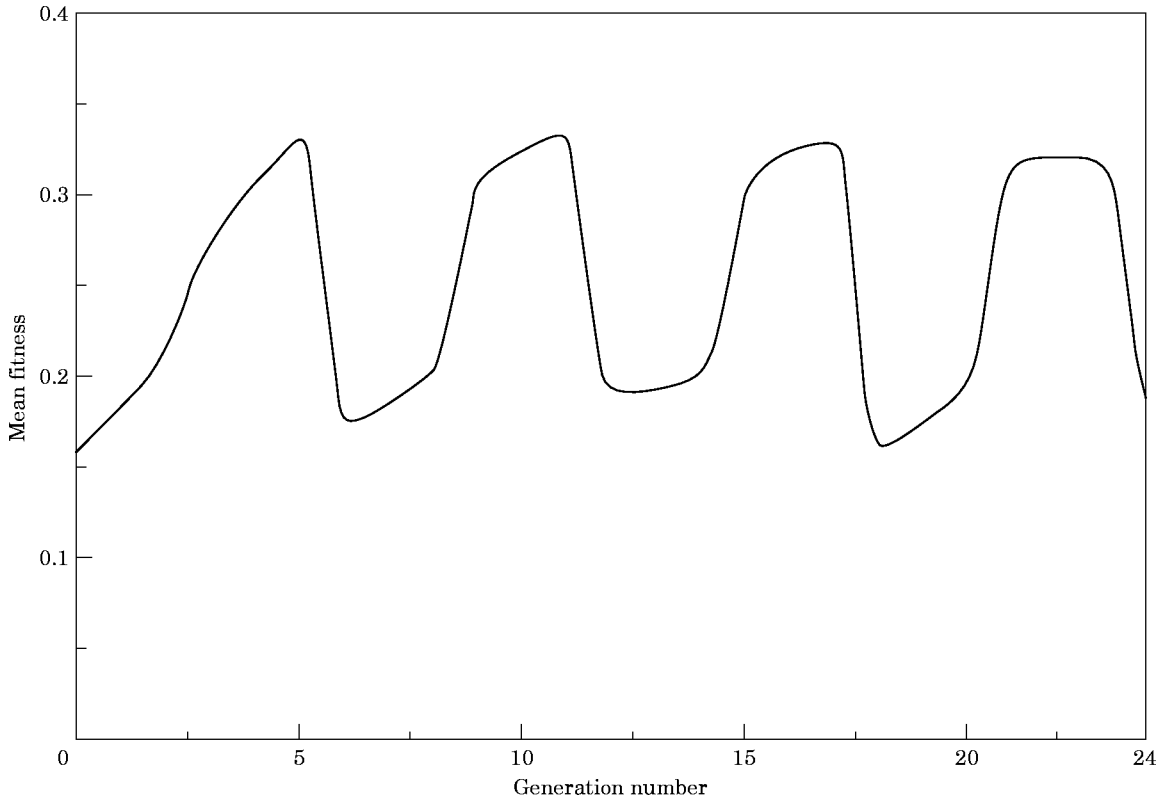

FIG. 3. Variation of mean population fitness as a function of generation number in a cyclically varying environment ("Combined" model, $p_X(0) = 0.39$, $p_1 = 0.03$ cycle length = 3). The environment at the beginning is specified by the target sequence $\{1, 1, \ldots, 1\}$; there is a switch back and forth every three generations between it and $\{0, 0, \ldots, 0\}$. The mean fitness is always computed with respect to the appropriate target sequence for that environment.

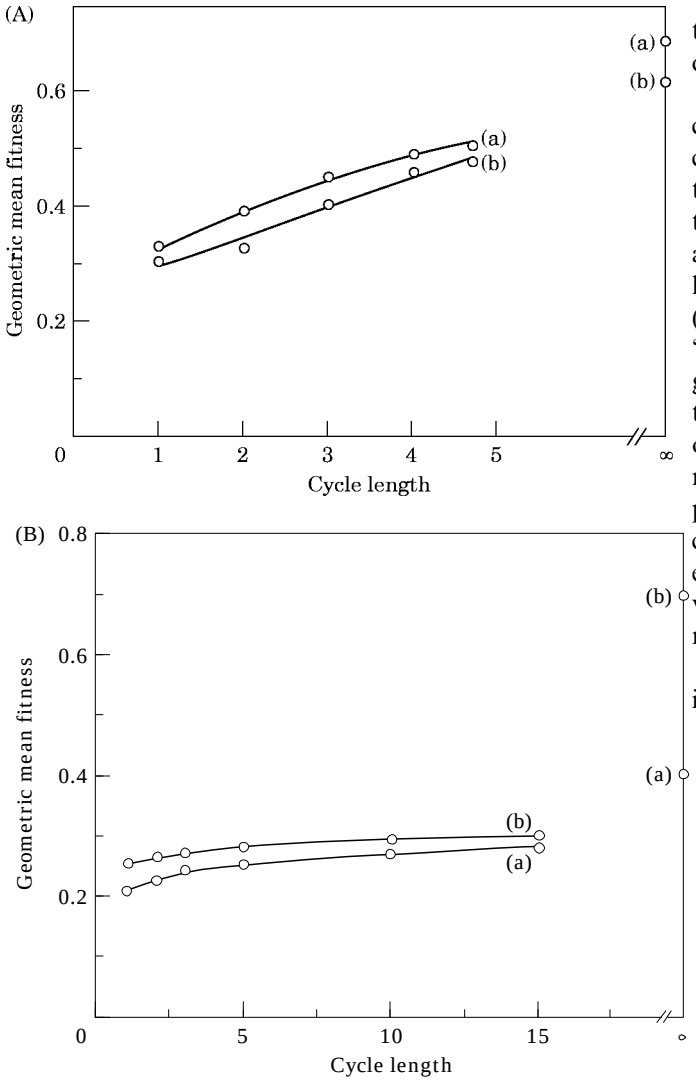

FIG. 4. The geometric mean population fitness at fixation in a cyclically varying environment as a function of cycle length (cycle length infinity corresponds to a fixed environment). (A) "Degree of matching" model. a, number of loci = 18; and b, number of loci = 36. (B) "Combined" model. a, $p_X = 0.16$ and b, $p_X = 0.50$. $p_1(0) = 0.03$ in all cases.

to explain why, we need to get ahead of the story a bit. One of our aims in this series of papers is to examine whether controlled stochasticity can play a role in adaptation. We hope to do so by studying, within the same overall framework, what happens when the expression of an allele at a structural ("visible") genetic locus is modifiable by alleles at regulatory ("invisible") loci. Specifically, we intend to extend our investigation to a model in which a subset of the genome can regulate the probability of an X switching to a 0 or a 1 (in preparation). The present paper, the second in a series, still lacks explicit genetic implementation of the full model; hence the misleading resemblance to developmental noise.

In our simulations selection intensities fluctuate cyclically, at least as far as the 0 and 1 alleles are concerned. This is because of the cyclic variation in the environment, and, therefore, in the preassigned target phenotype. Kimura (1964) showed many years ago that even if the population size was infinitely large, a random fluctuation of selection intensities (with selective neutrality on the average) leads to "quasi-loss" and "quasi-fixation": the distribution of gene frequencies has two peaks that move ever closer to 0 (loss) and 1 (fixation) without reaching them. In our case selection coefficients vary cyclically, not randomly, and populations are finite. Besides, the presence of X alleles makes it possible for the selection coefficient at a locus to be equally high in both environments. In short, in contrast to the situationen-visaged by Kimura (1964), in our case genotypes do reach fixation, and that for more than one reason.

The small population sizes that we have dealt with invites comment. The population size has been limited

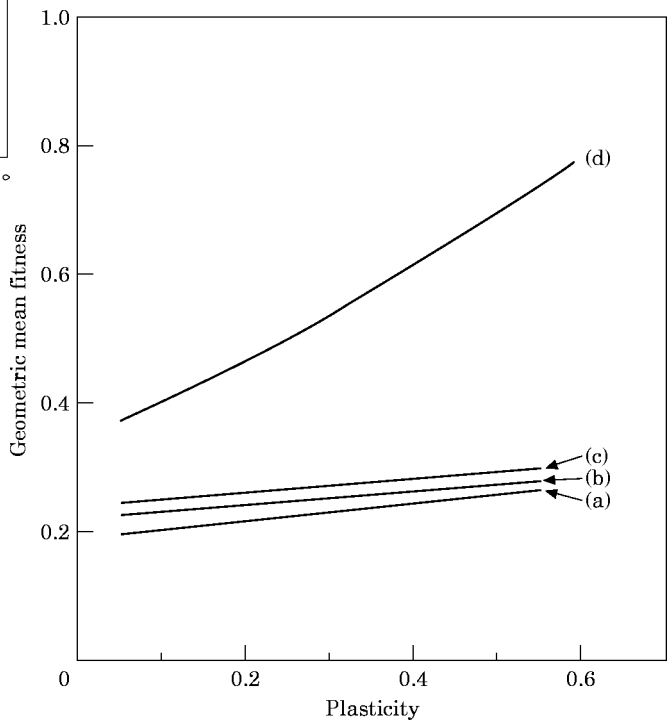

FIG. 5. The variation of the geometric mean population fitness at fixation in a cyclically varying environment as a function of plasticity (i.e. $p_X(0)$) in the "Combined" model. a, cycle length = 1; b, cycle length = 3; c, cycle length = 10; d, fixed environment. The fixed environment corresponds to $\{1, 1, \ldots, 1\}$ as the target. $p_1(0) = 0.03$ in all cases.

to 46 at mating (1035 after mating) and the number of genetic loci to 18 (or 36). This means that drift is likely to play a significant role in our simulations. We have tried to compensate for drift by repeating a simulation six times and taking averages; the fact that we observe clear trends appears to justify the approach.

Our choice of parameters is governed by the following considerations. In the "Degree of matching" model, W_D is the sole contributor to fitness [see eqn 1], and we choose $p_1(0) = 0.03$ because with smaller values, fixation is reached very far from the target more or less on account of drift alone. With higher starting values of p_1, the target is reached extremely rapidly and with virtual certainty. In the "Combined" model, where the fitness is composed of W_D and W_P, we have retained $p_1(0) = 0.03$ (and so $p_O + p_X = 0.97$ initially) in order to compare the fitness function W_T [eqn 3] with that of W_D [eqn 1]. When p_X is much larger than 0.5, limits on computer time became significant.

Genetic equilibrium, meaning fixation, is reached in all cases, whatever the periodicity with which the environment changes. The reason is, of course, drift, but recombination plays a prominent role in the process. In the absence of recombination, natural selection would delay fixation by favouring different alleles in successive cycles. Also, within the scope of the present model, it does not appear that there would be any significant consequence of incorporating multiple crossovers on the dynamics of our system (not shown). We think that this is because the effect of crossovers is masked by drift and other stochastic effects. We conjecture that multiple crossovers will become important in influencing the rate of evolution and population fitness at fixation only at large population sizes and with a large number of genetic loci; and further, that there exists an optimum number of crossovers at which the efficiency of evolution is maximized. These conjectures remain to be tested.

Practical considerations have made us limit the population to a finite size and also to restrict ourselves to a fixed number of 1035 at the zygotic (immediately post-mating) stage of the life cycle in every generation. Fitnesses are therefore relative and a fitness less than one does not mean that the numbers of that individual decrease from one generation to the next. Indeed, in a literal sense, once fixation has been attained, the fitness of the population is the same irrespective of genotype or selective regime. In order to compare the long-term consequences of evolution under differing environmental cycle lengths, or for different values of $p_X(0)$ (different "plasticities"), we imagine that our results remain valid for a situation in which (a) population sizes are not fixed and (b) fitnesses at fixation are scaled (with a value of 1 standing for the basal fitness). The assumptions amount to saying that we wish to examine the implications of our results for growing populations. After fixation, if the mean fitness of a genotype alternates between W_1 and W_2 in two environments and the starting population size is N_0, this would imply that the population goes from N_0 to $W_1 \cdot N_0$ to $W_1 \cdot W_2 \cdot N_0$ and so on each time the environment changes. Under these conditions a constant average fitness which leads to the same long-term population change as the real population undergoes is the geometric mean of W_1 and W_2. Accordingly, it is the geometric mean fitness (GMF) that we have computed.

Because the GMF should reflect the degree of adaptation of a population in the long run, our results enable us to conclude that the level of adaptation finally reached decreases in a variable environment relative to a fixed one. The extent of decrease is inversely correlated with the cycle length, that is, with the periodicity of environmental change (Figs 4A and 4B; in these figures the asymptotic limit of GMF is expected to be the same as that in a constant environment, i.e. with cycle length infinity).

At fixation the GMF is always less than that of a non-evolving population, i.e. one in which allele frequencies are frozen at their initial values [Table 2]. This might appear paradoxical but has a straightforward explanation. Basically, what the result shows is that a population that is well adapted to one set of environmental conditions is, *ipso facto*, poorly adapted to the other set of conditions. The reason is that the environments chosen by us are in a sense "exact opposites". The important point remains that whatever the decrease in long-term adaptation (as measured by the GMF) caused by environmental cycling, the extent of decrease is lower in the presence of phenotypic plasticity than in its absence (compare Fig. 4A with Fig. 4B). Suppose the degree of environmental change is quantified by a parameter K which is some measure of the "distance" between the optimal phenotypes in the two environments. We conjecture that there is a critical value of K below which the geometric mean fitness at fixation in an evolving population is greater than that in a non-evolving population.

The rate of evolution slows down in a variable environment: the more rapid the cycling between the environments, the longer it takes to reach fixation [Table 1(a–d)]. As the number of genetic loci in an individual is increased, both the rate of evolution and

population fitness at fixation decrease. This is true even in the absence of X alleles, i.e. in the absence of plasticity (Fig. 4A), and happens with fixed as well as variable environments and different cycle lengths. The mean fitness of a genetic system which has a small number of genetic loci and is in a rapidly changing environment can equal that of a system with a larger number of loci in a slowly changing environment (Fig. 4A). In other words, the degree of adaptation "lost" on account of a changing environment can to some extent be compensated by decreasing the total number of genetic loci. Decreasing the number of loci amounts in turn to decreasing one measure of complexity of the system. The implication is that other things being equal, systems with many genes—and in that sense more "complex"—take longer to adapt than those with fewer genes (and so "less complex"). Therefore, the larger the degree of uncertainty with regard to the external environment, the better off a system with few genes *vis-à-vis* one with many. The decrease in adaptation caused on account of a change in the environment can be partially or completely compensated, not only by reducing the size of the genome but by increasing the plasticity of the genetic system (see Fig. 5). This means that phenotypic plasticity must have been increasingly advantageous as genome sizes increased during evolution. We note that the inference remains valid until an optimal level of plasticity is attained. Thereafter, an increase in the level of plasticity can slow down the efficiency of evolution (Behera & Nanjundiah, 1995).

The present model makes the transition of an X to a 0 or a 1 entirely a random event. A more plausible model would incorporate an element of genetic regulation in this process. We are working on these lines within the framework of the "Combined" model to investigate the interaction between development and evolution in a variable environment. Our expectation is that there will be a selective advantage associated with modifier genes which facilitate the development of useful adaptations in response to environmental variability.

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