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A Geometric Model for the Evolution of Development

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The idea that early developmental characters are more constrained in their evolution than late developmental characters is one of the most widely accepted generalizations about the relationship between development and evolution. It is apparent, however, that evolutionary changes do occur in early ontogeny and that understanding the nature of these changes is essential if we are to understand a variety of evolutionary phenomena, such as the origin of higher taxa. In particular, we would like to know if certain ecological conditions are more likely than others to produce changes in early development. In this paper, I present a geometric model which predicts that changes in early ontogeny should most often be fixed in environments in which the adaptive landscape is changing drastically over time. In the model, a developmental program defines a set of subsets in a space of possible mutant phenotypes, a fitness function is then imposed on this space. It is demonstrated that early developmental characters, which correspond to high dimensional subsets, are only able to evolve when the fitness function is of low curvature. If the fitness function is thought of as an adaptive landscape, low curvature corresponds to great distance from any adaptive peak. The model is then used to investigate which ecological circumstances are most conducive to evolution of the basic ways in which organisms are put together. It is shown that this model predicts the pattern of nearshore innovation which has been observed in marine paleocommunities. The results are also used to discuss the concept of "developmental constraint". In particular, it is shown that mechanistic interactions in development can constrain the rate of evolution independently of its direction and that, in fact, this constraint on the rate of evolution may be greatest when constraints on direction are least.

Introduction

There has been considerable discussion in recent years of the importance of developmental biology to the understanding of evolutionary patterns (Bonner, 1982; Raff & Kaufman, 1983). Most of the actual research in this area has been directed towards the elucidation of particular developmental mechanisms and analysis of how these could explain patterns in the evolution of particular groups (e.g. Alberch, 1983; Hall, 1984). The paucity of general theoretical models for the evolution of development is largely a result of the fact that most of the developmental mechanisms themselves are still poorly understood.

In spite of this, a few general rules have been put forth about the effects of developmental patterns on evolution. Among the oldest and most widely accepted of these is the idea that early developmental characters are more "constrained" in their evolution than are those which occur later in ontogeny. Interest in this pattern goes back to Von Baer's laws, which simply state the observation that characters of

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early embryos tend to be much more highly conserved across taxa than do those of adults. The mechanism which is most often invoked to explain this pattern derives from the observation that development proceeds by some set of cascading interactions such that a change which is expressed early on may have consequences for a large number of later characters. Riedl (1978) has attempted to formalize this somewhat with the concept of "burden". In Riedel's terminology, early developmental characters are more highly burdened than later ones because a change in early ontogeny has consequences for many subsequent characters through developmental interactions. Such a change thus has a lesser chance of being adaptive than one which is expressed later on.

While most evolutionary biologists would agree that the principals discussed above are generally true, it is clear that they are at times violated. Furthermore, those cases in which early developmental characters, or the structure of the developmental program itself, do change are of particular interest to the study of macroevolution. (Throughout this paper I define a developmental program as a set of rules which tell tissues how to grow and differentiate, along with the temporal sequence in which these rules are read.)

Various authors have noted that "macroevolutionary" change may often involve the fixation of mutations which alter either early developmental structures, or the rules by which development proceeds (Wessels, 1982). Furthermore, by altering the ways in which organisms are put together, such changes may yield phenotypes with the potential to evolve in directions which were not previously available (Gould, 1980). Given that changes in early developmental characters and in the structure of developmental programs do occur and that they are important, our next step is to ask what factors effect the likelihood that such changes will arise. In particular, we wish to know whether certain sets of ecological circumstances are more conducive than others to evolution of the basic rules by which organisms are put together.

Most theoretical models for the evolution of development involve either quantitative genetics (Lande, 1979; Cheverud, 1984; Slatkin, 1988) or hypothesized mechanisms of morphogenesis for particular types of characters (Oster & Alberch, 1982; Oster *et al.*, 1988). I shall present a model which treats the ways that a phenotype can evolve as a collection of subsets of the space of all possible phenotypes, and describes the probability of change in a particular character as a function of the subset in which it exists and local properties of a selection function. This model will then be applied to the question of what environmental circumstances are most conducive to evolutionary change in early development.

Models and Analysis

I will define the one-mutant space $M(\Phi)$ of a phenotype, Φ, as the set of points in phenotype space which can be reached by a single mutation. This space can be thought of as being divided up into a collection of subsets $[m(\phi_i)]$ each of which span the set of points which are accessible by one mutation expressed at a particular stage, i, in development (Fig. 1). These one-mutant subsets $[m(\phi_i)]$ will be of different dimensions depending on how many other characters could potentially be

changed by a random mutation expressed in ϕ_i. The particular collection of subsets $m(\phi)$ which cover $M(\Phi)$ will be determined by the particular developmental program. The entire space $M(\Phi)$ is clearly equal to the union of all the separate $m(\phi_i)$. Figure 1 shows an example for a very simple system. I will assume that the subsets $m(\phi)$ are simply connected (they have no holes).

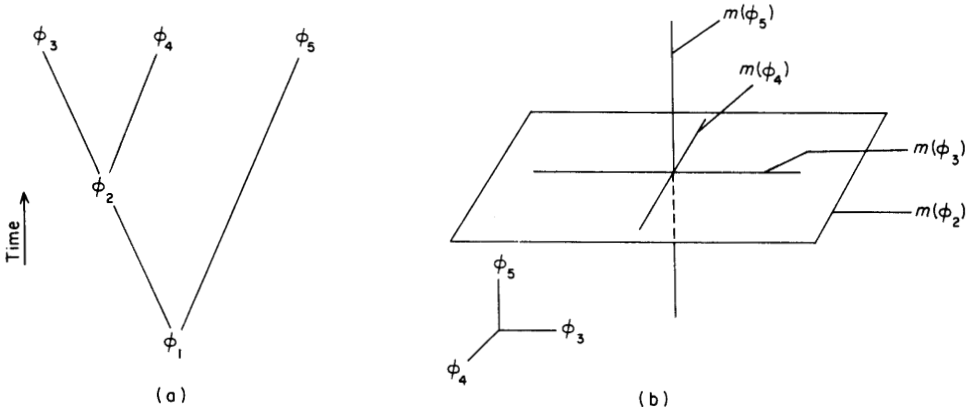

FIG. 1. A simple developmental sequence (a), and some of the one-mutant subsets which it generates. (b) Early phenotypic characters ϕ_1 and ϕ_2 give rise, through ontogeny, to adult characters ϕ_3, ϕ_4, and ϕ_5. For ease of drawing, the phenotype space shown in (b) represents only the adult characters. Mutations which are first expressed in one of the adult characters can shift the phenotype only along one of the lines $m(\phi_3)$, $m(\phi_1)$, or $m(\phi_5)$. A mutation expressed in ϕ_2 would shift phenotype in the two-dimensional subset $m(\phi_2)$.

The dimension of the entire space $M(\Phi)$ is simply the number of different characters being considered. If a mutation expressed in a particular character, ϕ_i, results in a change in phenotype for a total of c_i characters, and if these changes are uncorrelated with one another, then the dimension of the one-mutant subset $m(\phi_i)$ will be c_i. However, n need not be an integer, and fractional values of n correspond roughly to cases in which a mutation changes different characters in a correlated fashion. For example, if a mutation expressed in a particular character changes the phenotype of that character and one other, the dimension will be in the range [1, 2], with the specific value being determined by how tightly correlated the changes in the two characters are. Thus, if a total of N characters change, but in a concerted fashion, we will have $n < N$. Appendix D discusses the mathematics of n and shows how it could be estimated from experimental data.

In the example in Fig. 1 the subsets are all linear, this will not always be the case and is not necessary in the analysis that follows. What I will be assuming is that early developmental characters will tend to have one-mutant subsets of higher dimension than late developmental characters. This corresponds to the idea that a mutation expressed in early development will have consequences for a number of later characters. Early developmental characters will be "burdened" only to the extent that this last assumption holds.

The one-mutant space $M(\Phi_0)$ does not necessarily contain all possible phenotypes. Rather, it is constructed so as to span only the set of phenotypes which can be

reached from Φ by a *single* mutation. Thus, $M(\Phi)$ may look different for different initial values of Φ. This will not be a problem for the analysis that follows so long as nearby values of Φ have one-mutant spaces with similar structure. If this holds all phenotypes in a population would behave essentially the same. Note also that there tends to be little within population variance in early developmental characters. Thus, for such characters, it is reasonable to treat a population as a point. One could potentially construct 2-, 3-, or n-mutant spaces for a particular phenotype, but it seems reasonable to focus on the one-mutant case so long as mutations are rare and come one at a time.

The question that I wish to address is: Can we describe the probability that random mutation will be able to increase in frequency when rare as a function of the point in development at which that mutation is first expressed? In order to answer this question we will need to superimpose some fitness function on our phenotype space.

A Simple Fitness Function

Consider a two-dimensional phenotype subset [similar to $m(\phi_2)$ in Fig. 1] and a fitness function having a single maximum, ○, in the subset, with fitness dropping off uniformly from this point (Fig. 2). For simplicity, the contours of equal fitness considered in this section are spherical. In a later section ("Generalized fitness spaces") I show how to extend these results to surfaces which are not spherical. In the discussion that follows, the fitness function described above can be either frequency independent or frequency dependant (in which case it will change shape as a population moves through it). If the fitness function is frequency dependant, we can only make statements here as to whether or not a mutant can increase when rare.

For the discussion that follows, assume that each axis in our phenotype space represents some morphometric measure which describes a particular character (i.e. length). It will be convenient to scale each axis so that one unit represents the expected magnitude of change in a character caused by a mutation which effects only that character. The metric used will be Euclidian distance.

If the initial phenotype is at Φ_0, then we can imagine a mutation which is expressed in the ontogeny of these two characters as moving the phenotype some distance (D) in this space. Such a mutation will shift phenotype to some point on the circle of radius D centered at Φ_0. Keep in mind that this is all happening in one of the one-mutant subsets as discussed above.

For this simple case, the probability that the mutant phenotype will have higher fitness (at least when rare) than the initial phenotype (Φ_0) is equal to the proportion of the circumference of the circle centered at Φ_0 with radius D that lies inside the circle centered at ○ with radius r. This second circle simply represents a "contour" of equal fitness.

Throughout this discussion, I will treat D as a single value. This allows one to study the effects of the magnitude of change. Clearly, however, mutations expressed at a particular stage will have an associated probability distribution of values of D.

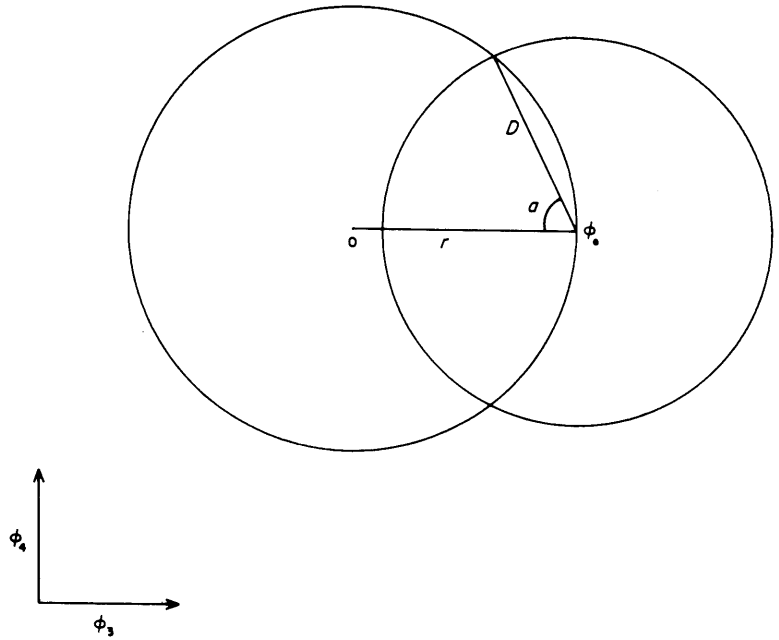

FIG. 2. Phenotype space representing two characters. The large circle on the left corresponds to the set of points with fitness (when rare) equal to that at the point Φ_0. The point ○ is a local fitness maximum. The circle on the right represents the possible results of a mutation of magnitude D acting on Φ_0. Only those mutations which land in the region bounded by the circle centered at ○ are adaptive.

The metric and scaling properties described above make it possible to think of the circle of radius D as a set of equal probability if the actual magnitude of change is described by a multivariate normal distribution.

In Fig. 2 the angle a is given by:

$$a = \text{Arccos}\left[\frac{D}{2r}\right]. \tag{1}$$

Thus, for the two-dimensional case, the probability, f, of increasing fitness is given by:

$$f = \frac{1}{\pi}\,\text{Arccos}\left[\frac{D}{2r}\right]. \tag{2}$$

It is easy to see how this model can be extended to consider any number of phenotypic characters. For a mutation which shifts phenotype in some n-dimensional subset, the circles in Fig. 2 become $(n-1)$-spheres (a circle is a 1-sphere). For this simple case the value of a, defined in (1), remains unchanged and the general equation for the probability of increasing fitness becomes:

$$f = \frac{\int_0^a \sin^{n-2}(\theta)\,d\theta}{\int_0^\pi \sin^{n-2}(\theta)\,d\theta}. \tag{3}$$

See Appendix A for derivation.

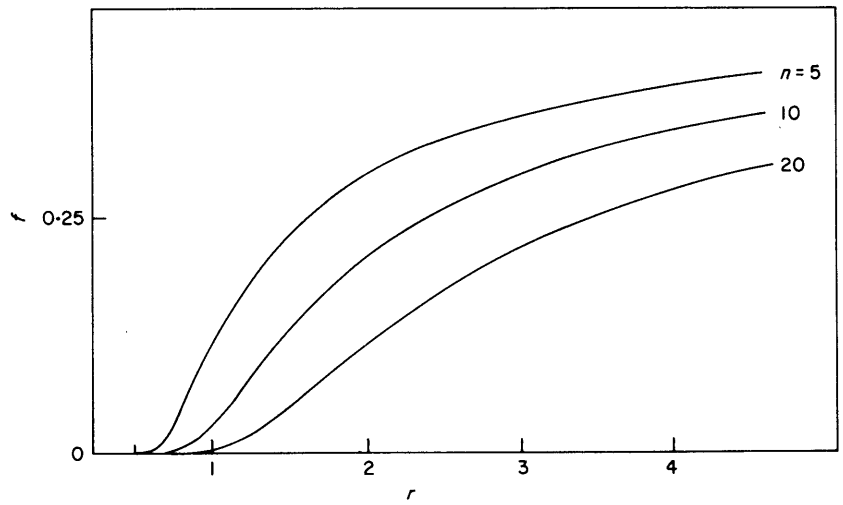

FIG 3. Curves illustrating the probability (f) that a mutant of unit magnitude ($D = 1$) can increase in frequency as a function of the distance from a fitness maximum (r). r can be replaced by $1/K$ as described in the text. n is the dimension if the subspace in which the mutation acts.

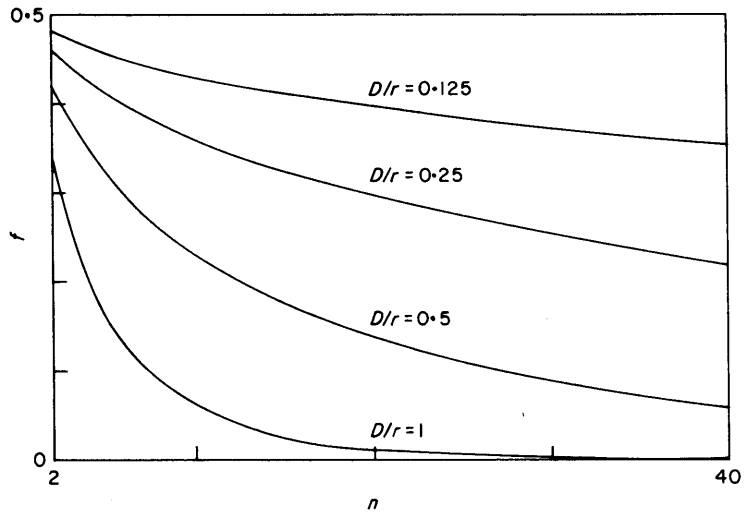

FIG. 4. Probability that a mutation can increase when rare (f) as a function of the dimension of the subspace in which it is expressed (n). DK can be substituted for D/r.

Fisher (1930) considered a similar problem and presented (but did not derive) equations for the probability of increasing fitness due to a shift in three dimensions as well as for the case of many dimensions. Fisher's three-dimensional case can be derived by letting $n=3$ in eqn (3) above. His equation for many dimensions is derived differently, but an identical curve is obtained when n is set very large (i.e. 1000) in eqn (3). Kimura (1983) also derives an equation which converges with Fisher's for many dimensions.

We can now examine the roles of the terms r, D and n in determining the value of f. From eqn (3) it can be shown that;

$$\frac{\partial f}{\partial D}=-\frac{1}{2Ar}\left[1-\left[\frac{D}{2r}\right]^2\right]^{(n-3)/2}, \tag{4}$$

and

$$\frac{\partial f}{\partial r}=\frac{D}{2Ar^2}\left[1-\left[\frac{D}{2r}\right]^2\right]^{(n-3)/2}, \tag{5}$$

where

$$A=\int_0^{\pi}\sin^{n-2}(\theta)\,d\theta. \tag{6}$$

The derivation of eqns (4) and (5) is explained in Appendix B. The term A is simply the denominator of eqn (3), and is constant for fixed n. As would be expected, either decreasing the phenotypic magnitude of a mutation (D) or increasing the distance from an apparent optimum (r) increases the probability that the mutation will be beneficial. Note that both of these values enter into eqn (3) via the dimensionless term D/r. Thus, the magnitude of a mutation is best measured relative to where the initial phenotype lies in an adaptive space. Analysis of eqn (3) with respect to the effects of changing the value of n (the dimension of the subset being considered) is somewhat more difficult to carry out and is discussed in the Appendix B. The effects of n can be seen visually, however, in Figs 3 and 4.

It is not surprising that, as one moves closer to a fitness maximum, the proportion of mutations which are adaptive drops off. The important point illustrated by eqn (3) and Fig. 4 is that, for mutations expressed at different stages in development, the rate at which this proportion drops off is strongly determined by what stage one considers. Thus, characters at different stages in ontogeny should be expected to respond differently to where the entire system finds itself in an adaptive space.

Complex Adaptive Spaces

The model presented above can easily be modified to take into account the possibility that the set of phenotypes which could increase when rare is not connected, as would be the case for a fitness function with multiple maxima. Figure 5 illustrates how this would be done. We are now interested in the proportion of mutants which fall into either of the regions bounded by the surfaces I_1 or I_2. In order to calculate the probability that a mutation of magnitude D shifts the initial phenotype (Φ_0) into an adaptive region whose boundary does not include Φ_0, we

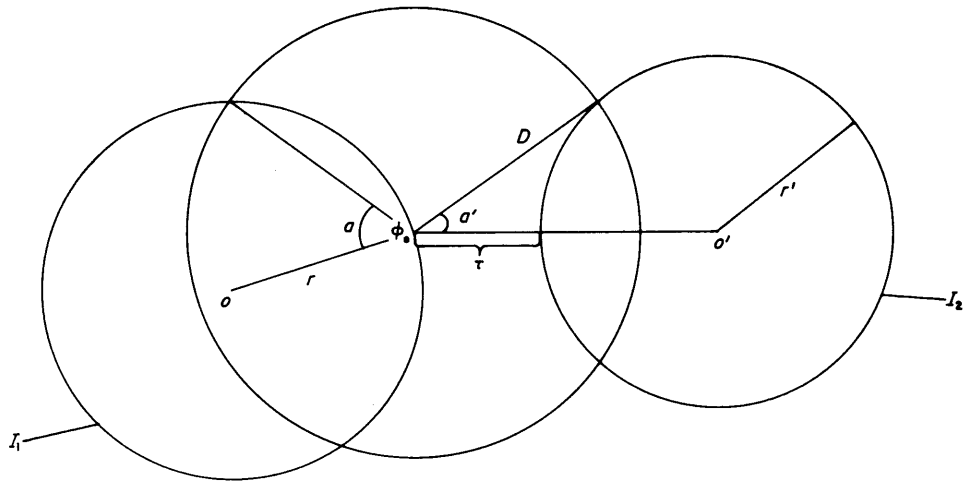

FIG. 5. Model for the case of multiple fitness maxima. A mutation could shift the phenotype Φ_0 into either of the regions of higher fitness, bounded by the surfaces I_1 and I_2.

replace the value a in eqn (1) with the value a', defined by:

$$a'=\text{Arccos}\left[\frac{D^2+2r'\tau+\tau^2}{2D(r'+\tau)}\right], \tag{7}$$

where D is defined as before, r' is the radius of the adaptive region bounded by I_2, and τ is the minimum distance from Φ_0 to I_2. The derivation of eqn (7) is presented in Appendix A. The value a' is now substituted for a in eqn (3).

If there are k non-overlapping regions in which a mutant could increase when rare, then the total probability of a random mutant landing in one of them is simply the sum of the independent probabilities of landing in each one. We could think of such a fitness function as defining an adaptive "landscape" which appears smooth from the standpoint of small scale changes but looks "rugged" (or uncorrelated, in the terminology of Kauffman & Levin, 1987) for large jumps.

Of particular interest is the effect of the value τ (distance to the boundary of an adaptive region) on the probability of jumping into that region. This is illustrated in Fig. 6, which shows the effect of increasing τ in subsets of different dimension. For all of the curves in Fig. 6, the value of D (the distance jumped) is scaled such that $D=r'+\tau$. Note that even for moderate values of n, the probability of successfully jumping from the domain of one "adaptive peak" to that of another becomes prohibitively small very quickly. Thus, when such jumps do occur, we should expect them to result from changes in characters with low dimensional one-mutant spaces.

Generalized Fitness Spaces

So far I have assumed that the boundary (I) of an adaptive region curves away from any initial phenotype at the same rate in all directions. This is inherent in the

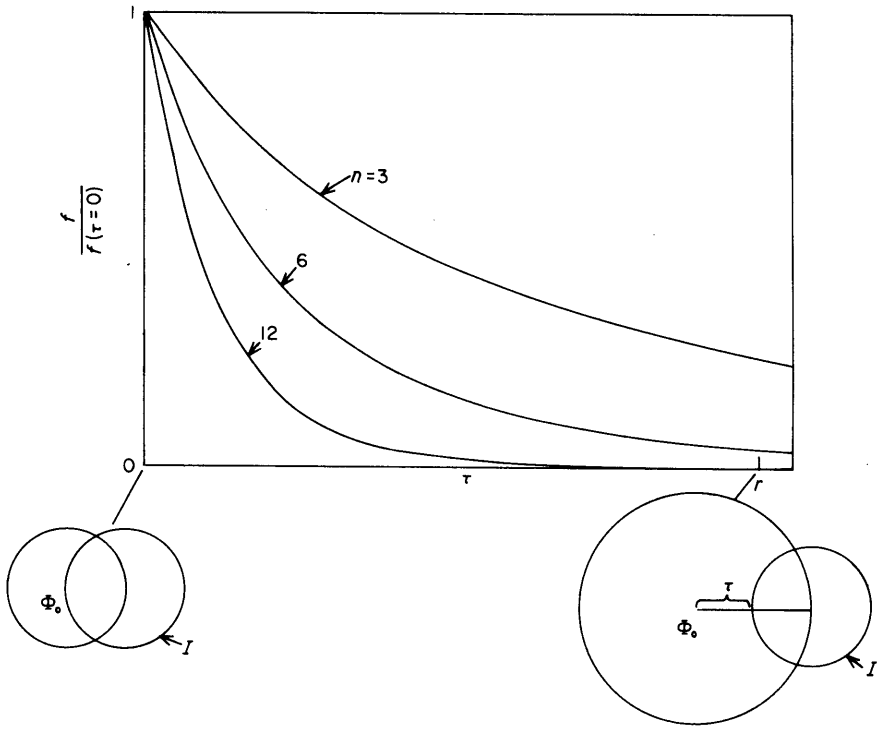

FIG. 6. The effect of the distance (τ) to the boundary of a region in which a mutant could increase when rare on the probability that such a mutant would land in that region. This probability is scaled to be relative to what it would be if the initial phenotype lay on the boundary of the adaptive region. The distance jumped (D) is always equal to $r'+\tau$, the distance to the optimum phenotype. Note that even if conditions were right for changes in early development, it would still be very unlikely that such changes would involve a shift to a new "adaptive peak".

assumption that all connected adaptive regions are spherical. While this assumption is reasonable for studying general trends, it will almost certainly not hold in most real cases. I would therefore like to generalize the model to include cases in which the equal fitness surface curves away to different degrees in different directions (Fig. 7). Furthermore, for both practical and philosophical reasons, it would be desirable to describe the probability of a particular change as a function of the local (with respect to D) properties of the fitness function, rather than having to invoke the existence of some distant "optimum".

In considering this more general case, the term r no longer has any real meaning since we are no longer dealing with spheres. Instead, I will consider the set of terms $k(i)$, with $i=1,2,\ldots,n-1$ for an n-dimensional subset. These k terms represent the degree to which the surface I curves away from the initial phenotype (Φ_0) in different directions. More specifically $k(i)$ is the curvature along the ith orthogonal curve lying on I and passing through Φ_0. These curves ($n-1$ of them) are chosen so that they represent directions of local extreme curvature. For further discussion

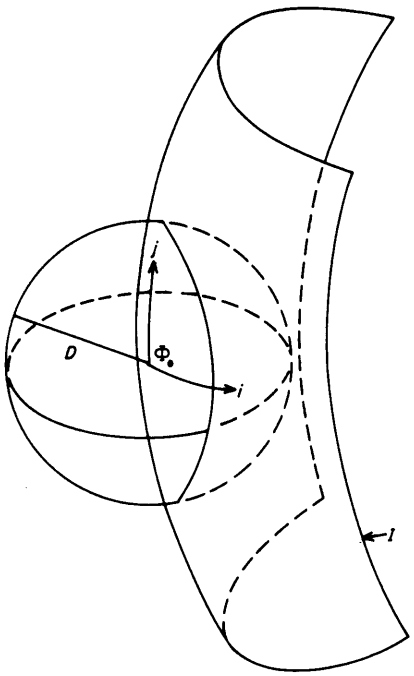

FIG. 7. Model for a fitness function for which the surface of equal fitness to Φ_0 (I) curves away from the point Φ_0 with curvatures $k(i)$ in direction i and $k(j)$ in direction j. In this case a mutation shifts the initial phenotype a distance D in a three-dimensional subspace.

of the concept of curvature, see Spivak (1979). For comparison with the models presented earlier, it is sufficient to state that the curvature of a circle of radius r is $1/r$. The $k(i)$ terms can be thought of as a measure of how fast fitness drops off in different directions.

We can now re-phrase eqn (1) and (3) to consider the case of generalized fitness functions. As is demonstrated in Appendix C, for n large, the actual value of f can be approximated by replacing the term a in eqn (3) with a_k, defined as:

$$a_k = \text{Arccos}\left[\frac{DK}{2}\right], \tag{8}$$

where

$$K = \frac{1}{n-1}\left(\sum_i \frac{1}{k_i}\right)^{-1}. \tag{9}$$

If none of the $k(i)$ terms is close to zero, then the "effective curvature", K, is the harmonic mean of the individual $k(i)$ values. Note that this quantity is most strongly determined by the smaller values of $k(i)$. Thus, directions in phenotype space in which the gradient of fitness is small have a disproportionate effect on the probability that a random mutant can increase. An important fact (demonstrated in Appendix

C) is that eqns (8) and (9) can also be thought of as describing the case in which the fitness function is changing such that the surface of equal fitness is changing shape over time. This means that time periods in which selection on many characters is relaxed (K is low) greatly increase the probability that mutations expressed early in development can become established. This last result means that, even if we do not know the exact geometry of a fitness function (as we never do), we can still make predictions based on environmental variance.

Discussion

I have presented a simple model in which a phenotype space is divided into a collection of (possibly overlapping) subsets. Each of these spans the set of phenotypes which could be reached by a single mutation expressed at a different stage in development. The principal parameters of interest are the distance over which a mutation shifts a point in phenotype space (D), the dimension of the subset in which this shift occurs (n), and some measure of the geometry of the fitness function in the vicinity of the initial phenotype (r and K).

Of these parameters, the first two are properties of the developmental program itself and the third is a function of the selective regime, which is determined by ecological factors. Thus, we can start to ask simple questions about how the ecological context that a developmental program finds itself in effects how it evolves.

In addition to having higher values of n, mutations expressed in early ontogeny may well shift phenotype a greater distance (D) than those expressed late. The model presented above can be used to analyze the effects of changing D as well as n. In this discussion, however, I shall focus on the effects of dimension (n). Kauffman (1989) discusses the consequences for evolution if mutations in early development shift phenotype a greater distance on a rugged landscape.

When Should Burden be Important?

Riedel (1978) has argued that early developmental characters should be more "burdened" in their evolution than those which are expressed later on. The manifestation of this is generally held to be the pattern described by Von Baer's laws. In the model presented above, the dimension of the subset in which a mutation is expressed (n) will be related to how early in development that expression occurs (Fig. 1).

Figure 4 illustrates the effect of n on the probability that a random mutation can increase in frequency for various values of D/r (or DK). When the fitness function is highly curved (D/r, DK large), the probability that a random mutation could get established drops off quickly as the dimension of the subset in which it is expressed increases. Thus, for highly curved fitness spaces, early developmental characters should in fact evolve much more slowly than later ones. Notice, however, that as the fitness function becomes less curved (as would be the case when a population finds itself far from an "adaptive peak") increasing n has a lesser and

lesser effect. Thus, burden becomes less of a factor when populations find themselves spending more time far from local optima.

This last result allows us to make some predictions about which sorts of ecological conditions should be conducive to evolution in the basic structure of developmental programs. Consider populations which spend much time far from local fitness optima, as might be the case in environments which are subjected to frequent perturbations (due to climate, local invasions by other species, etc). In such situations, the mutations which become fixed should involve a higher proportion of fundamental changes in the ways that the organisms are put together than in more "stable" environments.

Note that this argument can also be made without having to refer to "optimum" phenotypes. By eqn (9) and Appendix C, the effective curvature (K) is approximately equal to the harmonic mean of the curvatures over time. Thus, increasing the variance around the expected curvature will tend to decrease the value of K.

The arguments presented above indicate that shaking up the environment in which a population finds itself will have the effect of increasing the probability (f) that a random mutant expressed at any stage in development will be adaptive. It is important to note, however, that the magnitude of this increase will not be equal at all stages of development (indicated by the value of n). This is illustrated in Fig. 8, which shows the degree to which reducing the value of K increases the value of f throughout development. As K is decreased (as would occur in successively more disturbed environments), the probability that a random variant is adaptive goes up

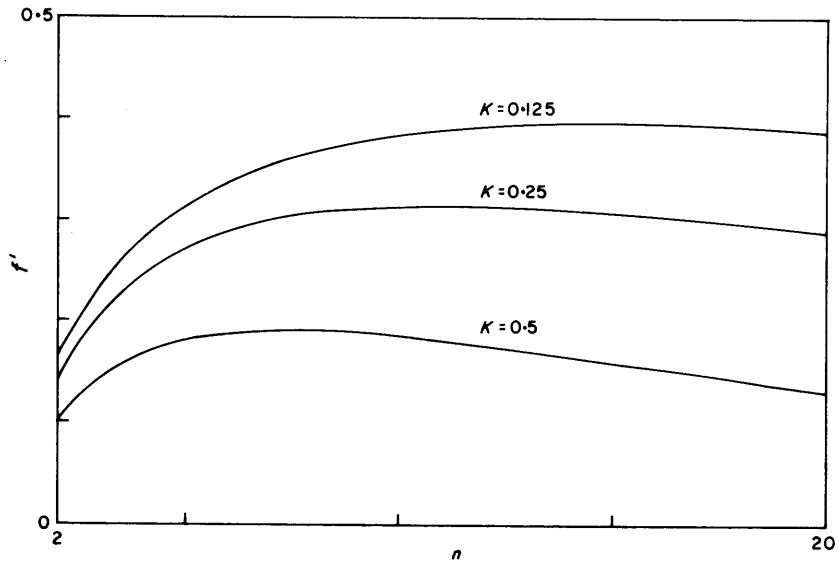

FIG. 8. The effect of reducing the value of K (as would occur when the environment becomes less stable) on the probability that a random mutant is adaptive. f' represents the change in f as K is reduced from $K = 1$ to the values shown, [i.e. $f'(K = 0{\cdot}5) = f(K = 0{\cdot}5) - f(k = 1)$]. Note that the change in $f(f')$ is greatest for intermediate values of n.

fastest for intermediate values of n. Thus, we should expect populations in highly disturbed environments to differ from those in more stable ones by having increased rates of evolution in early stages of development but not necessarily in the latest stages.

This pattern is exactly what has been documented by Jablonski *et al.* (1983) and Sepkoski & Miller (1985) for evolution in marine invertebrate communities. In their studies of paleocommunities, these authors found that, although speciation rates are higher in offshore environments, those speciation events which occur in nearshore environments more frequently result in the appearance new families and orders. Thus, the rate of evolution of general body plans of animals appears to be higher in nearshore environments. Jablonski *et al.* (1983) specifically note that the nearshore communities studied appear to have been more frequently disturbed and are more often subjected to local extinctions and recolonizations. Thus, these communities fit our expectation that those environments in which the fitness function is more variable over time should produce a greater proportion of novel developmental changes. It is important to note that the pattern of nearshore innovation was not found for lower taxa (genera and species). This fits exactly with the expectations of the present model (as discussed above and in Fig. 8) but is at odds with most of the suggested mechanisms so far put forward to explain the data.

A General Scenario

As is illustrated in Fig. 1, a particular developmental program defines a collection of one-mutant subsets. At any time, mutations could arise which would shift the phenotype in any on these spaces. In addition to this, low dimensional subsets are likely to harbor a certain amount of standing genetic variation such that shifts in these could occur even in the absence of mutation. Subsets of higher dimension, however, are not likely to harbor much variation since, as illustrated by Fig. 4, nearly all such variation would be deleterious.

We can imagine a scenario in which we start off a population of phenotypes in a space with a fixed fitness function. If we start our population off far from any stable point, the fitness surface will, on average, have relatively low curvature. Thus, new variation which is expressed over a large range of development (early and late) has a roughly equal chance of increasing when rare (Fig. 4). Evolutionary change will therefore be spread throughout development.

As the system gets close to some stable point, mutations expressed in early development will have a lower and lower chance of increasing in frequency as the adaptive surface becomes more curved (r, the distance to the stable point, gets small). Thus, early developmental characters will tend to get "stuck" downslope from their hypothetical optimum phenotype.

This is essentially the same result as was found by Kauffman & Levin (1987) for evolution on an uncorrelated landscape of high dimension. Using the model presented here, we can see that how long evolution would continue for a particular character is a function [defined by eqn (3)] of the dimension of the one-mutant subset for that character. It seems likely that the optimum phenotypes for late developmental

characters will be determined in part by the phenotypes of earlier characters. This would be the case if the phenotypes of early developmental characters determine the nature of "tradeoffs" (Rosenzweig *et al.*, 1987) between later characters. In the language of quantitative genetics, changes in early ontogeny could change the developmental correlations between later characters. Thus, even if late developmental characters do get near to some "optimum" (evolutionary stable) phenotype, it may not be possible ahead of time to predict where this point will be.

Note that, in this scenario, early developmental characters can only change under a limited set of circumstances. Thus, they should be expected to show a more punctuational pattern of evolution than late developmental characters.

Evolutionary Dynamics and "optimization"

The scenario presented above has consequences for the applicability of optimization or ESS theory (Vincent & Brown, 1988). Even if we imagine that all evolutionary trajectories ultimately settle down, it may not be possible to predict where they will settle down. Specifically, if an evolutionary trajectory involves changes in early developmental characters (or in the basic rules by which development proceeds), then these characters will only be changing so long as the fitness "surface" that they live on is of low curvature. But this is precisely the circumstance in which our ability to predict the direction of their evolution is lowest, since there is then a large region of phenotype space into which evolution could move a population.

To use the terminology of Brown & Vincent (1987), even if we can define a fitness generating function (G-function) for a character with a high dimensional one-mutant subset, it will not accurately predict the evolutionary trajectory of the character. We would still use such a function to describe the evolution of late developmental characters, but only so long as the system stays relatively close to an ESS. Otherwise, earlier characters which could themselves determine properties of the fitness functions for later characters may start to change also. Thus, modeling evolutionary dynamics with optimization or stability criteria will only be of value for systems which are closely tracking local fitness maxima (it should be noted that many interesting questions to which these types of models have been applied, such as those involving the coexistence of similar species, may well satisfy this last criterion).

The preceding discussion has assumed a fixed fitness function which will allow evolutionary trajectories to settle down. It is likely that not all evolutionary dynamics will be so well behaved (Shahshahani, 1979; Akin, 1982). Equations (8) and (9) allow us to describe the probability of a mutant increasing in frequency when rare as a function only of the local geometry of the fitness function, which need not have any stable fixed points.

Constraints

It is clear that the dimension of the one-mutant subset of a character, along with the selection regime to which it is subjected, can have a significant effect on the rate at which is can change. We therefore might be tempted to term this a constraint

on the rate of evolution. Note that this differs from the general use of the term "constraint" (Maynard Smith *et al.*, 1985; Levinton, 1988), which involves some restriction on the direction of evolution. Moreover, by the model presented here, the overall rate of evolution will be most constrained for characters for which a mutation could shift phenotype in the greatest number of directions.

If this sounds paradoxical, consider the fact that constraints on the direction of evolution are generally modeled in terms of correlations between characters (Cheverud, 1984). These correlations, however, effectively reduce the dimension of the subset in which phenotype can change (see Appendix D), thus, by eqn (3) (all else held equal), increasing the proportion of random mutations which could increase when rare.

As an example, consider the suite of characters which are allometricaly "correlated" with body size (Calder, 1984). Various authors have noted that allometric relationships can strongly effect the direction of evolution (Levinton, 1988). It has also been pointed out, however, that evolution in body size often occurs very rapidly, particularly in the case of dwarfing (Raff & Kaufman, 1983). A similar situation arises in the case of heterochrony (Gould, 1977; Alberch *et al.*, 1979). Large scale shifts in the timing of developmental events are likely to involve changes in many characters, but often in a correlated or predictable, way. Thus, we would expect there to be relatively little constraint on the rate at which such changes are fixed in populations. This may explain why heterochrony appears to be so important in macroevolution.

Throughout this discussion, I have assumed that early developmental characters will have one-mutant subsets of high dimension, whereas those which occur later in ontogeny will be of low dimension. While this is probably true in general, there will certainly be exceptions. It seems likely that some changes in early ontogeny could lead to either changes in few later characters or, more likely, highly correlated changes in a variety of later characters. This might be the case when there are inductive interactions between characters throughout ontogeny such that they develop in unison. The phenomenon of canalization (Waddington, 1957) represents another example in which a change in early ontogeny has few consequences later on. Thus, we should not be surprised to find some early developmental characters which are not highly "burdened".

The general model presented above makes a variety of simplifying assumptions (such as that mutations can only be considered one at a time). In spite of the simplifications, it allows us to investigate, and make predictions about, some general trends in the evolution of developmental programs which are not amenable to other methods. A variety of other questions might be amenable to this sort of geometric analysis. For example, changes in the developmental correlations between characters will have the effect of changing the orientation of some of the one-mutant subsets (Fig. 1). The effects of such changes on evolution could be investigated under different sorts of selection regimes. Similarly, one could assess the impact of changing the amount of random phenotypic variation on different stages in development. It is thus hoped that this model will compliment other approaches to the study of evolution and development.

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APPENDIX A

Derivation of Eqns (3) and (7)

We wish to calculate the proportion of the "surface area" (Hausdorff measure, for an n-sphere) of the sphere centered at Φ_0 that lies inside the sphere centered at (○). First note that the surface area (L) of an n-sphere can be written as $L = gD^n$ where D is the radius of the sphere and g is not a function of D. Given this, we can slice up the $(n-1)$-sphere centered at Φ_0 into a set of sections whose centers lie along the line connecting Φ_0 and 0. Each of these sections will have surface area;

$$L = gD^{n-1} \sin^{n-2}(\theta)\, d\theta. \tag{A.1}$$

The proportion of the first sphere which is bounded by the second is then this function integrated from (0) to a [defined by eqn (1)] divided by the same function integrated over the entire surface. Thus:

$$f = \frac{gD^{n-1}\int_0^a \sin^{n-2}(\theta)\, d\theta}{gD^{n-1}\int_0^\pi \sin^{n-2}(\theta)\, d\theta}. \tag{A.2}$$

Note that this assumes that in each subset, the distance (r) to the fitness maximum *in that subset* is the same as for all others. If the entire one-mutant space has a single fitness maximum, then subsets which do not include this point will "see" a projection of it. Depending on how a subset is oriented, the value of r in it will tend to be $\leq$ the value of r in other subsets of higher dimension. On average, the minimum distance from a random subspace to the actual fitness maximum decreases by a factor of $\sqrt{2}$ for each dimension that is added. Thus, for $n \geq 10$, values of r will be largely independent of n.

Equation (7) is simply the case in which the center of the first sphere does not lie on the surface of the second. Thus, we need only recalculate the value of a (the angle to which we integrate the area function). Figure A1 shows how this is done.

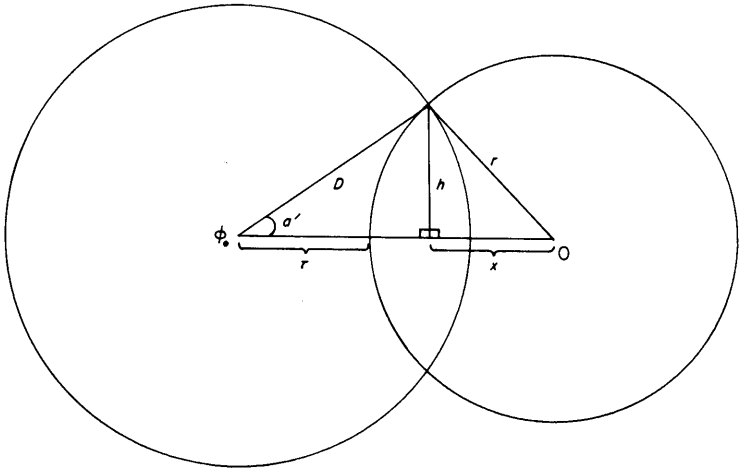

FIG. A1. Geometry of the derivation of a'.

If h and x are the legs of the right triangle with hypotenuse r, and a' is the new angle to which the area function is integrated, then:

$$h = D \sin(a'),$$

$$x = r + \tau - D\cos(a').$$

Substituting these equations into the identity $x^2 + h^2 = r^2$ and solving for $\cos(a')$ yields eqn (7).

APPENDIX B

The Effects of D, r and n on f

Equations (4) and (5) can be derived by noting that the terms D and r enter into the equation for f [eqn (3)] only through the term a, which is the limit of integration. Thus, for evaluating the effect of the distance jumped (D) as given in eqn (4):

$$\frac{df}{dD} = \frac{df}{da}\frac{da}{dD} = f(a) - \left[1 - \left[\frac{D}{2r}\right]^2\right]^{-0.5}. \tag{A.3}$$

The same procedure can be followed to derive eqn (5). For both of these cases, it is useful to note that:

$$\sin^n(x) = [1 - \cos^2(x)]^{n/2}.$$

The partial derivative of eqn (3) with respect to n involves even more unsolvable integrals and is difficult to interpret. We can, however, address essentially the same question by noting that:

$$\int \sin^n(x)\, dx = \frac{n-1}{n}\int \sin^{n-2}(x)\, dx - \frac{\sin^{n-1}(x)\cos(x)}{n}. \tag{A.4}$$

This will allow us to represent the term $A(n+2)$ [eqn (6)] in terms of $A(n)$. This can be done by defining;

$$A(n+2) = 2\int_0^{\pi/2} \sin^n(x)\, dx = \sqrt{\pi}\,\frac{\Gamma\left[\frac{n+1}{2}\right]}{\Gamma\left[\frac{n}{2}+1\right]}, \tag{A.5}$$

(Beyer, 1981).

Similarly we can write $A(n)$ as:

$$A(n) = \sqrt{\pi}\,\frac{\Gamma\left[\frac{n-1}{2}\right]}{\Gamma\left[\frac{n}{2}\right]}. \tag{A.6}$$

Combining eqns (A.5) and (A.6), and using the fact that $\Gamma(x+1)=x\Gamma(x)$, yields:

$$A(n+2)=\frac{n-1}{n}A(n). \tag{A.7}$$

We can now write $f(n+2)$ as a function of $f(n)$ by first using (A.4) above:

$$f(n+2)=\frac{\int_0^a \sin^n(\theta)\,d\theta}{A(n+2)}$$

$$=\frac{1}{A(n+2)}\left[\frac{n-1}{n}\int_0^a \sin^{n-2}(\theta)\,d\theta-\frac{\sin^{n-1}(a)\cos(a)}{n}\right]. \tag{A.8}$$

Substituting for $A(n+2)$ [eqn (A.7)] and [eqn (1)] in the right hand side of this equation yields:

$$f(n+2)=f(n)-\frac{\left[1-\left[\frac{D}{2r}\right]^2\right]^{(n-1)/2}\frac{D}{2r}}{(n-1)A(n)}. \tag{A.9}$$

Note that $f(n+2)$ is always less than $f(n)$ for $n>1$ and $D>2r$.

APPENDIX C

Derivation of eqn (9)

I will assume that portion of the sphere centered at Φ_0 and lying inside the surface of equal fitness is simply connected. Also, assume that the curvatures along the lines i and j in Fig. 7 are roughly constant over the distance D. Then, locally, these lines approximate segments of circles with radii $1/k(i)$ and $1/j(j)$ (these are the "osculating" circles, see Spivak, 1979).

We now divide the sphere up into "sections" as indicated in Fig. A2. The key is to note that, in terms of the proportion which lies inside a particular region, these sections behave like the entire sphere. This can be seen by the fact that the area of any of these sections out to an angle a is simply the area of the sphere out to that angle [as calculated in eqn (A.2)] multiplied by $d\delta/2\pi$. This quantity drops out when we calculate the ratio just as the g terms dropped out in the derivation of eqn (3).

If the angle a changes as we vary the angle δ, then the total proportion of the surface of the sphere which lies inside the surface of equal fitness is given by;

$$f=\frac{1}{2\pi}\int_0^{2\pi} f(a_\delta)\,d\delta. \tag{A.10}$$

If we define α as the intersection of the sphere centered at Φ_0 and the surface I [for an n-dimensional space, α is then an $(n-2)$-dimensional surface], with points on α defined by spherical co-ordinates centered at Φ_0, then we are simply integrating

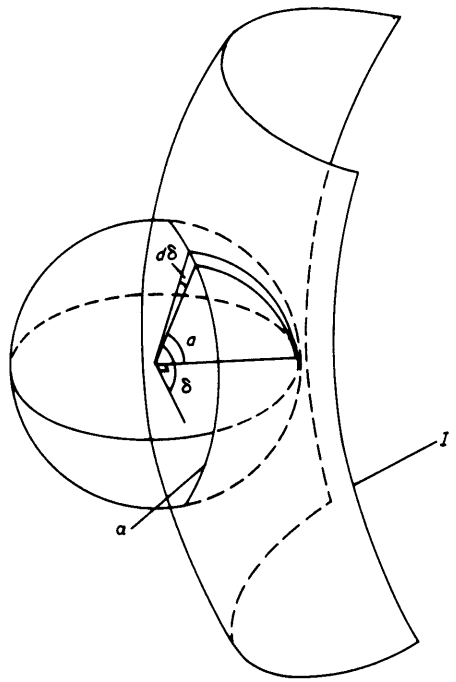

FIG. A2. Method for dividing the surface of a one-mutant sphere into sections (one of which is shown as a hatched region). Integrating over the angle δ is equivalent, in the n-dimensional case, to integrating over the space formed by the intersection of the sphere and the surface I (denoted by α).

f around α and dividing this quantity by the housdorff measure of an $(n-2)$-dimensional unit sphere. Thus, if the surface I curves differently in many directions,

$$f=\frac{1}{H(n-2)}\int_\alpha f, \tag{A.11}$$

where $G(\alpha)$ is the area of α.

Equation (A.11) basically states that if the angle a is changing as we move around the intersection of the two surfaces, then the probability of increasing fitness (f) will be equal to the mean value of f, averaged over δ [or, $a(\delta)$ in the example].

In any particular direction, $a(\delta)$ will be given by

$$a(\delta)=\text{Arccos}\,[Dk(\delta)/2]. \tag{A.12}$$

This follows from eqn (1) and the fact that $k=1/r$, where r is the radius of the "osculating" circle (Spivak, 1979) along the direction in which k is measured. Think of this as the circle that best fits into the surface along this direction.

The consequence of all this is that if a varies as we move around the intersection of the two surfaces, then the overall value of f will be the same as if the region in which mutants can increase when rare (bounded by I) were a sphere with the radius varying over time. This could be thought of as a case in which the distance to some "adaptive peak" is changing.

To see how eqn (9) arises, note that the curves of f vs. r in Fig. 3 become relatively linear over small stretches as the value of n gets large. Thus, if $\bar{r}$ is the arithmetic mean value of r over time, then the average value of $f(r)$ will be approximately given by $f(\bar{r})$. Since $k = 1/r$, the effective value of k will be $1/\bar{r}$. This is simply the harmonic mean of k.

APPENDIX D

Subsets with Fractal Dimension

As described in Appendix A above, the dimension of the system (n) enters into the analysis when calculating areas on the surface of a sphere centered at Φ_0. This is accomplished by slicing up that $(n-1)$-sphere into a set of $(n-2)$-spheres, the area of each of these being proportional to its radius raised to the $(n-2)$ power. Non-integer values of n arise when these "slice" spheres have non-integer dimension.

If all axes are independent, and we scale them appropriately, then a sphere of integral dimension corresponds to a case in which phenotype is equally likely to shift in any of the possible directions. Intuitively, a non-integer dimension ought to represent a case in which the chances of jumping in some directions are higher than for others.

To construct such a sphere, we choose a set of polar co-ordinates $(D, \theta_1, \theta_2, \ldots, \theta_n)$ in which D represents radius. I shall consider here the case of a sphere of dimension (n) between 1 and 2, the extension to higher dimensions is straightforward. For a fixed value of D, points on a two-sphere can be represented by (θ_1, θ_2) with $\theta_1 \in [-\pi/2, \pi/2]$ and $\theta_2 \in [0, 2\pi)$. A sphere which behaves as expected by eqn (3) for $n = 1+\alpha$ corresponds to a case in which points fall differentially along θ_1 such that $1/x$ of the points fall in the range $(-1/x^{1/\alpha} \cdot \pi/2, 1/x^{1/\alpha} \cdot \pi/2)$. Thus, for $\alpha = 0{\cdot}5$, if we imagine throwing points into the space at random, 1/2 of the time points fall within 1/4 of the range of θ_1, 1/3 of the time they fall within 1/9 of the range, and so on.

Construct a $(1+\alpha)$-sphere as follows: divide up the range of θ_1 from 0 to $\pi/2$ into m pieces, each corresponding to an ark of $\pi/2m$, as shown in Fig. A3. (The construction is the same for the range $-\pi/2$ to 0). If points fall differentially along θ_1 as described above, the segment (s_1) bounded by $\theta_1 = 0$ will contain $1/m^\alpha$ of the points. (Thus, if $m = 4$ and $\alpha = 0{\cdot}5$, 1/2 of the points fall in s_1), segment s_2 will contain $[(2/m)^\alpha - 1/m^\alpha]$, and in general, segment s_i will have point density μ_i given by:

$$\mu_i = (i/m)^\alpha - [(i-1)/m]^\alpha.$$

This is still a two-dimensional space but with density of ∞ along the line $\theta_1 = 0$ as m goes to infinity (for a discussion of density, see Falconer, 1985). To remedy this, divide each μ_i by $\mu_i m$. The measure of each segment is now:

$$\mu_i' = \frac{1}{m}[i^\alpha - (i-1)^\alpha] \tag{A.13}$$

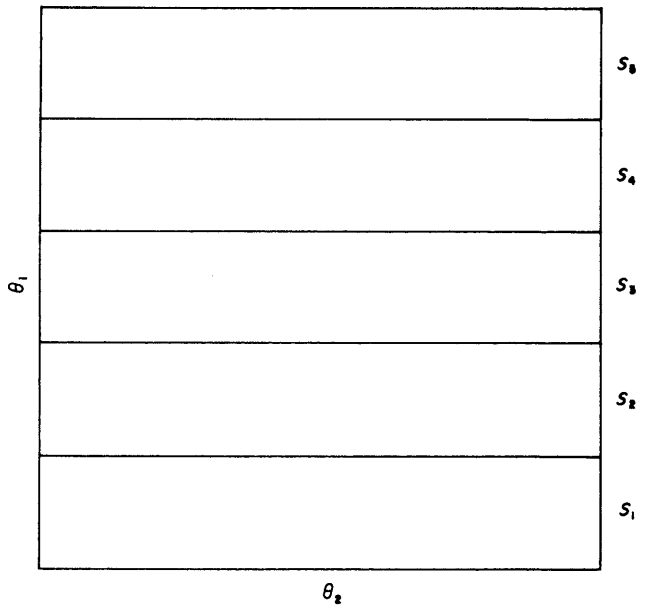

FIG. A3. The range $[0, \pi/2]$ of θ_1 is divided into m segments. Here, $M = 5$.

We can now build a $(1+\alpha)$-sphere by dividing the ark into m pieces but only filling in a portion of each piece, each portion being defined by eqn (A.13) above. This process is illustrated in Fig. A4. Note that the first segment will always be filled in completely. Thus, letting $m \to \infty$, as $\alpha \to 0$ the set behaves like a one-sphere. Adding up all m of these yields a measure, μ_T, for the whole range $(0, \pi/2)$ of:

$$\mu_T = \frac{m^\alpha}{m}. \tag{A.14}$$

Note that this goes to 0 as $m \to \infty$.

To show that, as $m \to \infty$ the set constructed above does in fact have Hausdorff dimension $1+\alpha$, cover it with squares of side $1/m$. This requires dividing the θ_2 axis into m sections but only requires m^α partitions along the θ_1 axis. Thus, as m increases, the number of squares needed to cover the entire set goes as $m^{(1+\alpha)}$. The Hausdorff dimension of the set is then the supremum of n such that;

$$\lim_{m\to\infty}\left[\frac{m^{1+\alpha}}{m^n}\right] = 0$$

(Morgan, 1988). The entire set therefore has $(1+\alpha)$-dimensional Hausdorff measure one and two-dimensional measure 0. Figure A4 shows the sequence of constructing a 1·5-sphere for increasing values of m.

Such a set constitutes a $(1+\alpha)$-dimensional sphere which behaves as eqn (3) predicts for $n = 1+\alpha$. If the distance jumped (D) is a random variable with its own probability distribution, then a system with the sort of fractal structure that I have

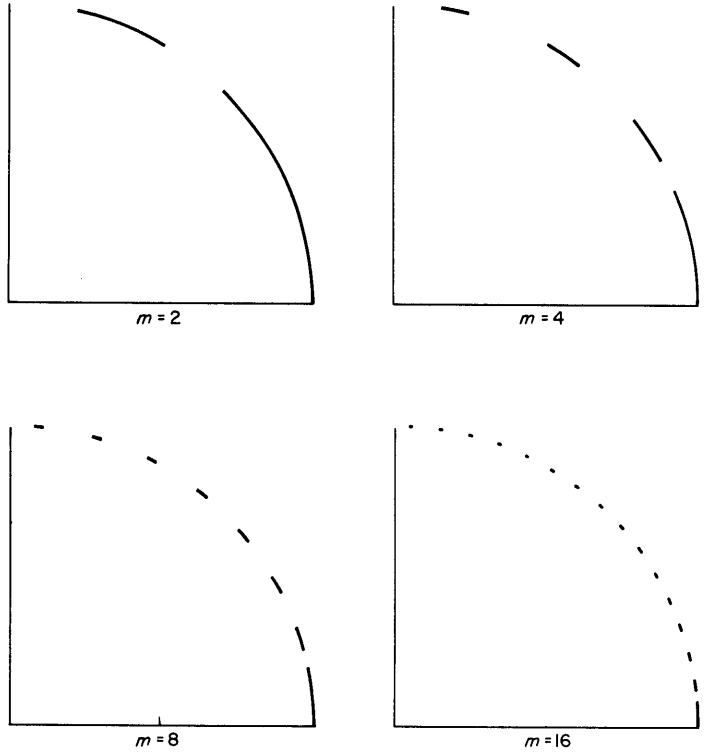

FIG. A4. Sequence in the construction of a fractal sphere. Only one quadrant is shown. As $m \to \infty$ this process produces a sphere of Hausdorff dimension 1/2. Note that for $\alpha = 1$ this process yields a circle (a one-sphere) and for $\alpha = 0$ it produces a 0-sphere (two points, equidistant from the origin).

described will produce a cloud of points which appear "compressed" along some set of axes. An example, for $n = 1{\cdot}5$, is given in Fig. A5. Note that such a system cannot accurately be described by a multivariate probability density function. It does, however, produce a scatter which looks quite similar to that produced by a multivariate distribution and which, depending on how axes are oriented, could be thought of as representing a "correlation" between characters.

The dimension of a one-mutant subset could be estimated from data by using the fact that, for each axis, 1/2 of the points should lie in the range $(-1/2^{1/\alpha} \cdot \pi/2, 1/2^{1/\alpha} \cdot \pi/2)$, where α is the contribution of that axis to the total dimension of the subset. An initial set of statistically independent axes could be obtained as the eigenvectors of a covariance matrix, as is done in principal components analysis. The points themselves would have to be obtained experimentally by mechanically altering the character in ontogeny and then observing the resulting adult phenotypes. Alternately, one could use information from somatic mutations which are expressed at a known stage in development.

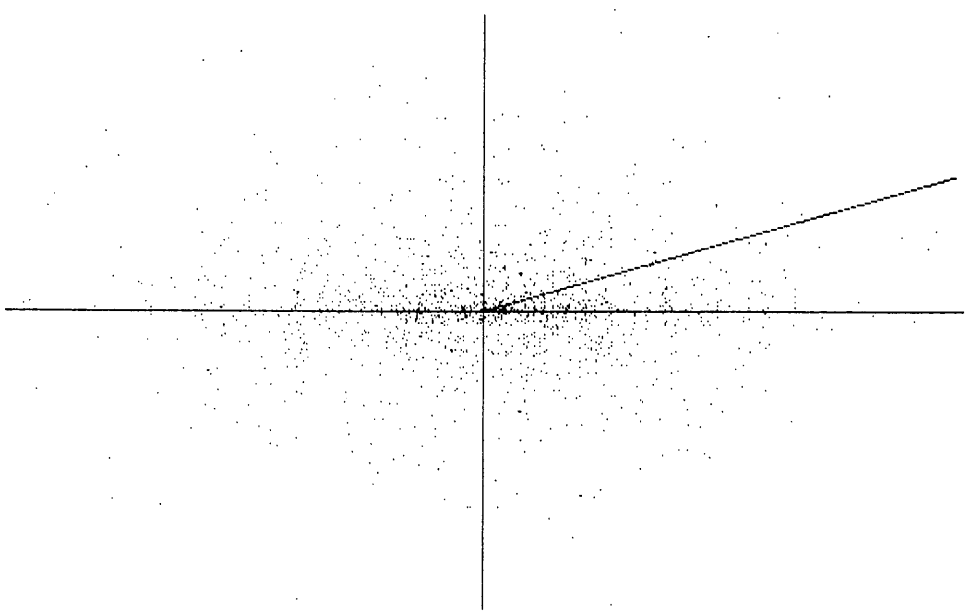

FIG. A5. A scatter of points in a space of dimension 1·5. This was produced by letting D (distance from the origin) vary according to a normal distribution and setting $m = 600$. Here, $\alpha = 1{\cdot}5$. The line in the upper right quadrant divides the points in that quadrant in half.