Size and Shape in Ontogeny and Phylogeny

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Pere Alberch, Stephen Jay Gould, George F. Oster, and David B. Wake*

Abstract.—We present a quantitative method for describing how heterochronic changes in ontogeny relate to phyletic trends. This is a step towards creating a unified view of developmental biology and evolutionary ecology in the study of morphological evolution. Using this representation, we obtain a greatly simplified and logical scheme of classification. We believe that this scheme will be particularly useful in studying the data of paleontology and comparative morphology and in the analysis of processes leading to adaptive radiation. We illustrate this scheme by examples drawn from the literature and our own work.

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Introduction

Since the collapse of Haeckel's "Biogenetic law," the way in which ontogeny is, or is not, related to phylogeny has been a continuing source of confusion and frustration to evolutionists. Much of this confusion stems from the plethora of definitions put forth by various authors in their attempts to classify the patterns they perceived both in the fossil record and among living species. Among these patterns two have received special attention from evolutionary theorists. First is the fact that some organisms, during their ontogeny, seem to "recapitulate" the adult stages of their ancestors. Conversely, adults of descendant species can resemble juvenile stages of the ancestor, a phenomenon called paedomorphosis; historically, it presented a major challenge to the universality of Haeckel's biogenetic law. Recently, Gould (1977) provided a historical evaluation of the subject, along with some concrete proposals for reducing the burden of vocabulary that has rendered the field so forbidding. In this paper we elaborate, in more quantitative terms, the "clock model" put forth by Gould. In so doing we hope to bring more order and logical structure to the subject and to clarify how heterochronic disturbances during development can result in particular phylogenetic sequences. Gould introduced size, "shape" and age as independent variables in order to dissect the processes underlying the observed phenomena of recapitulation and paedomorphosis. His "model" was essentially static. In order to make this approach more dynamic, we have added several features, including the specific incorporation of growth laws and differentiation events. We have attempted to cull from the literature and from our own work examples to show how the formalism we propose can be used to relate changes in size and shape during ontogeny and phylogeny. Finally, we discuss how adaptations in size and shape can be incorporated into the framework of existing theory in population ecology.

We emphasize that the formalism developed below is not really a "model" in the strict sense. Rather, we have attempted to structure the existing ideas on evolution of size and shape into a conceptual framework which is easily visualized and can be discussed in quantitative terms.

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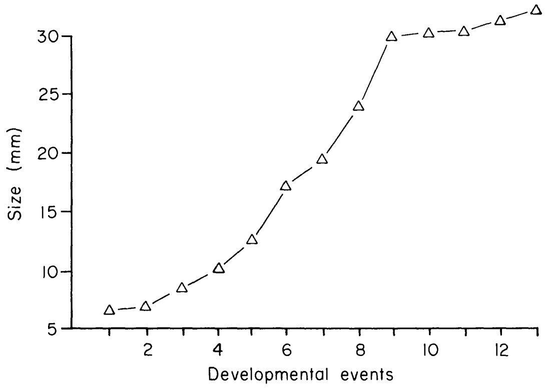


FIGURE 1. Developmental events in the urodele *Triturus vulgaris* as a function of size (data from Erdmann 1933). Ossification of cranial elements occurs over an extended period of time, with bones appearing sequentially as animals grow. The first appearance of a bone is recorded as a developmental event, as follows: 1. splenial, vomer, palatine; 2. dentary; 3. pterygoid, squamosal, premaxillary; 4. prearticular; 5. frontal; 6. parietal; 7. opisthotic; 8. first basibranchial, orbitosphenoid; 9. prootic; 10. quadrate; 11. nasal; 12. maxilla, parasphenoid; 13. prefrontal.

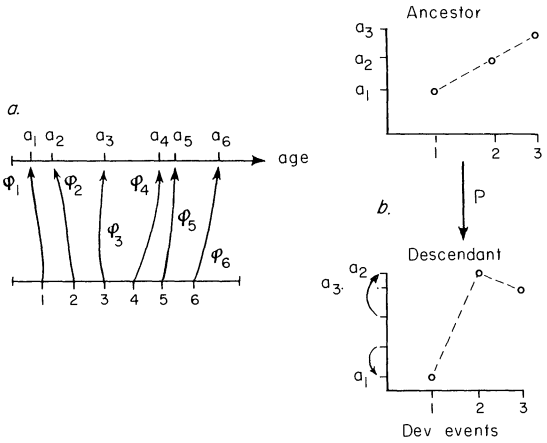


FIGURE 2. (a) The mappings, ϕ_i, of developmental events onto the age axis. (b) The sequence and timing of developmental events are altered during the course of phylogeny, P.

We hope that our attempts to construct a quantitative theory will stimulate others to delve more deeply below the level of pure phenomenology and come to grips with the central issue underlying evolutionary diversification of size and shape—that is, the morphogenetic unfolding of genetic programs in ontogeny and their alteration in the course of phyletic evolution.

1. *Describing ontogeny.*—A. *Developmental events.*—In the classical literature on comparative embryology the ontogeny of an organism is divided into discrete stages, each characterized by the onset of particular developmental phenomena, such as the mineralization of a matrix or a cellular arrangement initiating a new organ.

Let us denote by $d_1, d_2, \ldots, d_i$, a sequence of differentiation processes. For convenience we will let each d_i refer to both the process itself and to a discrete stage in each process for the structure or organism under consideration: the appearance of the first differentiated cell in a series of structures, the first indication of mineralization in each of a series of skeletal elements, or the first observable stage in a series of developmental processes.

Each d_i is thus a developmental event. For example, in Figure 1 we have plotted the sequence of appearance of calcification in the different skull bones of a newt, *Triturus vulgaris,* against the size at which each developmental event was observed. Each developmental event signals a developmental process having an age interval $(a_i, a_i + \Delta a_i) = (\alpha_i, \beta_i)$ which specifies the time of onset (α_i) and of cessation (β_i). That is, the structure appears at age α_i and grows until $\beta_i = a_i + \Delta a_i$.

In Figure 2a we have plotted the developmental events and their corresponding age intervals; here we have denoted by $\phi(\cdot)$ the function which assigns the appropriate age interval to each d_i (i.e. $\phi_i{:}d_i\ [\alpha_i, \beta_i] = \phi_i(d_i)$). Each event can be shifted during phylogeny. Evolutionary changes in developmental events can produce features that might be eliminated from the paedomorphic descendant while in other descendants the sequence can be altered (e.g. reversed) (Figure 2b).

Figure 3 is an example illustrating how the sequence of developmental events can be altered in phylogeny. A Guatemalan urodele, *Bolitoglossa rostrata,* is taken as a prototypical ancestor with normal, well developed phalanges (Figure 4). In Figure 3 the sequence of appearance of the first distinguishable mineralization in the metatarsal and three phalanges of the fourth digit is plotted against the size at which mineralization is first observed. The ontogeny of two derived species is also plotted in Figure

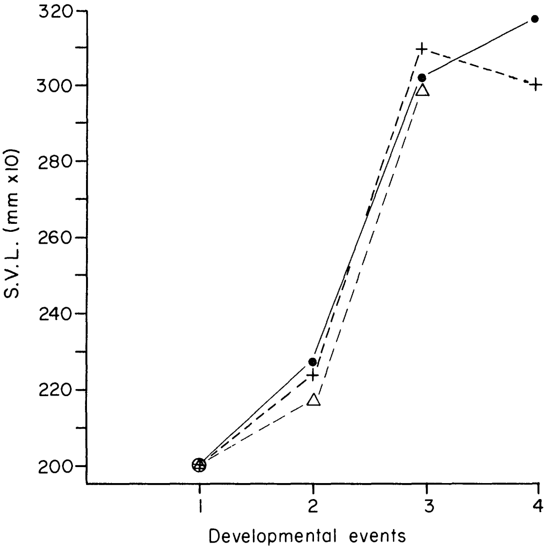


FIGURE 3. Developmental events in the feet of tropical salamanders as a function of size (S.V.L. is distance from tip of snout to posterior angle of vent). Data from Alberch (unpublished). The sequence of ossification in the fourth digit of the hind foot of three species is illustrated. Developmental events, based on *Bolitoglossa rostrata* (●) (see Figure 4), are: 1. metatarsal 4; 2. phalanx 1 (most proximal); 3. phalanx 2; 4. phalanx 3. In *B. helmrichi* (+) developmental events 3 and 4 are reversed. In *B. rufescens* (Δ) developmental events parallel those of *B. rostrata*, but development ceases before event 4 occurs.

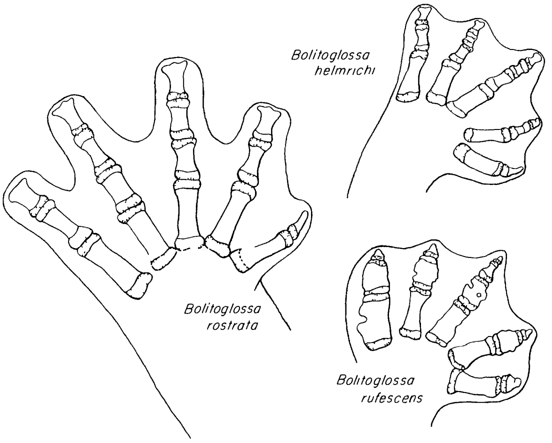


FIGURE 4. Left feet of three species of tropical salamanders, drawn to the same scale from cleared and stained preparations. Cartilage is stippled. *Bolitoglossa rostrata* has the most generalized foot, and *B. rufescens* is relatively paedomorphic. *B. helmrichi* has a reversal of a developmental event in digit 4 (cf. Figure 3).

3. *B. rufescens,* a paedomorphic species (Wake and Brame 1969), has its development truncated relative to *B. rostrata* and its third phalanx fails to mineralize. Note that the preceding three developmental events occurred in the same sequence and at the same relative time (assuming an approximately constant relation between size and age) as in the "ancestral" form. The other species plotted, *B. helmrichi,* has had the timing of mineralization of the second and third phalanges reversed, producing a consequent change in the proportions of these bony elements. The overall changes in the final shape of the structure are striking (Figure 4).

The importance of sequence in developmental events cannot be overemphasized. In salamanders the order of appearance of anterior cranial elements during ontogeny varies from group to group (Figure 5). The maxillary bones appear early in the sequence of developmental events in some groups of salamanders (e.g. ambystomatids) but late in others (e.g. salamandrids, plethodontids). Different groups of salamanders are paedomorphic as a result of achieving *sexual* maturity before all of the developmental sequence of cranial elements has taken place. If derived from ancestors in which maxillary bones develop early, the heads are relatively broad, for those bones are the most lateral elements in the anterior region of the skull (for example, *Cryptobranchus*). In contrast, when maxillary development is shifted to a later position in the sequence of developmental events, the bones do not appear in paedomorphs and the heads are relatively narrow (*Necturus, Proteus, Siren*) (Figure 6).

B. *Shape and size.*—We shall restrict our attention to those systems whose functions are characterized by their morphological appearance. For example, we diagnose reproductive maturity by the size and shape of the reproductive organs, although we recognize that the actual event of reproductive maturity may correspond to, say, the genetically controlled differentiation of gonadotropin and the corresponding hormonal signal acting on organs that may have already reached their adult proportions. For example, in a series of elegant experiments, Kallman and Schreibman (1973) have shown that the remarkable intraspecific variation in size among adult males of platyfish (*Xiphophorus maculatus*) results from differences in timing in the onset of sexual maturity (Figure

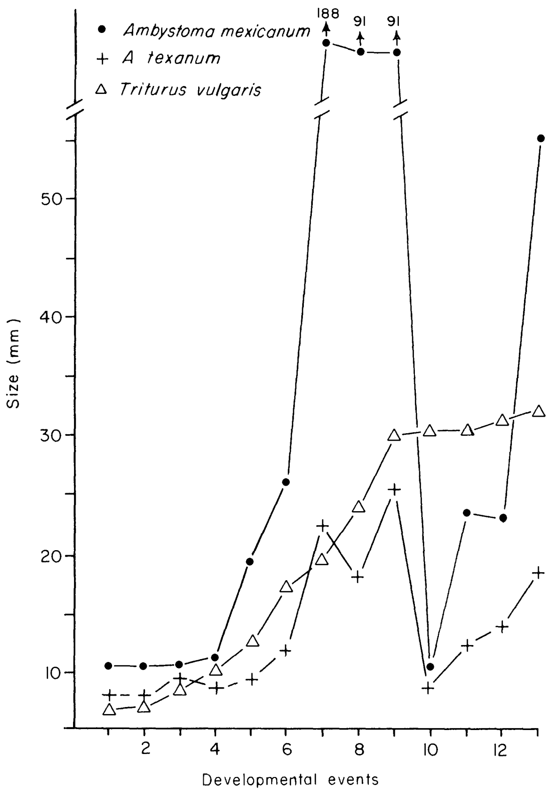


FIGURE 5. Developmental events as a function of size in three species of urodeles. The base is *Triturus vulgaris* (Δ) (see Figure 1), and the other species are compared to it. *Ambystoma texanum* (+) closely parallels *Triturus* for a part of its development, but, for example, the opisthotic bone (developmental event 7) appears relatively late and the quadrate (10) appears early (data from Bonebrake and Brandon 1971). *A. mexicanum* (●), a larger species than either of the others, differs greatly in the delay of developmental events 7, 8, and 9, but it resembles *A. texanum* in the early appearance of the quadrate (10) (data from Keller 1946). Note that maxillary bones (12) appear at an earlier stage of development in the species of *Ambystoma* than in *Triturus*.

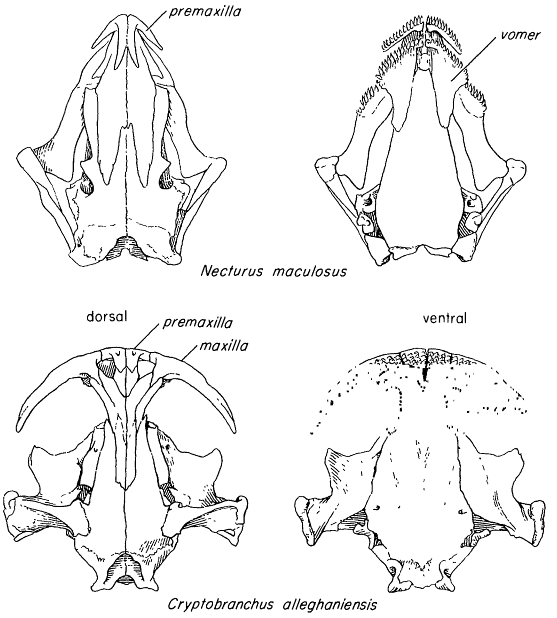


FIGURE 6. When developmental events are shuffled so that a given event is delayed and eventually eliminated from an ontogeny, a major shape change can occur. An example is the very different skull shapes of two urodeles. In the paedomorphic *Necturus* maxillary bones do not develop, and the skull (and consequently, the head) have very different shapes than in the less paedomorphic *Cryptobranchus*.

7). They identified a sex-linked gene controlling timing of differentiation of the gonadotropic zone of the adenohypophysis. Physiological activity is accordingly accelerated or retarded.

The problem of how to quantify the size and shape of geometrically complex organs has an extensive literature (see Bookstein 1976, 1978 and Lestrel 1974 for recent reviews). Most of the methods proposed for describing size and shape ultimately reduce to specifying a set of parameters $X_i, \ldots, X_n$. These parameters may represent certain measurements of dimensions, or a set of Fourier components of the function describing the shape of the structure. Often, a "size" variable X_1 is selected, such as the volume of a structure or its area. The remaining variables are rendered non-dimensional by normalizing by X_1 to produce a set of "shape" coordinates $\sigma_1 = \frac{X_i}{X_1}$. Thus the geometrical appearance of a structure can be specified by a set of sizes, $S_1, \ldots S_K$, with dimensions of length, area or volume, and a set of non-dimensional shape parameters, $\underset{\sim}{\sigma} = (\sigma_1, \ldots, \sigma_n)$, which give the relative proportions of the structure. Therefore, we can represent the morphology of an organ by a point $(\underset{\sim}{S}, \underset{\sim}{\sigma}) = ((S_1, \ldots, S_n), (\sigma_1, \ldots, \sigma_m))$ in "size-shape" space as shown in figure 8. For pictorial purposes we consider but a single size and shape coordinate, however, the picture remains the same no matter how many coordinates are required to specify the system.

C. *Ontogenetic trajectories.*—Using the notion of size-shape described above we can follow the growth of a system from its inception to its mature form. This is shown schematically in

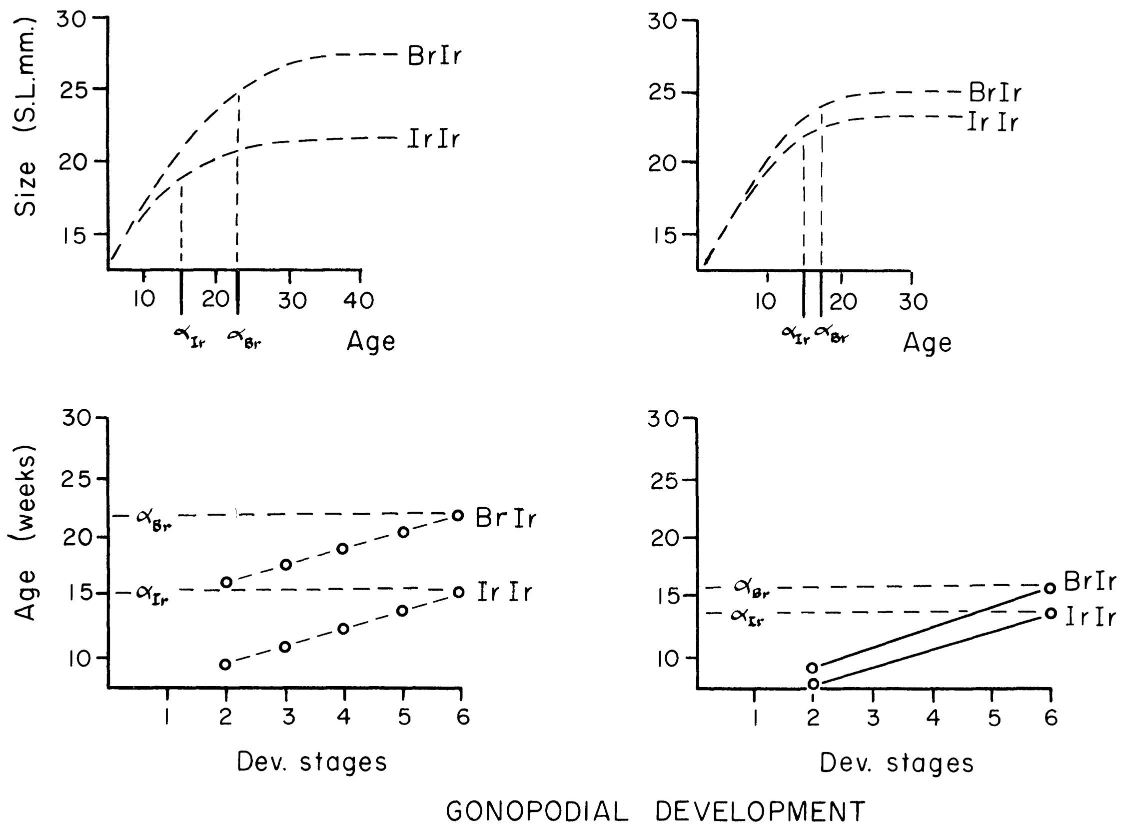


FIGURE 7. Growth rate curves of male *Xiphophorus maculatus* of different genotypes, *IrIr* and *BrIr*. The average timing of the stages of gonopodial development is plotted below. Two sets of data, with individuals from different strains, are shown to illustrate the correlation between rate of gonopodial development, i.e., attainment of sexual maturity, and differences in adult size. Data from Kallman and Schreibman (1973).

Figure 9, with axes for size (S), shape (σ), and age (a). The differentiation event initiating the growth phase occurs at age α. The size and shape changes trace out a trajectory, $\underset{\sim}{X}(t) = (a,S,\sigma)$. We shall call this the "ontogenetic trajectory." It is a complete record of the physical appearance of the system. In Figure 10a we have plotted the projection of the ontogenetic trajectory onto the (S,σ) plane for the growth of *Poecilozonites bermudensis zonatus*. The data for this plot are taken from Gould (1977, pp. 253–254). In many (but not all) cases the system ultimately reaches a final, or adult, size $\underset{\sim}{X}_A$, whereafter little or no morphological change takes place.

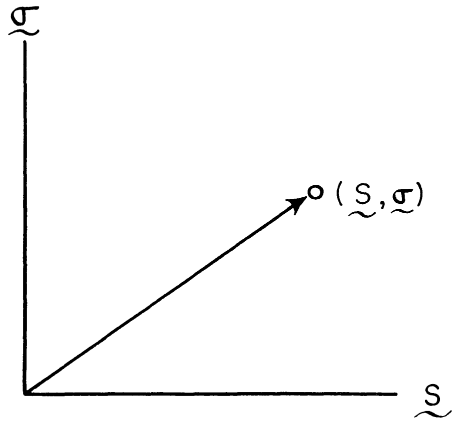


FIGURE 8. The size and shape of an organism can, in principle, be represented by a collection of size measurements, $\underset{\sim}{S}$, and shape measurements, $\underset{\sim}{\sigma}$, which we visualize as a point ($\underset{\sim}{S},\underset{\sim}{\sigma}$) in "size-shape space."

The ontogenetic trajectory is an idealization which refers to the growth of a single individual in a population. In principle, if enough size and shape coordinates are included, we can describe the ontogeny of an entire individual. In practice, however, we can only trace one, or a few, body parts at a time. A different individual, even an identical twin, will have a somewhat different trajectory, because a multitude of influences intervene during development. A pop-

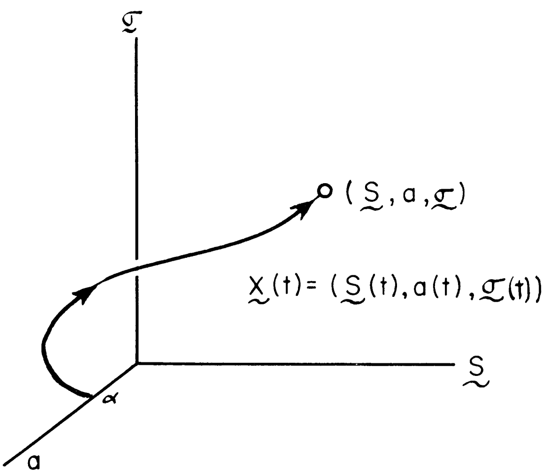


FIGURE 9. As an organism ages (from conception), the form coordinates, i.e. size and shape, of any structure or organ trace out a trajectory, $\underset{\sim}{X}(t)$ in "age-size-shape space." We call $\underset{\sim}{X}(t)$ the "ontogenetic trajectory."

ulation can be viewed as a "cloud" of points, each tracing its own ontogenetic trajectory. Later we shall discuss how selection acts to shift this cloud in size-shape space to create a phylogenetic sequence.

2. *Models for ontogenetic trajectories.*—Our next task is to formulate the growth laws underlying the ontogenetic trajectory. We can visualize a growth law as the "force field" propelling the point $\underset{\sim}{X}(t)$ through size-shape space, tracing out the ontogenetic path. That is, the trajectory is generated by the dynamical system

$$\frac{da}{dt} = 1 \text{ (i.e. age and time advance at the same rate)}$$

$$\frac{dS}{dt} = G(\cdot) = \text{growth rate in size}$$

$$\frac{d\sigma}{dt} = g(\cdot) = \text{rate of change in shape}$$

The solution to these differential equations is the ontogenetic trajectory.

The cellular processes underlying $G(\cdot)$ and $g(\cdot)$ involve cell proliferation and rearrangement. However, at the level of description we can treat the processes phenomenologically. Let us denote by $y(t)$ *either* size, $S(t)$, *or* shape, $\sigma(t)$, since our discussion will apply to both. Then, a general growth law will have the form

$$\frac{dy}{da} = f(y \mid \alpha 3, \beta, k_y, S_0) \qquad (*)$$

where α = age of onset of growth and k_y = rate of change in size and shape, are growth rate parameters. β is an "off-signal" which ter-

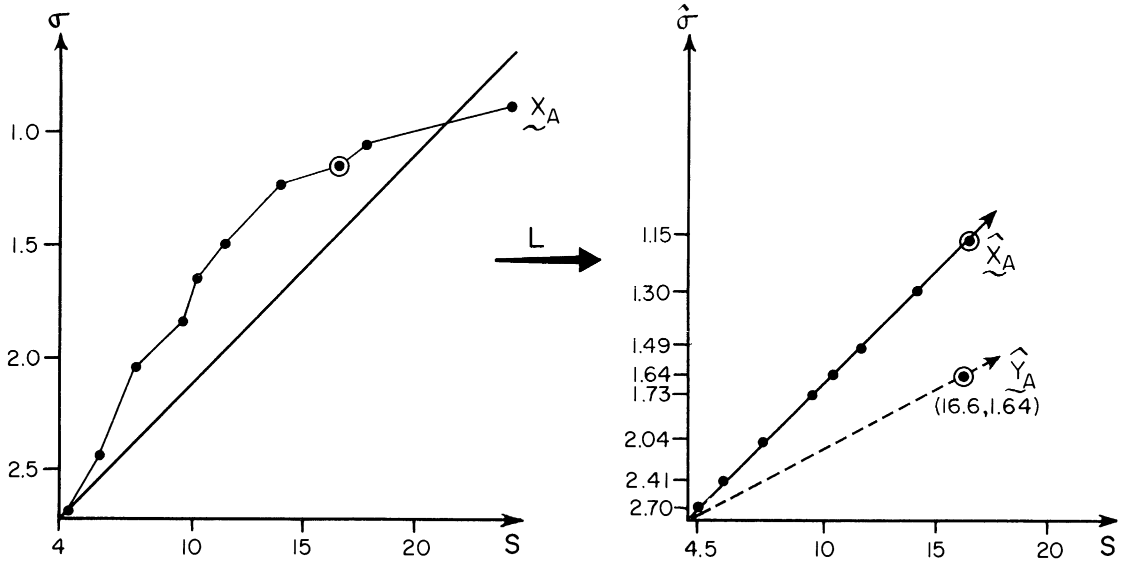


FIGURE 10. (a) The projection of the ontogenetic trajectory and the size-shape plane for the growth of the land snail *Poecilozonites bermudensis zonatus*. Here S = width + height, σ = width/height (from Gould 1977). (b) By changing the shape scale the ancestral trajectory $\underset{\sim}{X}$, can be linearized. Also shown is the adult shape of the descendant, $\underset{\sim}{Y}_A$, along with an *assumed* linear ontogenetic trajectory.

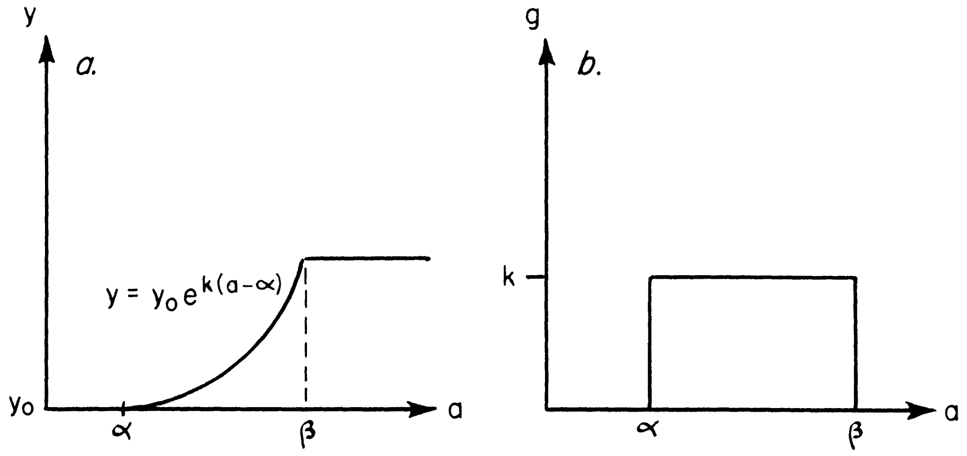


FIGURE 11. (a) The exponential growth law for an organ given by equation (1). (b) The form of the growth function $g(y \mid \alpha,\beta,k)$.

minates growth and S_0 is the initial size at age α of the system. β can correspond either to a particular age, or to the terminal size of the growing structure. The simplest of all growth laws (Figure 11a) is

$$\frac{dy}{da} = \begin{cases} 0, a < \alpha \\ ky,\ \alpha < a < \beta \\ 0, a > \beta \end{cases} \tag{1}$$

That is, y grows according to $y(a) = y_0e^{ka}$ between the onset age, α, and the offset age, β (Figure 11b).

There is a substantial literature dealing with so-called "allometric growth" which derives from growth laws of this form (see Bertalanffy 1960; Gould 1966; Laird et al. 1968 and Laird 1969 for reviews). If y_1 and y_2 refer to two size and/or shape parameters referring to body parts of the same individual, and if each grows according to equation (1) — but with growth rates k_1 and k_2, the y_1 and y_2 will be related by the "allometric law"

$$y_1(t) = \lambda y_2(t)^b \tag{2}$$

$$\text{where } b = \frac{k_1}{k_1}, \quad \text{and} \quad \lambda = \frac{y_1(0)}{y_2(0)}.$$

A second basic type of growth law is described by the growth equation

$$\frac{dy}{da} = \begin{cases} 0, a < \alpha \\ k(y_m - y),\ a > \alpha \end{cases} \tag{3}$$

This yields the "saturating" growth trajectory $y(t) = y_m(1 - e^{-k(a-\alpha)})$ shown in figure 12.

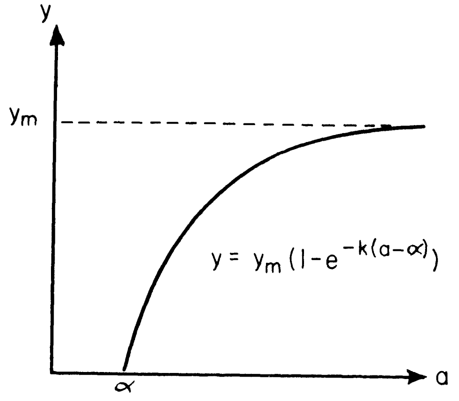


FIGURE 12. The saturating growth law given by equation (3).

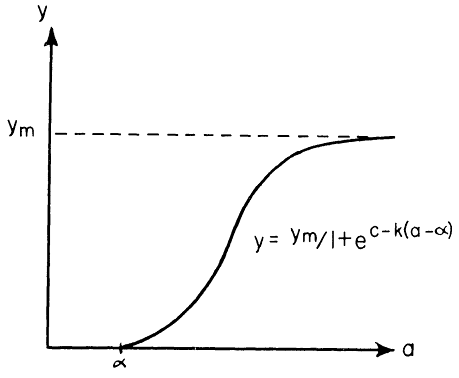


FIGURE 13. The sigmoidal growth law given by equation (4).

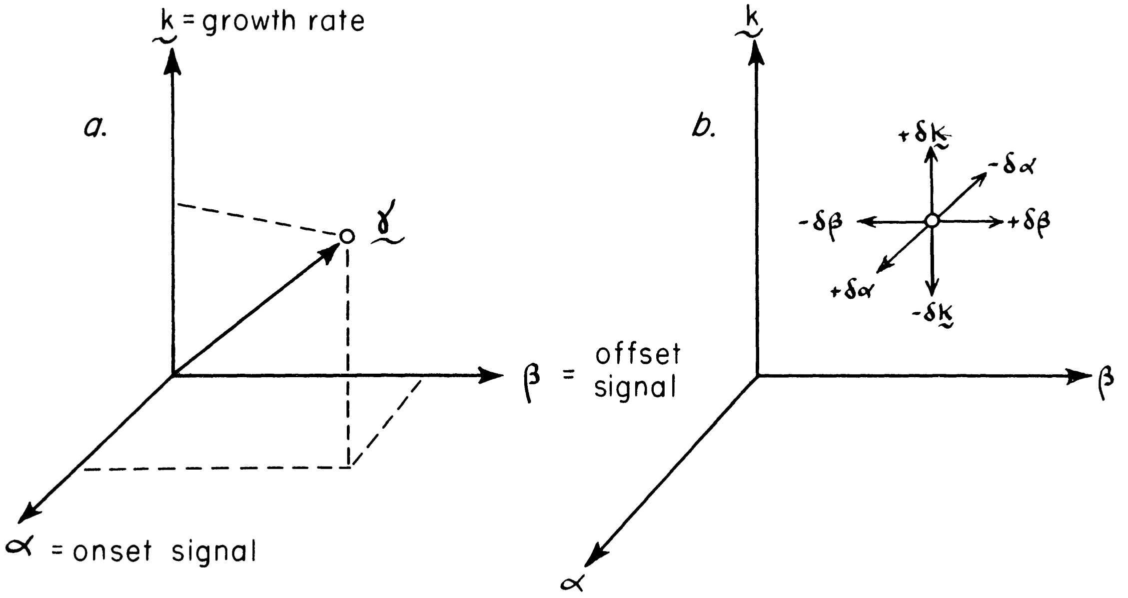


FIGURE 14. (a) The elementary growth laws are characterized by the parameters α = onset age, β = offset signal (either age or organ size), $\underset{\sim}{k}$ = growth rates (size and/or shape), which we visualize as a point $\gamma = (\alpha,\beta,\underset{\sim}{k})$ in 3 dimensional Euclidian space. (b) There are 8 "basic mutations" corresponding to positive and negative perturbations in each of the growth parameters. (Changes in S_0 have not been included.)

This is appropriate to the snail data in figure 10. According to this growth law, there is a negative feedback which damps growth as the system approaches its adult size or shape, y_m, and, in this case, the "off signal," β. By measuring growth as a fraction of $\hat{y}_m$, $y = (y_m - y)/y_m$, relative growth is also "allometric" (i.e. obeys equation (2) with $b = -\frac{k_1}{k_2}$).

A third type of growth is "sigmoidal," as is shown in figure 13. There are many growth models which yield this pattern; perhaps the simplest is the logistic:

$$\frac{dy}{da} = \begin{cases} 0, & a < \alpha \\ ky(1 - \frac{y}{y_m}), & a > \alpha \end{cases} \qquad (4)$$

This implies a growth law, $y(t) = y_m/(1 + e^{c-k(a-\alpha)})$, where $c = \ln\left(\frac{y_m - y_0}{y_0}\right)$.

After an initial period of exponential growth, growth slows down and asymptotically approaches its limiting value, y_m. By defining a new growth variable $y = \frac{y_m - y}{y}$, this growth law can also be rendered "allometric."

Another growth curve of sigmoid type is the Gompertz law, $\frac{dy}{dt} = -ay \ln \frac{y}{y_m}$. A change of coordinates, $u = \ln \frac{y}{y_m}$, results in exponential growth in u; thus the Gompertz curve is also "allometric" if measurements are taken in units of $\ln \frac{y}{y_m}$.

More general growth models can be constructed, many of which can be cast in "allometric" form. When this can be done it simplifies certain practical aspects of curve fitting and so forth; however, the formalism developed here can accomodate any empirically determined growth relation.

3. *A classification of heterochronic phenomena.*—The important feature common to all of the growth models we shall consider is that the development of the ontogenetic trajectory is regulated by the following set of "control parameters":

α = onset age of growth
β = offset signal for growth, either a specific age, or a limiting size or shape
k = growth rate during the period of growth

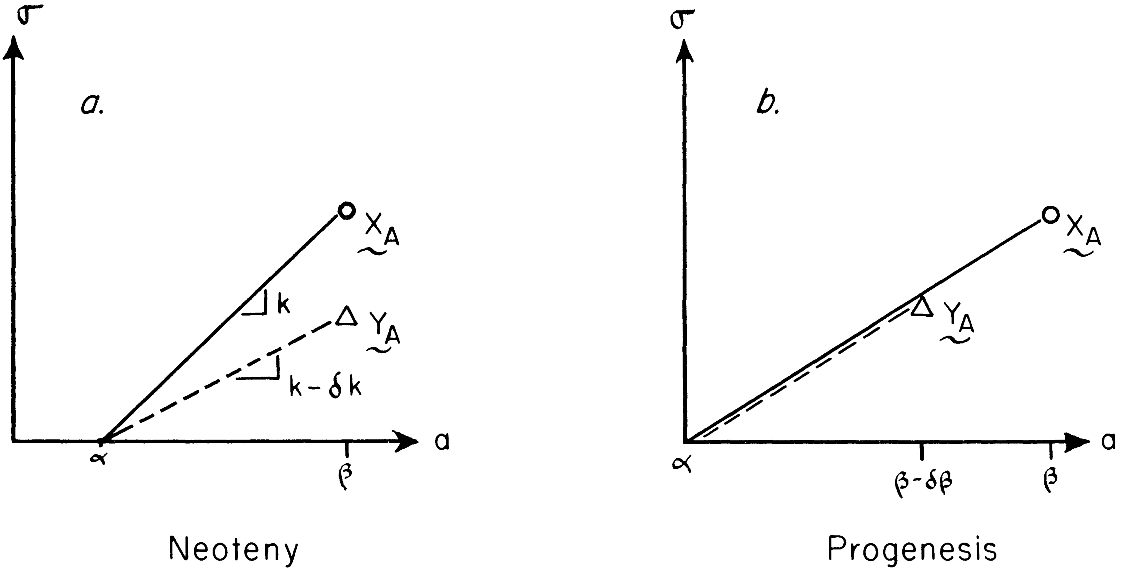


FIGURE 15. (a) A negative perturbation, $-\delta k$, in shape growth rate produces a paedomorphic descendant by the process of neoteny. (b) A decrement in β also produces a paedomorphic descendant by the process of progenesis.

S_0 = initial size at the commencement of the growth period.

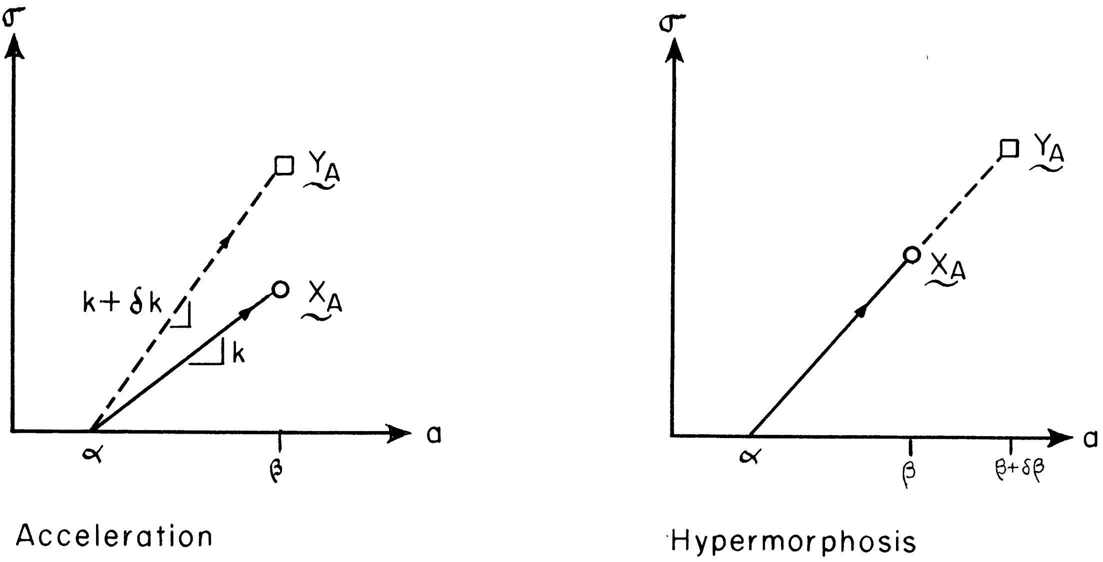
By convention, we shall assume that the initial shape $\sigma_0 = 0$.

It is useful to note that by an appropriate change of scale all the above growth models can be rendered linear, so that the ontogenetic trajectory is a straight line from ($a = \alpha$, $S = 0$, $\sigma = 0$) to the adult configuration, $\underset{\sim}{X}_A$. For the purpose of illustrating certain features of the model we shall frequently draw the ontogenetic trajectories as straight lines (Figure 10); this is not necessary, of course, and in practice it may not be possible, when real data do not conform exactly to any of the idealized growth models.

By restricting our attention to growth laws of the form $\frac{dy}{da} = g(y \mid \alpha, \beta, k_y, S_0)$ we can obtain a simple scheme of classification that summarizes much of the classical terminology. Since the ontogenetic trajectory generated by the growth law is completely defined (up to random effects which we are neglecting for the time being) by the parameters (α, β, k_y, S_0), we can visualize the state of the system as a point $\underset{\sim}{y} = (\alpha, \beta, k_y, S_0)$ as shown in figure 14a. Then there are but 10 possible "pure" perturbations which can take place ($\alpha \pm \delta\alpha$, $\beta \pm \delta\beta$, $k_S \pm \delta k_S$, $k_\sigma \pm \delta k_\sigma$, $S_0 \pm \delta S_0$) (figure 14b). If the perturbation $\delta \underset{\sim}{y}$ is small, then any possible variation can be expressed as a sum of these pure types.

Let us take advantage of the fact that most of the ontogenetic trajectories generated by elementary growth models can be linearized and plot the trajectory, $\underset{\sim}{X}$, for an ancestral form as a straight line with unit slope. Suppose that there is a "pure" perturbation whose only effect is to increase the size growth rate, $k_S \rightarrow k_S + \delta k_S$. This will produce an adult descendant with the same shape but of larger size. Conversely, a negative change, $k_S = k_S - \delta k_S$, will produce a descendant that has the same shape as the ancestor but smaller size. These processes are known in the literature as *proportioned gigantism* and *proportioned dwarfism*, respectively (Gould 1977, p. 259).

Similarly, along the k axis in Figure 14b, positive or negative changes in rate of change in shape, $k_\sigma \rightarrow k_\sigma \pm \delta k_\sigma$, can produce descendant phenotypes which either have a morphology like a juvenile ancestor or transcend the ancestral form, respectively (Figures 15a and 16a). In the latter case, when the descendant phenotype is speeded up with respect to the ancestor, the process is called *acceleration*. In the former case, the process of producing a paedomorphic descendant by retardation in k_σ is known as



Acceleration

Hypermorphosis

FIGURE 16. (a) A positive perturbation $+\delta k$ in shape growth rate produces a peramorphic descendant. This process is called acceleration. (b) A positive perturbation, $+\delta\beta$, in offset signal, also produces a peramorphic descendant by a process of *hypermorphosis*.

neoteny. A "pure" case of neoteny is found in *Poecilozonites bermudensis zonatus*. This Pleistocene Bermudian land snail grows with a series of characteristic allometries in form, color, and shell thickness (Gould 1968). Juveniles are thin shelled, triangular in apical shape, and colored with discontinuous bars. As growth proceeds, the shell becomes relatively thicker, domed in apical shape (by positive allometry of height vs. width), while the bars become more numerous and closely spaced, and finally fuse to bands. At least four times during the history of this taxon, probably as a response to lime-poor soils, neotenic populations evolved. They reached the same size and whorl number as their ancestors but remained thin shelled, apically triangular and colored with discontinuous bars. These retardations occurred at the same rate for this set of apparently disparate characters; the neotenic descendants are scaled up replicas of the juvenile stages of their ancestors. Since protoconchs of ancestors and adults of descendant species are the same size, we may conclude that no changes occurred in times of onset and cessation. In this case, paedomorphosis results from the general slowing down of developmental rates in shape, i.e. by neoteny. Figure 10b displays this example of neoteny for allometry of the spiral.

A paedomorphic descendant can also be produced by a mutation which decreases the growth period (or limiting signal), $\beta \rightarrow \beta - \delta\beta$, a process known as *progenesis*, as shown in Figure 15b. An example of progenesis has been illustrated in Figures 3 and 4, where *Bolitoglossa rufescens* is a progenetic species, with respect to the ancestral form. The opposite pure mutation, $\beta \rightarrow \beta + \delta\beta$, produces a descendant which transcends the ancestral form, a process called *hypermorphosis* (Figure 16b).

Incremental changes in the onset age, $\alpha \pm \delta\alpha$, we shall call *post* and *pre displacement*, respectively (c.f. Figure 17). These "perturbations" produce changes in adult morphology only if β, the offset signal, does not undergo a corresponding change. The phenomenon of displacement is extremely common in phylogenesis. Gould (1977, pp. 244–245) united the speeding up of change in shape and negative displacement, calling both *acceleration*, though distinguishing the two modes. Slowing of change in shape was joined with post displacement as *neoteny*. In each case, Gould united the two modes because they share the property of placing descendants beyond the ancestral adult ontogeny (or keeping them at an ancestral juvenile stage) at the preferred standard of common age for ancestor and descendant. The for-

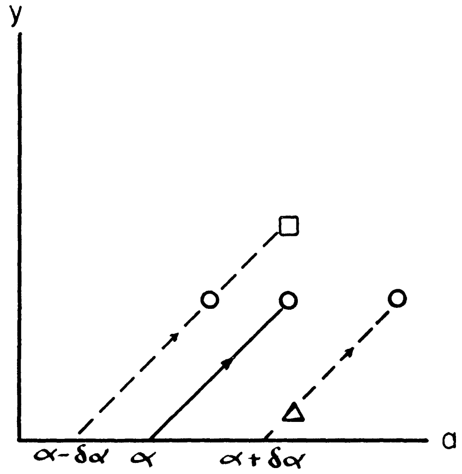


FIGURE 17. Changes in the age at which a structure commences its ontogeny may well result in a paedomorphic ($+\delta\alpha$) or a peramorphic ($-\delta\alpha$) descendant, so long as β and k remain unchanged. However, if the interval ($\beta-\alpha$) remains constant as well, adult shape is unchanged. These processes are termed post ($+\delta\alpha$) and pre ($-\delta\alpha$) displacement.

malism of this paper requires that the modes be separated. In addition, post and pre displacement can yield other kinds of results and may involve biological mechanisms different from those that yield speeding up and slowing down. Thus we restrict acceleration and neoteny to speeding and slowing in rate of change in shape, while we designate change in onset as post and pre displacement.

A good example of pure displacement producing a common pathology is encountered in the developmental basis of "creeper" fowl (chondrodystrophic forms with abnormally shortened limb bones). The creeper gene is an autosomal semi-dominant, lethal in homozygous form and producing creeper fowl in heterozygous form. Cock (1966) plotted tarsometatarsal length vs. body weight for creepers and normals in prenatal growth from seven days until hatching. Creepers and normals have the same slopes, but the y-intercept of creepers is significantly lower. Apparently, the tarsometatarsus of creepers differentiates later but then grows at the same rate as in normals. Since the endpoint (time of hatching) does not vary, creeper limbs end up short (paedomorphosis by post displacement) simply because they begin to grow later.

Another example has been presented recently by Bard (1977) who hypothesized that differences in striping patterns in zebras might result from displacement. He hypothesizes a single mechanism for generating vertical stripes in embryos. Depending on the embryo's stage of growth when this mechanism operates, head stripes in adults will be wider or narrower, branching patterns will differ, spots may appear, and a wide variety of patterns can arise. In fact, relatively few color patterns are found, and these can be explained by operation of this mechanism during the third (*Equus burchelli*), fourth (*E. zebra*) or fifth (*E. grevyi*) weeks of development.

The morphological effects of post and pre displacement can be even more profound, resulting in qualitative differences, including loss of bony elements. For example, Figure 5 illustrates how the timing of appearance of mineralized cranial elements differs in various species of urodeles. As the onset of appearance is delayed to later and later periods of development, the probability that such an element might be entirely eliminated from the ontogeny of a descendant is increased. Thus, the relatively late-appearing septomaxillary bones, characteristic of generalized salamanders, are absent in many of the highly derived plethodontids (Wake 1966) and are absent in the entire family Salamandridae.

Perturbations in initial conditions can be amplified during growth, resulting in major shifts from ancestral conditions. A dramatic example of this will be discussed in section 4, where a small increase in initial size of the apical ectodermal ridge in the chick limb produces a polydactylous adult.

Heterochronic changes can be classified according to the nature of perturbations in growth parameters. One class "promotes" earlier developmental stages of ancestors to adult stages in the organism under study. The other class of perturbations results in new morphologies that have not been observed in the lineage leading to the organism. Negative perturbations in growth rate and offset signal produce descendants whose morphology resembles juvenile stages in the ancestor. We call these forms, collectively, paedomorphs, and the retarding process which produces them *paedomorphosis* (Figure 15). This is the usual use of this term. The

TABLE 1. Summary of relationships between heterochronic processes of development, controlling parameters, and morphological and phylogenetic results of the processes. *Paedomorphosis and peramorphosis refer to shape related phenomena, and it is not appropriate to use these terms for pure size changes.

Control parameter	Incremental change	Process	Morphological expressions	Phylogenetic phenomenon
k_s	$+\delta k_s$	Proportional giantism		*Recapitulation
	$-\delta k_s$	Proportional dwarfism		*Reverse Recapitulation
k_σ	$+\delta k_\sigma$	Acceleration	Peramorphosis	Recapitulation
	$-\delta k_\sigma$	Neoteny	Paedomorphosis	Reverse Recapitulation
β	$+\delta\beta$	Hypermorphosis	Peramorphosis	Recapitulation
	$-\delta\beta$	Progenesis	Paedomorphosis	Reverse Recapitulation
	$+\delta\alpha$	Post displacement	Paedomorphosis	Reverse Recapitulation
	$-\delta\alpha$	Pre displacement	Peramorphosis	Recapitulation

opposite process occurs when positive perturbations in the growth rate or offset signal produce descendant organisms whose form transcends that of the ancestor. No appropriate term exists, so we shall call these, collectively, *peramorphs* ("shapes beyond") and the associated accelerating process *peramorphosis* (Figure 16). If β remains constant, post displacements produce paedomorphs, while pre displacements produce peramorphs. However, if the growth interval remains fixed, then no adult morphological change occurs (Figure 17). Our terminology to this point is summarized in Table 1, columns 1, 2, 3 and 4).

We do not expect "pure" changes of the type described in this section; observed morphological patterns are usually produced by combinations of basic events. In most cases this means that there will be some ambiguity in the events that produce the observed pattern. As an example of an effect produced by combined events, Newell (1949) reported an interesting case yielding recapitulation in sutural complexity within a lineage of Paleozoic ammonoids. Here we can separate out the joint effects of all three processes producing recapitulation: acceleration, pre displacement, and hypermorphosis. Newell plotted the regression of sutural length vs. shell diameter in ancestors and descendants. (Information on α usually cannot be obtained directly from fossils. However, perturbation of α can be inferred from data such as those presented by Newell. If the slopes of related species are the same, positive changes in y intercept ($+\delta y_{\text{int}}$) always correspond to negative perturbations in age of onset ($-\delta\alpha$). Conversely, negative changes ($-\delta y_{\text{int}}$) correspond to positive perturbations in age of onset ($+\delta\alpha$).)

The allometry of the ammonoids in Newell's study is positive and the suture increases in relative length throughout ontogeny. In some cases among the five genera, the slope of the descendant's curve is higher, but it also has a higher y-intercept and the two curves do not converge at small shell sizes. (In other cases, the curves are parallel and recapitulation results from pre displacement and hypermorphosis only). Therefore:

A.) pre displacement. Since the two curves do not converge, the descendant begins its ontogeny with a longer and more complex suture than its ancestor had at the same size and growth stage. The descendant begins with a "head start," probably (though we can't prove it) because the growth forces responsible for sutural allometry start to operate earlier and produce a more complex suture at the initial stage.

B.) acceleration. The descendant's slope is higher than the ancestor's. Descendants not only begin with a head start, but they differentiate their sutures faster during ontogeny. These two processes combine to produce a suture of ancestral length when the descendant is still a juvenile, one fifth the size of its ancestral adult.

C.) hypermorphosis. This lineage exhibits marked phyletic size increase. Hence it continues its sutural allometry well beyond the size of ancestral adults and develops an even more complex suture as a result.

4. *Recapitulation and reverse recapitulation.*—Using the classification scheme outlined in Table 1 we can reexamine the phenomenon of recapitulation in phylogeny. For example, consider a hypermorphic "mutation," $+\delta\beta$, which produces a descendant morphology, $\underset{\sim}{Y}_A$,

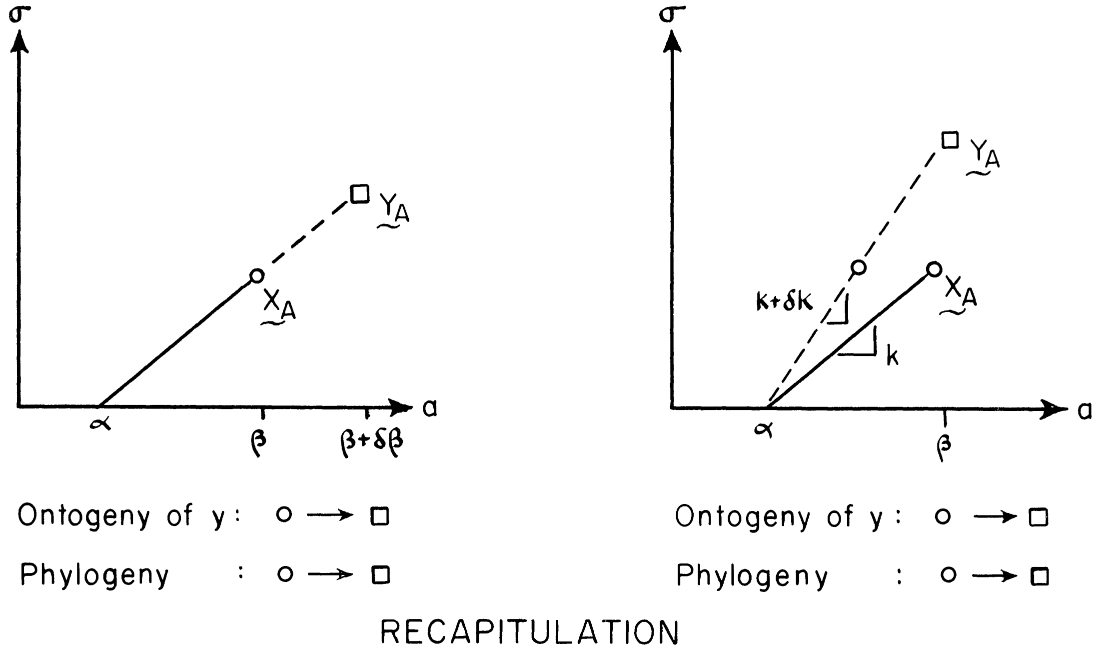


FIGURE 18. Hypermorphosis ($+\delta\beta$) and acceleration ($+\delta k$) produce recapitulation. That is, the ontogeny of the peramorphic descendant recapitulates the phylogenetic progression.

as shown in Figure 18. The ontogenetic progression in form (0 → □) is the same as the phylogenetic sequence (ancestor, $\underset{\sim}{X}_A = 0$; descendant, $\underset{\sim}{Y}_A = \square$), providing we compare adult forms. Thus, hypermorphic changes produce recapitulation, i.e. the descendant's ontogenetic trajectory passes through a sequence of forms in the same order as the phylogenetic progression of adult forms. Similarly, accelerations, $+\delta k$, produce recapitulations, as shown in Figure 18.

Conversely, progenetic ($-\delta\beta$) and neotenic ($-\delta k_\sigma$) "mutations" produce a developmental sequence in the ancestor which is the reverse of the phylogenetic sequence (reverse recapitulation). This is illustrated in Figure 19. By similar reasoning, we see that pure post displacements, $+\delta\alpha$, produce paedomorphosis and result in a phylogenetic pattern of reverse recapitulation—providing the offset signal, β, is unchanged (Figure 20a). However, if the growth period ($\beta-\alpha$) remains constant, the post and pre displacements produce neither recapitulation nor paedomorphosis (c.f. Figure 20b), i.e. the ancestral adult shape is reached by the descendant, but at an earlier or later time than in the ancestor. Table 1, columns 4 and 5, summarizes this terminology. Note that we are maintaining a clear distinction between the morphological expression of growth perturbations (peramorphosis and paedomorphosis) and the question of how ontogenetic sequences relate to phylogenetic sequences. We maintain this logical distinction even though the *processes* of peramorphosis are always associated with recapitulation and the *processes* of paedomorphosis always produce reverse recapitulation. By separating the ontogenetic processes from their phylogenetic consequences, we hope to lessen the confusion of terminology when discussing actual examples.

5. *Relation to "functuational change."*—Most growth models exhibit an initial phase of exponential growth. (This is due simply to the geometrically multiplying population of newly differentiated cells.) This initial growth phase has the property that small perturbations in growth rate, δk, or onset age, $\delta\alpha$, are amplified enormously. For example, over a developmental time of 100 ln 2 age units, an increase of only 1% in the growth rate ($\delta k = .01$) will result in a twofold increase in size. This means that the

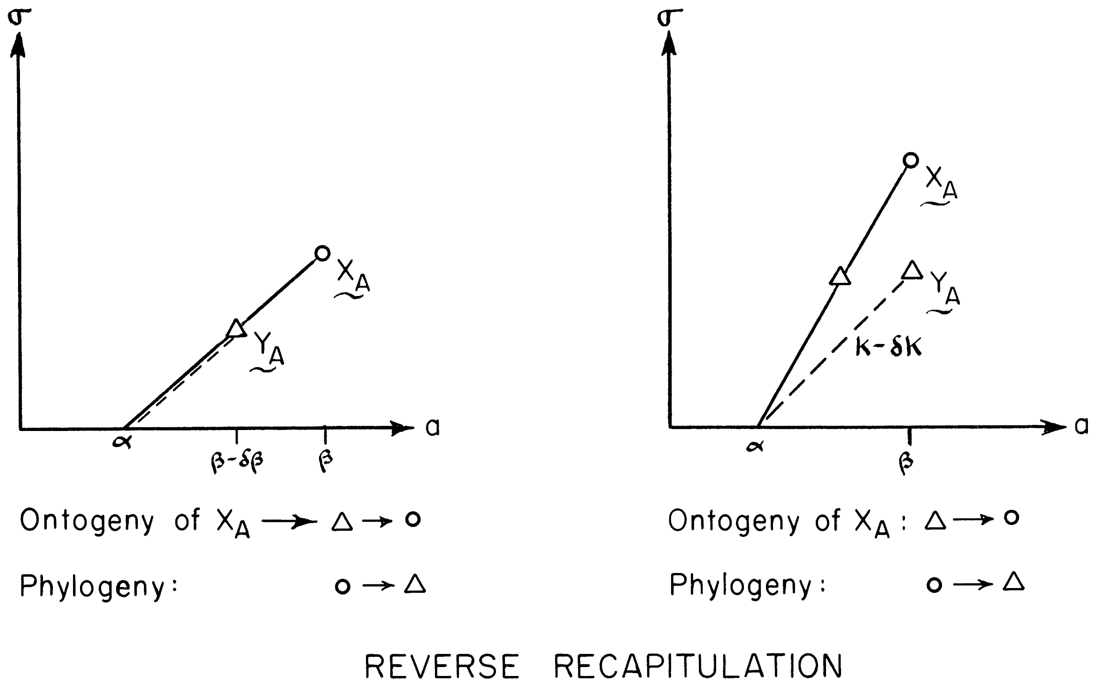


FIGURE 19. Progenesis ($-\delta\beta$) and neoteny ($-\delta k$) produce reverse recapitulation. That is, the ontogenetic sequence of the ancestral shapes is the reverse of the phylogenetic progression from ancestor to (paedomorphic) descendant.

relative adult proportions of an organ—or of adjacent organs or tissue types—can be grossly altered by relatively minor shifts in growth parameters. Since the course of normal development is directed to a large extent by tissue interactions, a distortion of tissue juxtapositions can result in a qualitatively different organ structure, and even a complete restructuring of the local architecture (see Wessels 1977 for a review of this subject). This phenomenon provides a developmental basis for producing major morphological shifts within but a few gen-

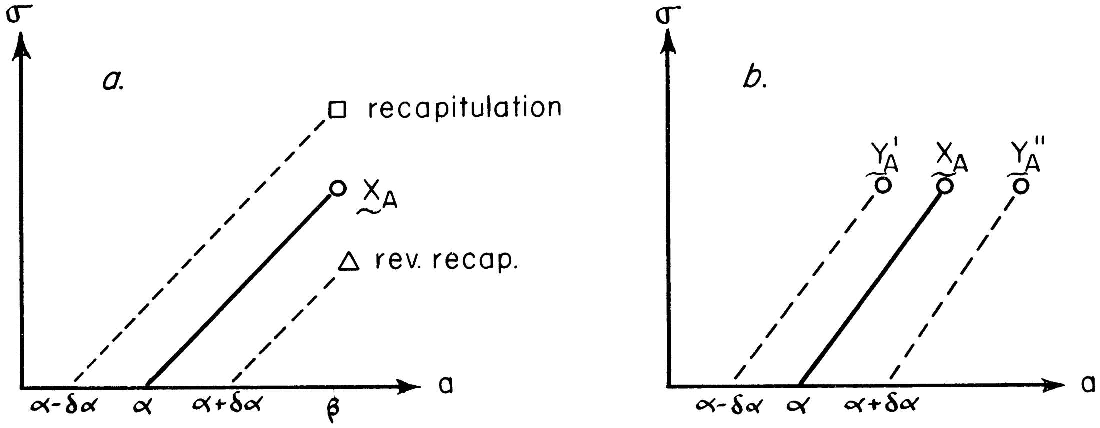


FIGURE 20. (a) Pre displacements ($-\delta\alpha$) produce recapitulation and post displacements ($+\delta\alpha$) produce reverse recapitulation. (b) If the growth interval ($\beta-\alpha$) remains unchanged during phylogeny, neither recapitulation nor reverse recapitulation is observed.

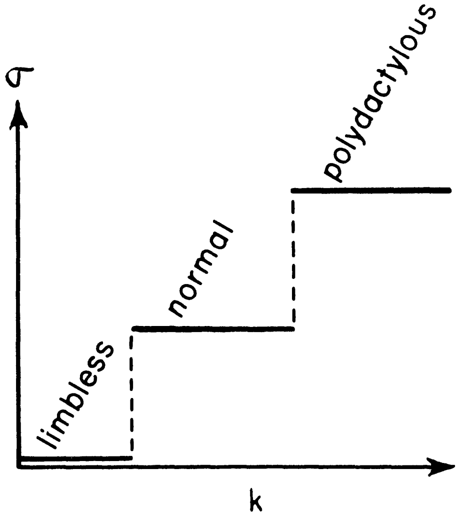


FIGURE 21. Hypothetical relationship between shape and rate of proliferation of cells in the apical ectodermal ridge of birds. Continuous perturbation in *k* (growth rate) of the apical ectodermal ridge can lead to discontinuous shape phenotypes, having the characteristics indicated.

erations, providing that heritable mutations do not create reproductive barriers or incur selective disadvantages.

In the classical embryological literature on developmental defects, it is relatively easy to find examples of how a small "mutation" in a growth parameter causes a series of developmental alterations that produce a phenotype qualitatively different from the normal one. The factors controlling the rate of cell division are not known with certainty, but the process whereby a tissue alters the growth and proliferation rate of neighboring tissues is well described (cf. Chandebois 1976).

It is not the purpose of this paper to provide an exhaustive review of tissue interactions and the phenomena of induction. Rather we wish only to point out that developmental dynamics can explain how continuous mutations at the level of controlling parameters can produce discontinuous phenotypes. An example illustrating this point is the phenomenon of polydactyly in chickens.

Limb morphogenesis is the product of interactions between the ectodermal and mesodermal tissues in the limb bud. The underlying mesoderm induces a thickening in the apical zone of ectodermal epidermis, forming a region known as the apical ectodermal ridge (AER). The AER, in turn, produces an inducing factor acting on the mesoderm responsible for regulation and control of the limb mesodermal element. We know little about these factors or their mode of action, but the regulatory properties of both tissues on each other have been clearly demonstrated (see Ede 1971 for a review on limb morphogenesis).

The only apparent difference between a normal limb bud and one from a polydactylous mutant is a larger AER in the latter. In polydactylous individuals either more ectodermal maintenance factor is synthesized or its distribution in the mesoblast is abnormal, so as to induce more apical ridge and additional sites of distal morphogenesis. Here we hypothesize that in the mutant the maintenance factor from the mesoderm causes an alteration in the rate of proliferation (δk_s) of the apical ectodermal cells. This larger apical ridge causes an increase in the mesodermal area in contact with it, and this in turn increases the potential sites of distal morphogenesis. Another possible explanation, also consonant with our model, is that an increase in the number of cells in the AER produces a proportional increase in the amount of AER-inducing factor affecting mesodermal differentiation.

As an opposite result, we have the wingless mutation in which the absence of AER causes a wingless phenotype. Figure 21 illustrates a hypothetical model of how continuous perturbation in *k* (growth rate) of the AER can produce discontinuous phenotypes.

Another example is provided by Hampé (1959), who through a series of ingenious experiments has been able to produce a complete fibula in the leg of chickens by various manipulations during development. Modern birds lack a complete fibula. By starting with a fate map of a chick limb bud, Hampé demonstrated that manipulations directed at the fibular region often resulted in production of a fibula that was not only complete but that also bore close similarity to the fibula of *Archaeopteryx* (Figure 22). This was accomplished in various ways: 1) by removing the terminal cap of an advanced limb bud and replacing it with an entire early limb bud; 2) by augmenting the "fibular" zone of the limb bud with tissue from the identical

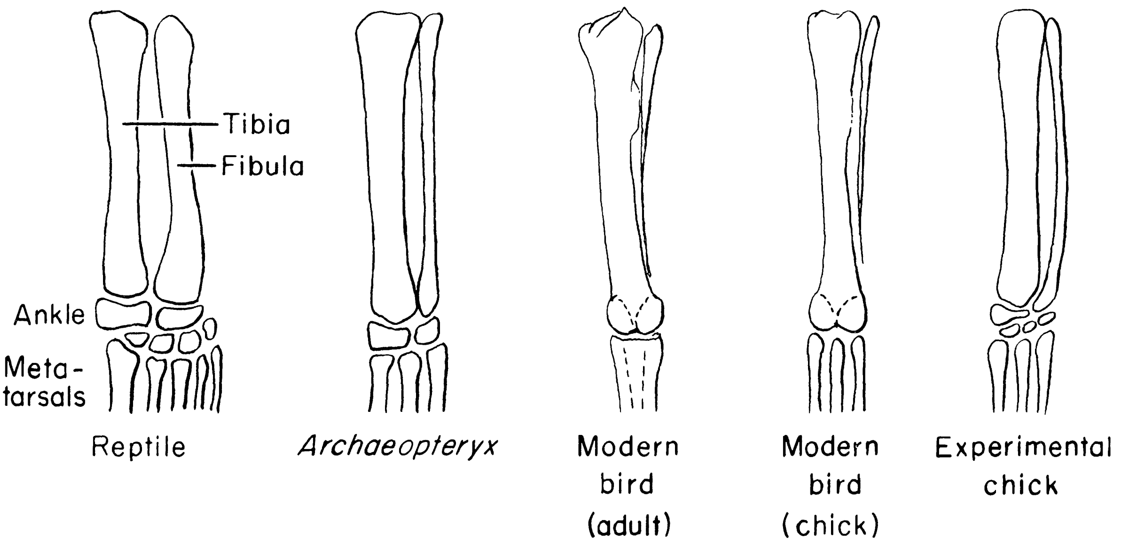


FIGURE 22. The legs of several organisms, to show the significance of the work of Hampé (1959). Modern birds lack a complete fibula and have a simplified ankle. Using a variety of experimental techniques, Hampé was able to induce full development of the fibula and of some ankle bones. The developmental program for this region has apparently been little altered in the course of evolution, and by keeping the program "turned on" structures appear in a form closely resembling those of ancestral conditions (figure modified after Frazzetta 1975, Fig. 47).

zone of another limb bud; 3) by insertion of a physical barrier between the "fibular" and "tibial" zones of the limb bud. In the first two instances the development of a fibula results from augmentation of the fibular zone with similar cells; in the latter instance a tissue interaction was eliminated. When this fibula grows to its full ancestral length (as in *Archaeopteryx*) and establishes contact with the metatarsals below, a series of stunning correlative changes occur. First of all, the metatarsals remain unfused (as in reptiles and *Archaeopteryx*). (They are also unfused in hatchling chicks but fused in adults.) Secondly, a series of ankle bones differentiates freely between the tibia-fibula and the metatarsals (normal chickens, both hatchlings and adults, form two ankle bones, but they are fused to the tibia) (Figure 22). These ankle bones, more than in *Archaeopteryx* but fewer than in standard reptiles, may be homologous with the ankle bones of avian ancestors.

The most reasonable inference we can make from these results is that the fibula acts as an inducer for ancestral structures (or at least as a preventer of fusions). It is probable that the genetic capacity for producing these ancestral structures was never lost during more than 200 million years of avian evolution but merely "turned off" by failure of the inducing fibula to establish contact with the metatarsals. The failure of the fibula to reach this full length in normal birds (where it remains as a splint adjacent to the proximal end of the tibia) probably results from small changes in growth rates early in embryonic differentiation—since it can be reversed simply by adding more limb bud in the fibular region or by preventing "competition" from the differentiating tibia for the limited material available in the joint limb bud (as if this complex set of events in avian evolution arose merely from increased growth rate of the tibia with consequent reduction of the fibula and failure of the structures it induced at full size). Thus, a train of complex evolutionary events may follow from a simple alteration of growth rates early in ontogeny.

Latent capacities for the generation of ancestral structures probably exist in the genomes of all specialized animals. Raikow (1975), for example, reports the recurrence of different ancestral muscles in two passerine bird specimens with developmental anomalies. The absence of both these muscles forms part of the standard definition of Passeriformes. In his discussion,

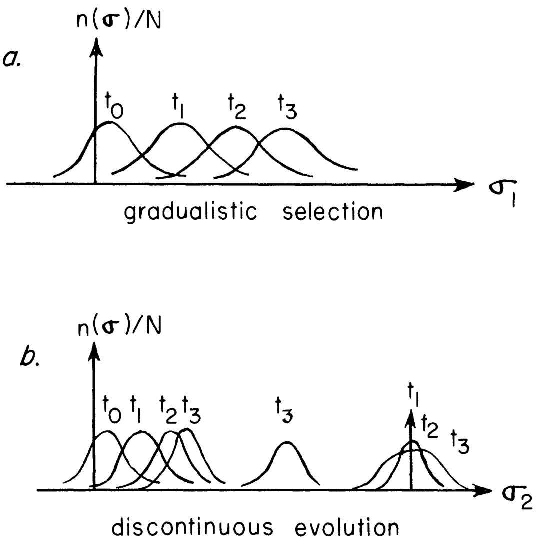


FIGURE 23. Diagramatic illustration of the two main modes of morphological evolution. If higher σ values are favored by natural selection, they can be obtained through phylogeny by a gradual drift of the shape distribution due to directional selection as shown in (a). Alternatively, discontinuous jumps to higher σ-values can also occur by a variety of random developmental mutations; a new σ-value can become fixed in evolution through differential survival between the morphological pools. This process is shown schematically in (b): At time 1, a high σ mutant arises; these phenotypes spread out due to Mendelian variance. The new mutant phenotype is not necessarily reproductively isolated from the original type and this is illustrated by the appearance of a new discontinuous phenotype at time 3 due to backcrossing. Note that backcrossings do not necessarily produce intermediate phenotypes.

Raikow reviews other cases including the presence of incisor or canine teeth in the malformed skulls of two cyclopic lambs (the absence of these teeth is a diagnostic character of ruminant artiodactyls) and the ability of chick oral epithelium to exert an organizing effect upon mouse dental mesenchyme. Kollar (1972, p. 134), who reported this unrealized property of edentulous birds, concluded: "During evolution, the ability to make teeth may have been lost—not the genetic information; perhaps some subtle change in the timing of developmental events may be responsible for toothlessness in birds."

Depending on the system-specified developmental dynamics, the back crossing of mutant types with normal phenotypes can produce a great diversity of discontinuous phenotypes (Figure 23). Some of these types may possess discrete structural differences (cf. extra digits, new fibula, . . .) allowing descendants to use "mutant" structures for functions and behaviors not found in their ancestors. Concurrent changes in behavior and habitat utilization will subject them to a new selective regime. If selection is disruptive, a process of rapid evolution ("punctuation event" or "quantum evolution") leading to reproductive isolation will occur. Thus, our conception of evolutionary dynamics encompasses both the slow morphological shifts characteristic of phyletic gradualism and the dramatic changes associated with "discontinuous" evolutionary perturbations.

6. *Population dynamics and selection.*—The foregoing treatment is fundamentally a phenomenological method for describing heterochronic processes. In order to relate growth perturbations, $\delta\underset{\sim}{\gamma}$, to forces of natural selection, we must present the above descriptive formalism in a model for population growth and selection. The formal machinery for doing this exists in the literature. Explicating the model requires some rather formidable mathematics. Here we sketch qualitatively the way heterochronic changes in size and shape can result from the action of natural selection. We relegate a complete discussion of the mathematical model to a subsequent publication.

A population of maturing individuals can be represented by a "cloud" of points moving through age-size-shape space. At any moment the state of a population with N individuals is specified by the distribution of points X_i, $i = 1, \ldots N$ in age-shape-size. If N is large, we can represent the population mathematically by the density function

$n(t, a, S, \sigma \mid \alpha\beta k)$ = number of animals at time t, of age a, size S, and shape σ, and with growth rate parameters (α, β, k).

In order to simplify notation, we shall write $n(t,a,\underset{\sim}{y} \mid \underset{\sim}{\gamma})$, where $\underset{\sim}{y} = (S,\sigma)$ and $\underset{\sim}{\gamma} = (\alpha,\beta,k)$. The density function $n(\cdot)$ carries all of the relevant information concerning the population structure. Once it is known we can compute the average size and shape parameters for the population, as well as their variances, e.g.

$$\bar{\sigma}_{(t)} = \frac{1}{N}\int_0^\infty \sigma n \, \mathrm{d}\sigma \qquad (6)$$

$$\mathrm{var}(\sigma_{(t)}) = \frac{1}{N}\int_0^\infty (\sigma - \bar{\sigma})^2 n \, \mathrm{d}\sigma \qquad (7)$$

where $N(t) = \int\int\int_0^\infty n \, \mathrm{d}a \, \mathrm{d}S \, \mathrm{d}\sigma$ is the total population, i.e. the total number of points in age-size-shape space. This is shown schematically in figure 24.

Each individual, X_i, in the population traces out its own ontogenetic trajectory, governed by a growth law of the form given in equation (*). Thus, the evolution of the population is governed by how the density of points, $n(\cdot)$, shifts in time. It is possible to write an equation describing the evolution of the density function, $n(\cdot)$ (Oster 1977). It has the form

$$\frac{\mathrm{d}n}{\mathrm{d}t} = \text{Birth} - \text{Death} - \text{Growth} \qquad (8)$$
$$= B - D - G$$

That is, the number of individuals in a given age, size, and shape class can change by one of 3 mechanisms: (a) individuals can be born into the zero age-class, B; (b) individuals can be lost from any class by dying, D; and (c) individuals enter and leave a class by growth and aging, G. G is the growth law defined in section 2.

In a population with a distribution in growth parameters, $\underset{\sim}{\gamma}$, and where each individual's growth trajectory is deterministic, the distribution of adult phenotypes is determined uniquely by equation (8), as shown in Figure 24.

If birth and death rates are size and shape dependent, then we can use equation (8) to follow how natural selection shifts the distribution in response to environmental fluctuations, providing we can model the following processes:

(i) the mechanism of selection, e.g. how the environment differentially culls individuals according to the size and shape of the organ under discussion,
(ii) the mechanism of generating diversity in size and shape, e.g. mutation and recombination,
(iii) the mechanism of inheritance of size and shape parameters,
(iv) a model for environmental variability.

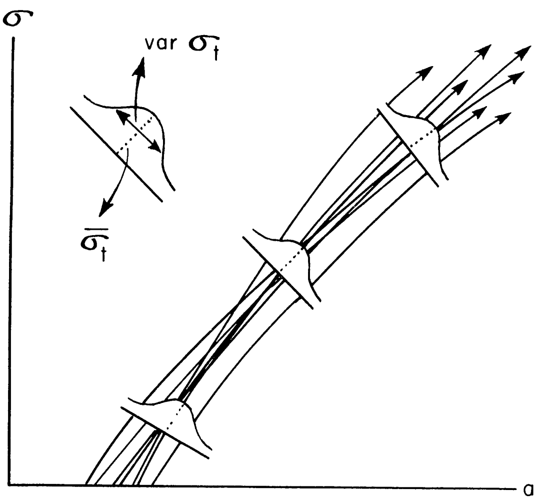


FIGURE 24. Each individual in a population has its own ontogenetic trajectory. The population is viewed as a "cloud" of points of density $n(t,a,s,\sigma)$ moving through the state space. $\bar{\sigma}_t$, var σ_t denotes the mean and variance of the population shape distribution, calculated according to equations (6) and (7).

It is clearly not possible to construct a general model to cover all circumstances; each situation requires its own set of submodels (i)–(iv). An example of how this program can be carried out in detail will be presented in a subsequent publication.

In Figure 25 we show schematically what the components must be of any model purporting to relate the ontogeny and phylogeny of size and shape. The environment, $E(t)$, is first filtered through the organism's physiological equipment, including its size and shape, $S(t)$, $\sigma(t)$. These, in turn, change as the organism matures, and they partially determine the age specific mortality and fecundity functions wherein selection actually occurs. Ultimately, $\bar{S}$, $\bar{\sigma}$, the average population size and shape *may* conform to the optimal average design for that particular environment, $E(t)$. However, this will generally *not* by the case, because of contravening constraints (Oster and Rocklin 1978). In any event, for a given environment, there may be an asymptotic population structure, $\bar{n}(t,a,S,\sigma)$, from which one can compute the "descendant" size and shape distributions. That is, if we regard the environment $E(t)$ as a stochastic "input" to the model, the stochastic "outputs" are the population quantities $\bar{\sigma}$, $\bar{S}$, the average size

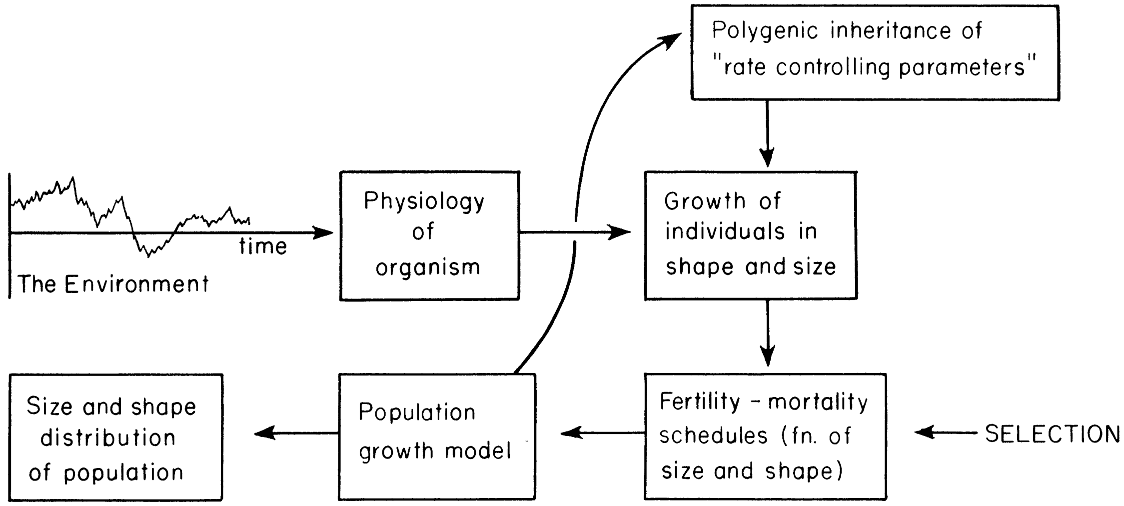


FIGURE 25. A schematic representation of how a population's size and shape distribution responds to selective forces. The stochastic environment $E(t)$ is filtered through the organism's physiological structure and affects its growth which, in turn, affects its fitness.

and shape of an individual (and their variances, etc.). Thus we can investigate, quantitatively, the relationship between the statistical properties of an environment and the evolutionary shape and size adaptations it produces.

We present this discussion to illustrate that the growth parameters, $\underset{\sim}{\gamma}$, which control the size and shape distributions of a population, are random variables reflecting a population's adaptations to its environment. There is a substantial literature relating various "life history strategies" to environmental variability, and here we see how size and shape may be added to the list. However, the complications of the model schematized in Figure 25 suggest no simple and universal relationship between environment and the life history of size and shape. Nonetheless, a number of authors have noticed several allometric relations between overall body size (or weight), lifespan, specific metabolic rate, and reproductive potential (for example, Southwood 1976).

Gould (1977, chapters 8 and 9) stressed the ecological significance of heterochrony. By presenting a series of examples he argued that progenetic species tend to live in uncertain environments characterized by ephemeral and randomly fluctuating resources. These species have high reproductive rates, mature at a relatively early age, and have high density independent mortality rates as well as other life history parameters traditionally associated with "*r*-strategists" in the literature. On the other hand, neotenic species tend to inhabit predictable environments and are characterized by life history parameters traditionally attributed to "*K*-strategists." Gould presented heuristic arguments why one should expect correlations between progenesis and "*r*-environments" and neoteny and "*K*-environments." This does not imply an acceptance by the authors of the "*r-K*" dichotomy, now the subject of much controversy (see Stearns 1976, 1977, for a critical review of this subject). Nonetheless, the most obvious suggestion of our outline is the absence of strongly predictable relationships between environment and shape and size, since the effects of the environment depend on the particularities of the physiology and behavior of the organism under study. Yet, the empirical correlations are real and the approach sketched in this section should allow the relationships between heterochronic processes and environmental characteristics to be investigated quantitatively.

Conclusions

Our goal has been to show how the evolution of size and shape can be related to developmental, populational, and environmental phenomena in order to generate testable hypotheses relating to the interface of growth in individuals and populations.

Most of the changes in size and shape that we consider fall under the general category of phenomena termed heterochrony (DeBeer 1958; Gould 1977). As much as possible, we have tried to use existing terms in the manner proposed by Gould (1977), but we have adapted them to our formalism. Our focus has been on particular developmental events. The phenomena described can apply equally well to processes that involve many seemingly disparate but actually linked events. When metamorphosis fails to occur in salamanders, for example, features of the skin, skull and other parts of the body can be affected in a unitary way. Thus, heterochrony can produce not only a simple advance or delay in the appearance of structures, but also a set of *subtle* consequences, pervading the organism, and not obviously related to simple changes in the timing of development.

Attention should be focused on rate parameters, timing, and direction of change rather than on the names used for the processes, expressions, and phenomena. We have mainly used existing terms to describe essentially mathematical phenomena, i.e. perturbations of the rate-controlling parameters along different axes in our α, β, k space. Accordingly, the terms as we have used them have more precise definitions than in the past.

We hope that the formalism used herein will encourage workers to approach problems of morphological evolution from a truly dynamic perspective. It is unsatisfying and incomplete to do an analysis of phenotypic change through time simply by describing forms as static entities and subsequently linking them according to trends and similarities. We want to know why phenotypes change the way they do and we want to make testable predictions. Since epigenetic interactions are so complex, it is usually impossible to reduce morphological evolution to a problem in population genetics. Thus, we are forced to devise phenomenological methods that allow us to analyze and describe processes of phenotypic change quantitatively. The models and methods of Lande (1976) and Slatkin (1970) were a valuable first step in bridging the gap between genetic theory and phenotypic evolution, but they dealt with basically static morphologies. By dealing with ontogenetic trajectories, and thus making contact with development, we have added another dimension. Developmental dynamics and its genetic controls must be probed by evolutionary biologists if we are ever to understand fully the paradoxical lack of correlation between morphological and genetic evolution (Wilson et al. 1977). We know that slight perturbation in the ontogenetic trajectory of an organ can be amplified through time, by the dynamics of growth and tissue interactions, to produce an adult phenotype drastically different from that of the ancestor. Clearly, major morphological changes can be the product of minor genetic mutations. In this paper we have explored ways of dealing with variations on a theme—the theme of general body plans (or developmental programs). Many elements of morphological evolution can be interpreted as minor reshuffling within a fundamental developmental program during phylogenesis. We think that many so-called evolutionary novelties can be explained this way, but others are the results of changes in the developmental program itself.

Our ontogenetic trajectories should be viewed as analogous to Waddington's (1957, 1962) "chreods" or developmental pathways which have complex epigenetic controls. Hence, the phylogenetic processes associated with evolution of ontogenetic trajectories can be reanalyzed as a problem in developmental dynamics. For example, evolutionary experimentation is expected mainly in later stages of development. Complex non-linear interactions occur during development, so that small perturbations of the kind outlined in this paper can be dramatically amplified through ontogeny. To produce a viable product these perturbations more likely can be accommodated in later stages of ontogeny. Ontogenetic trajectories are resilient, to some degree, due to feedback interactions between rate controlling parameters. For example, post and pre displacement processes can occur, but a "normal" adult phenotype may still result if the termination of the growth process, β, is appropriately altered (Figure 20b). The above discussion tries to show how the model outlined in this paper is more than a formalism to classify heterochronic processes. We consider it to be a systematic way of dealing with and analyzing processes of morphological evolution by treating them as problems of developmental dynamics controlled at their basic level by programs of gene regulation.

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APPENDIX

List of symbols used in the text

a = age
B = birth
b = allometric exponent
D = death
d = a developmental event

G = growth rate of size
g = rate of change in shape
k = growth rate parameter
N = total number of organisms in the population
n = number of organisms of a given age, size, and shape
P = phylogenetic map
r = intrinsic rate of increase of a population
S = size
t = time
X, Y = ontogenetic trajectory
Y_{m} = size or shape at maturity
y = ontogenetic path of either size or shape
α = onset in time of a developmental event
β = cessation in time of a developmental event
$\underline{\gamma}$ = genotypic state of system
$\underset{\sim}{\sigma}$ = shape
ϕ = mapping of developmental event (d) onto a age (a)