

MORPHOMETRIC HETEROCHRONY AND THE EVOLUTION OF GROWTH

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Abstract.—Heterochrony has been an influential perspective on the evolution of morphologies, a circumstance mostly due to a strategic shift of the theory to the analysis of growth and measurable traits. A difficulty in testing hypotheses of heterochrony in the morphometric realm, and therefore in establishing its evolutionary relevance, has been the absence of an explicit criterion of homology in comparisons supposed to reveal paedomorphosis and peramorphosis. Based on the formalism of ontogenetic and allometric trajectories, we defined a criterion of primary homology in the context of morphometric characters that requires only a comparison between metric traits from ontogenetic series of two or more taxa. On the one hand, such a criterion allows for the calculation of values of shape slopes and allometric coefficients in descendants supposedly affected by changes in ontogenetic timing, thereby supplying an analytical tool for testing hypotheses of heterochrony. On the other hand, the concept of morphometric homology establishes the descriptive limits of paedomorphosis and peramorphosis, showing, for example, that the model of sequential hypermorphosis applied to the evolution of human encephalization is not within the descriptive scope of the morphological markers of heterochrony. Sequential hypermorphosis is a successful model of morphometric evolution, as further illustrated by the match between our mathematical deductions and the empirical results obtained by analyses of brain growth data. By exploring the properties of multiphasic polynomial functions, we deduce equations that define the relationship between developmental delay or acceleration and their effect on adult brain size. Together with the primary criterion of homology, we demonstrate that sequential hypermorphosis could generate the large modern human brain, but such brain is neither paedomorphic nor peramorphic. Our approach based on homology and allometry indicates that the evolution of growth is richer in phenomena than heterochrony can account for, and accordingly we argue that morphometric theory can expand its descriptive and heuristic scope by looking beyond the limits imposed by paedomorphosis and peramorphosis.

Key words.—Allometry, heterochrony, homology, human encephalization, sequential hypermorphosis.

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Stephen Jay Gould's *Ontogeny and Phylogeny* (1977) was a reinstatement of heterochrony as an alternative approach to evolutionary studies. The claim was justified on two grounds. First, heterochrony relied on morphological markers that apply to whole morphologies, namely paedomorphosis (the evolution of juvenilized shapes by the subtraction of terminal ontogenetic stages) and peramorphosis (overdeveloped morphologies by terminal addition of ontogenetic stages). The concept of adaptation would be relegated a secondary role, thus avoiding a fragmentation of organisms into functional units denounced by Gould and Lewontin (1979). Second, heterochrony, or evolution by "changes in developmental timing that produce parallels between the stages of ontogeny and phylogeny" (Gould 1977, P. 2), would bring about a shift to a more mechanistic perspective based on the study of ontogenesis.

Heterochrony has been further explored as a theoretical approach to morphometric evolution (Alberch et al. 1979; Shea 1983a; McKinney and McNamara 1991; Vrba 1994, 1998; Godfrey and Sutherland 1995, 1996; Rice 1997) and applied to the study of various animal groups (Emerson 1986; Wayne 1986; Edgecombe and Chatterton 1987; Klingenberg and Spence 1993; Friedman and Carmichael 1998; Rice 2001). But heterochrony has to some extent neglected a key element of any comparative study, namely an explicit assessment of homology. This is a particularly important issue in morphometric studies, where shape values are often encoded as pure numbers, the comparison of which cannot in itself distinguish similarity by the sharing of homologous shape stages in two taxa (an assumption of hypotheses of heterochrony) from similarity by mere phenetic resemblance.

In the following, we argue that a primary assessment of homology in morphometric studies requires the use of the formalism of ontogenetic trajectories. Based on the latter we formulate a criterion of homology to be used in studies of heterochrony. The need for a criterion of homology and for the formalism of ontogenetic trajectories is evident from an examination of the clock model (the first version of morphometric heterochrony), as discussed below.

Heterochrony and Displacement of Ontogenetic Stages: An Extended Clock Model

Gould's measurable or morphometric heterochrony originally took the form of the clock model, which limited heterochrony as a process to changes in ontogenetic timing, and with regard to its products to recapitulation (peramorphosis) and antirecapitulation (paedomorphosis), that is, to the diagnosis of displacements of ontogenetic stages. It represented ontogeny as a three-dimensional vector Ω (S , adult size; σ adult shape; t , total ontogenetic time), and variations imposed on it as a perturbation vector $\Delta = (\pm\delta_S, \pm\delta_\sigma, \pm\delta_t)$. Paedomorphosis and peramorphosis could therefore be quantitatively defined respectively as $\delta_\sigma < 0$ and $\delta_\sigma > 0$ in putative heterochronic descendants.

The clock model was never fully explored as a predictive device, as exemplified by the almost unnoticed fact (with the exception of Klingenberg 1998) that it implies 26 kinds of heterochrony—27 ($= 3 \times 3 \times 3$), because size, shape, and time can vary positively, negatively, or remain the same, minus one, because $\Delta = (0, 0, 0)$ represents the ancestral ontogeny. But the model originally refers to seven possibil-

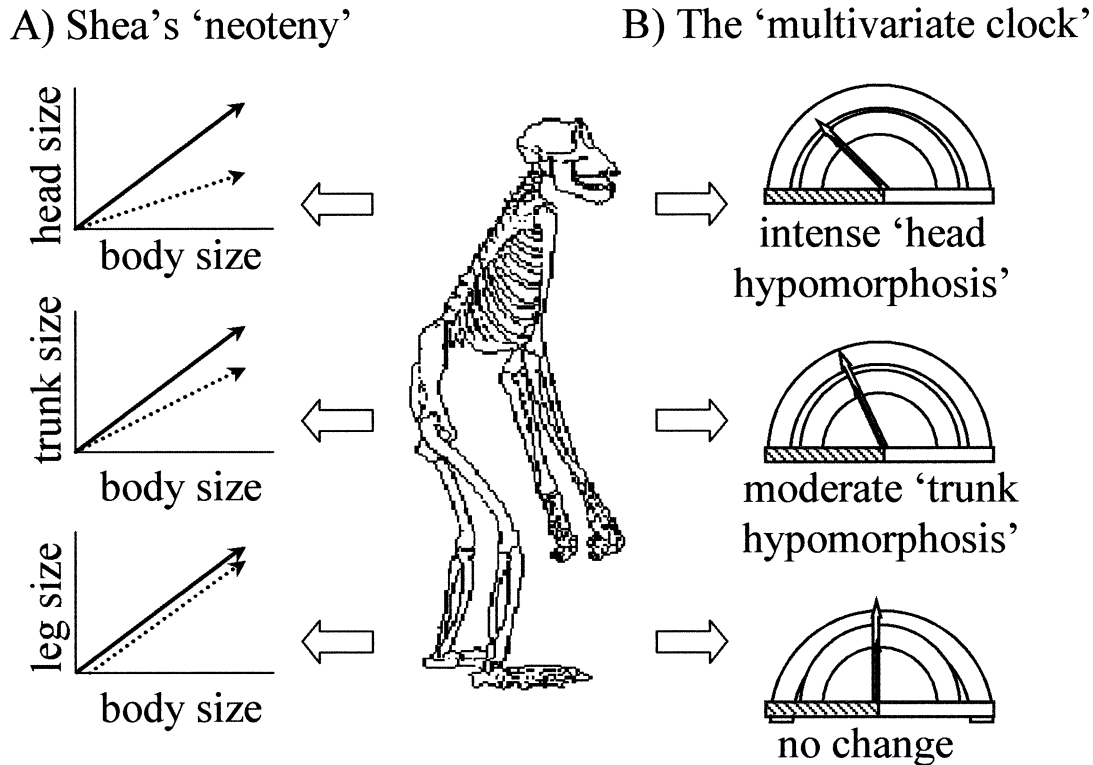


FIG. 1. The multivariate clock model. According to Shea (1983a), decrease in bonobo trait sizes was not uniform in a comparison with common chimpanzees. Godfrey and Sutherland (1996) interpreted bonobo morphology as a case of dissociation of growth fields, with decreasing intensity of hypermorphosis in the anteroposterior direction.

ities or heterochronic modes only (progenesis, neoteny, human neoteny, hypermorphosis, acceleration, proportioned gigantism, and dwarfism), and except for Shea's hypothesis of rate hypermorphosis (Shea 1983b), it has never been used to discover or predict new modes. This is probably a consequence of the dubious concept of ontogeny underlying the model. Perturbation vectors denoted patterns of dissociations between the three fundamental processes of ontogenesis, namely growth, development, and maturation, as expressed in the view that "some correlations [between growth, development and maturation] are more tenacious and difficult to disrupt than others; yet all can be dissociated" (Gould 1977; P. 260). The fact is that the properties of ontogenetic dissociability or modularity recently unveiled by developmental genetics do not correspond to the tripartite ontogeny clock model, which for this reason remains closer to an ad hoc description of some phylogenetic patterns (those that pedomorphosis and peramorphosis are believed to describe) than to an operative model of ontogenetic and morphometric evolution.

An alternative extension of the clock model is its multivariate formulation pioneered by Shea (1983a) and explicitly formulated by Godfrey and Sutherland (1996). Shea observed that bonobos (or pygmy chimpanzees, *Pan paniscus*) exhibit a nonuniform reduction of allometric coefficients in comparison with *Pan troglodytes*, being more intense in the head, moderate in the forelimbs and trunk, and absent in the hindlimbs (Fig. 1). The combination of less intense skull shape development and similar body mass in *P. paniscus* led Shea

to a diagnosis of skull shape neoteny. Godfrey and Sutherland (1996) argued that the bonobo is not an example of neoteny as defined by Gould (a descendant with similar size, similar developmental time and juvenilized shape), showing that the correct allometric translation of Gould's (1977) neoteny is a global trend of allometric coefficients toward isometry (i.e., they decrease if greater than one and increase if smaller than one). Consequently, the nonuniform reduction of allometric slopes in the bonobo cannot be described by either pedomorphosis or peramorphosis. As such, the bonobo exemplifies a morphology beyond the descriptive domain of heterochrony: it is something new, which neither recapitulates nor anti-recapitulates a *troglodyte*-like ontogeny (as pointed out by Shea himself).

Godfrey and Sutherland (1996) tried to rescue the formalism of quantitative heterochrony with a multivariate ontogenetic clock and the idea of dissociation of growth fields, defined in developmental biology as the relatively independent evolution of distinct epigenetic systems, such as in the premaxilla, autopodium, and feeding system of plethodontid salamanders (Wake and Larson 1987). The bonobo was interpreted as a mosaic of distinct pedomorphic body parts produced by local ontogenetic scaling, with decreasing intensity along the anteroposterior axis, resulting in a pedomorphic head, a less pedomorphic trunk, and normal hindlimbs, or a heterochronic gradient.

However ingenious this theoretical move may be, the multivariate clock also presents difficulties. First, it introduces the additional mechanistic difficulty of explaining mosaic

heterochrony, that is, how we define growth fields and how they could be independently or differentially affected by heterochrony. Whereas the genetic and developmental basis of cell field and body segment identity is known to rely on a hierarchy of patterning genes (Carroll et al. 2000), little is known about the behavior and regulation of those fields as growing entities. Second, despite being a device that can appropriately describe morphological differences, the multivariate clock arguably implies that the markers of heterochrony lose their original appeal. Under the multivariate formulation, heterochrony is no longer a theory of integrated evolution. In other words, it could be argued that by not constraining heterochrony to its original scope, the multivariate clock introduces mechanistic difficulties regarding the testing of hypotheses of heterochrony in morphometric studies.

Allometry as a Metric for Growth: A Criterion of Primary Homology in Heterochrony

The unsolved problems generated by the clock model in its morphometric use were arguably the main reason for the introduction of allometric methods into heterochronic analysis. This defined the tradition of allometric heterochrony sensu lato (Shea 1983b; McKinney and McNamara 1991; Vrba 1994; Godfrey and Sutherland 1995, 1996), in which allometry appears as a metric for heterochrony. Growth and allometric curves replaced ontogenetic vectors as units of ontogeny, and allometry is supposedly supplying morphometric heterochrony with the mechanism it required—namely growth as the ontogenesis of morphometric traits. But most important, allometry allows for the indispensable assessment of homology in heterochrony, as seen below.

Heterochrony involves intra- and intertaxonomic comparisons, thus belonging to the field of comparative biology, with the two special circumstances that taxa are often compared at different developmental stages and that the aim of the analyses is mainly to establish ancestor-descendant (instead of sister group) relationships. Given that the diagnosis of pedomorphosis and peramorphosis is based on the presence of morphological similarities between adults and other developmental stages from different taxa, the question of how informative such similarities are applies to heterochrony, too. According to systematic phylogenetics, only the sharing of derived character states of homologous traits (synapomorphies) can establish evolutionary kinship (Forey et al. 1992). This equals excluding other forms of phenotypic similarity, such as analogy and plesiomorphy, as phylogenetically informative.

Unfortunately, heterochronic studies based on morphometric data have seldom addressed directly the problem of homology. If one takes the clock model as an example, while homologous traits can be defined (e.g., when a ratio or angle is defined on the basis of homologous organ or surface landmarks), their character states (the values of proportions or angles themselves) happen always to be pure numbers. Establishing phylogenies and assessing homologies from numerical data is a complex and controversial issue (Harvey and Pagel 1991), but it seems undeniable that direct comparisons between shape values in two taxa A and B cannot

per se distinguish between similarity by displacement of a homologous ontogenetic stage and similarity by mere coincidence. For instance, Gould (1977, P. 256) stated that “[human] shape is clearly retarded [i.e., pedomorphic], since in many respects an adult human skull resembles the standard juvenile condition of most primates,” showing that the clock model is intrinsically phenetic and grounds phylogenetic inferences on morphometric similarities.

It was believed that the introduction of allometric methods into heterochrony would provide such criterion of homology (or similarity by common ontogeny) between ontogenetic stages, leading to the formalism of ontogenetic trajectories (Alberch et al. 1979), or a n -dimensional coordinate system consisting of a set S_k of k coordinates for size (including body size and any trait sizes), a set σ_j of j coordinates for shape (i.e., ratios obtained from S_k , or angles), and one time coordinate. Ontogeny is described by the trajectory followed by the point $X(S_k, \sigma_j, t)$ in the n -dimensional ontogenetic space. However, the crucial question of how to identify Gould’s heterochrony in the ontogenetic space remains partially unanswered. Rice (1997, 2001) correctly argued that heterochrony requires the preservation of an ancestral ontogenetic trajectory under uniform changes in timing, but his model refers to growth curves (and not to shape curves) and leads to conclusions not necessarily corresponding to the morphological categories of heterochrony. In the following, we demonstrate some essential properties of the ontogenetic trajectory and formulate a minimum or primary criterion for homology between developmental stages implicit in heterochrony. Based solely on morphometric analysis of ontogenetic series, the criterion allows for a clear mathematical distinction between heterochrony and other allometric phenomena.

Heterochrony denotes a displacement of ontogenetic stages (so that adult stages become fetal or juvenile and vice versa) caused by the differential extension of a common (i.e., homologous) sequence of shape stages in ancestors and descendants. The morphometric equivalent of this criterion is the conservation of a common shape trajectory (the projection of the global ontogenetic trajectory onto σ_j , the subspace of shape trajectories) and its differential extension (Fig. 2).

Now suppose that a given descendant preserves all the ancestral correlations between trait size, body size, and time and undergoes a truncation or extension of its global ontogenetic trajectory. That would trivially imply the same sequence of shape stages in ancestors and descendants, and either ontogenetic recapitulation (peramorphosis) or anti-recapitulation (pedomorphosis) necessarily follows. Such behavior of the shape trajectory is compatible with the claim that morphometrically similar ontogenetic stages may be homologous. Ontogenetic scaling (Shea 1983b), therefore, corresponds to an allometric process that in principle generates heterochronic consequences.

Take now the case (that includes neoteny and acceleration) of dissociations between development and growth. Godfrey and Sutherland (1996) analyzed dissociations between shape and size stages, proving geometrically that developmental retardation or acceleration requires all trait allometries to tend, respectively, toward and outward isometry (Fig. 3). However, the purely geometric approach does not establish

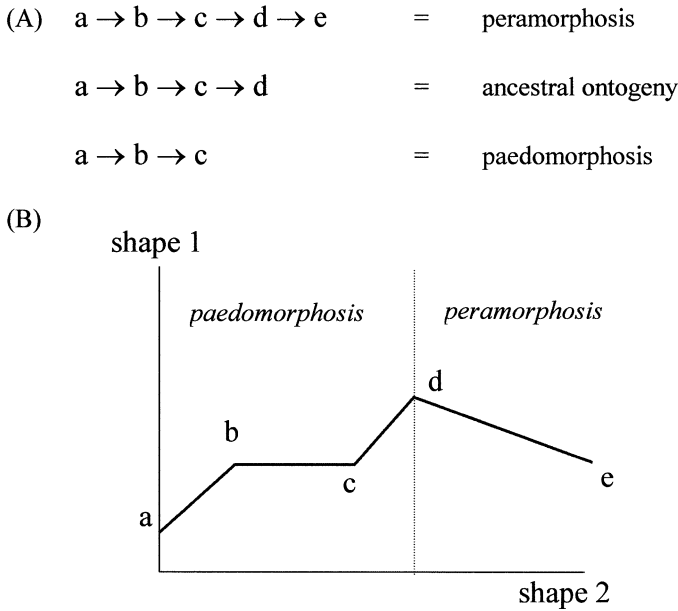


FIG. 2. (A) Heterochrony is the displacement of ontogenetic stages in evolutionary descendants. Peramorphic descendants (abcde) add new adult stages to the ancestral ontogeny, while paedomorphic descendants lose terminal stages. (B) In morphometric heterochrony, the same developmental sequence is now represented by a continuous shape trajectory σ_j , and morphological markers are defined by ratios or angles. Any point to the right (left) of d corresponds to peramorphic (paedomorphic) descendants.

the precise quantitative relationship between an observed change in developmental timing and the expected measurable variation in allometries, shape trajectories, and morphometric shape. To provide a tool for a quantitative testing of hypotheses of developmental dissociation by heterochrony, an analytic proof of Godfrey and Sutherland's (1996) geometric argument is presented below that applies to bivariate plots of shape against size, but it can be easily generalized to the global shape trajectory σ_j .

As a starting point, we take the example of neoteny. We want to know how a juvenile shape stage can be displaced to an adult size stage and generate paedomorphosis, and this can be done with the use of shape and allometric trajectories. This requires the definition of the curve for shape w/x against body size x , where w is any trait size. We can also assume the existence of an allometric relation between w and x . A classic criticism to the use of the allometric equation (Huxley 1932, P. 81) is that if the body size allometry of different parts of an organ is described by allometric (i.e., polynomial) equations with different allometric coefficients, then the whole organ (the sum of the parts) cannot itself be described by an allometric equation (i.e., the sum of polynomials with different coefficients is not a polynomial). But this difficulty is generally avoided by the exclusive examination of body parts or traits. Therefore, if the body size allometry of trait w is given by

$$w = a_w x^{k_w}. \tag{1}$$

Then the shape trajectory of w/x can be directly obtained by dividing both sides by x

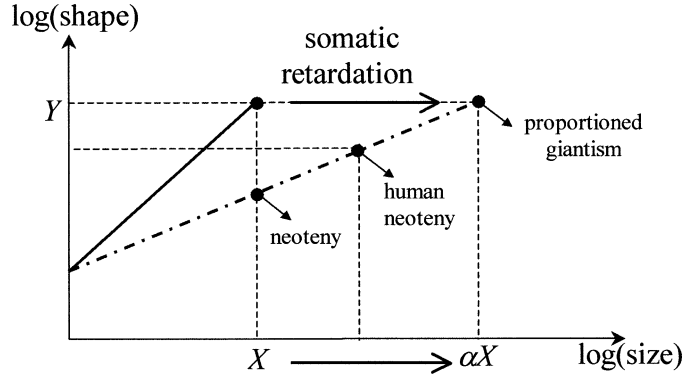


FIG. 3. Somatic retardation requires the displacement of any arbitrary shape stage Y from size stage X to αX in descendants (dashed line), where α stands for the retardation factor. If maturation is less retarded than development, the shape trajectory is truncated. Human neoteny, an intermediate outcome between classic neoteny and proportioned giantism, is a combination of larger body size, longer ontogeny, and paedomorphosis—according to Gould (1977), the landmarks of human evolution from apelike ancestors.

$$\left(\frac{w}{x}\right) = a_w x^{k_w - 1}. \tag{2}$$

The shape coefficient is therefore $k_\sigma = k_w - 1$. In the context of ontogenetic trajectories (more easily represented by bivariate logarithmic plots), retardation of development relative to growth defining neoteny can be modeled as the displacement of any given shape value Y from X to αX , where $Y = \log(y)$, $y = w/x$, $X = \log(x)$ and α is the displacement factor (the same conclusions follow if we replace x with αx and consequently X with $X + \log(\alpha)$; our choice was based on the ground of simplicity). This implies that ancestral shape values or stages w/x (e.g., a given ratio 0.5 observed when body size is x) are shifted to new body size stages in a descendant (i.e., the same ratio 0.5 now occurs at size $2x$). What is important is that we can show what happens to the shape trajectory of w/x as a function of α , or, in other words, we can calculate the effect of a change in the rate of development on morphometric shape. Using a double logarithmic plot, for any value $Y = \log(w/x)$ in the ancestor we have

$$\log\left(\frac{w}{x}\right) = k_\sigma X + A_w, \tag{3}$$

where $X = \log(x)$ and $A_w = \log(a_w)$ is the Y -intercept. As seen in Figure 3, in a given descendant undergoing such developmental retardation the trajectory has a different slope and corresponds to

$$\log\left(\frac{w}{x}\right) = k_\sigma^D X + A_w, \tag{4}$$

where k_σ^D is the new the shape coefficient. The relationship between the two coefficients can be found by taking any common Y -value in ancestor and descendant to define the equality

$$\log\left(\frac{w}{x}\right) = k_\sigma X + A_w = k_\sigma^D X^D + A_w = k_\sigma^D \alpha X + A_w. \tag{5}$$

From the above, we conclude that the new shape coefficient will be

$$k_{\sigma}^D = \frac{k_{\sigma}}{\alpha}. \quad (6)$$

In polynomial form, the descendant curve is therefore

$$\left(\frac{w}{x}\right) = a_w x^{k_{\sigma}/\alpha}. \quad (7)$$

This demonstrates that, if $0 < \alpha < 1$ (acceleration of development) we have $k_{\sigma}^D > k_{\sigma}$. For $\alpha > 1$ (retardation), k_{σ}^D tends to zero and shape curves become flatter, and an infinite α implies a flat trajectory or no change in shape (absolute retardation). We can also show what happens to measured allometries when neoteny or retardation occurs. Because $k_{\sigma} = k_w - 1$, the new allometric coefficient for w is simply

$$k_w^D = \frac{k_w - 1}{\alpha} + 1. \quad (8)$$

It follows that k_w^D is one for infinite α and tends to infinite when α approaches zero. This proves analytically that allometric coefficients tend toward or outward isometry as a result of developmental retardation or acceleration, respectively. Therefore, the equations above allow for the calculation of new shape and allometric curves predicted by hypotheses of neoteny or acceleration.

It could be argued that the results above apply only to shape measures involving x , or body size. However, they apply to shape indexes calculated as a proportion between any two traits w and z (e.g., body width and length). Suppose we want to obtain the ancestral shape trajectory for w/z . If trait allometries are

$$w = a_w x^{k_w} \quad \text{and} \quad (9)$$

$$z = a_z x^{k_z}, \quad (10)$$

then dividing w by z gives the curve for the proportion w/z

$$\left(\frac{w}{z}\right) = \frac{a_w}{a_z} x^{k_w - k_z}. \quad (11)$$

Additionally, it is possible to calculate the allometric relationship between the two traits. Body size allometry of z can also be written as

$$\left(\frac{z}{a_z}\right)^{1/k_z} = x. \quad (12)$$

Substituting in the allometric equation for w , the two characters will be related as

$$w = a_w \left(\frac{z}{a_z}\right)^{k_w/k_z} = (a_w a_z^{-k_w/k_z}) (z)^{k_w/k_z}. \quad (13)$$

Now we want to know how the displacement factor α affects the shape trajectory w/z and the allometric relationship between w and z in a putative heterochronic descendant. First, because allometric coefficients change from k in the ancestor to $k - 1/\alpha + 1$ in the descendant, the new allometric curves for w and z following dissociation between development and growth will be given by

$$w = a_w x^{[(k_w - 1)/\alpha] + 1} \quad \text{and} \quad (14)$$

$$z = a_z x^{[(k_z - 1)/\alpha] + 1}. \quad (15)$$

Consequently, the new descendant shape curve for w/z is

$$\left(\frac{w}{z}\right) = \frac{a_w}{a_z} x^{(k_w - k_z)/\alpha}. \quad (16)$$

Therefore, the shape coefficient of w/z is the ancestral coefficient divided by α . As previously shown for the case of w/x , flattening or steepening of the shape curve will be respectively observed for $\alpha > 1$ or $0 < \alpha < 1$. Additionally, by using the new equations for body size allometries of w and z calculated above, it can be shown that the relation between w and z also changes in the descendant and is given by

$$\begin{aligned} w &= a_w \left(\frac{z}{a_z}\right)^{\{[(k_w - 1)/\alpha] + 1\} / \{[(k_z - 1)/\alpha] + 1\}} \\ &= a_w (a_z)^{-(k_w - 1 + \alpha)/(k_z - 1 + \alpha)} (z)^{(k_w - 1 + \alpha)/(k_z - 1 + \alpha)}. \end{aligned} \quad (17)$$

This means that as α tends to infinite (meaning infinite retardation), the allometric coefficient relating the two traits also tends to one, as previously shown for the relationship between w and x . This demonstrates that the equations deduced above estimate changes in allometric and shape curves for any traits and are therefore appropriate for testing hypotheses of developmental retardation or acceleration in general.

We are left with a final question, namely how is it possible to prove analytically that such dissociations regulated by the factor α generate paedomorphosis and peramorphosis. Given that heterochrony requires the preservation of an ancestral shape trajectory, such proof would equal identifying which property of the ontogenetic trajectory remains invariant under shape-size dissociations. This property can be found by using the equations deduced above for the shape trajectories of w/x and z/x :

$$\left(\frac{w}{x}\right) = a_w x^{k_w - 1} \quad \text{and} \quad (18)$$

$$\left(\frac{z}{x}\right) = a_z x^{k_z - 1} \leftrightarrow \left(\frac{z}{a_z x}\right)^{1/(k_z - 1)} = x. \quad (19)$$

We can therefore calculate the ancestral relationship between two proportions w/x and z/x , or the trajectory of the proportions relative to one another. By substituting the second into the first equation, the shape versus shape trajectory is shown to be

$$\left(\frac{w}{x}\right) = a_w (a_z)^{-(k_w - 1)/(k_z - 1)} \left(\frac{z}{x}\right)^{(k_w - 1)/(k_z - 1)}. \quad (20)$$

The next step is to compare this trajectory with the trajectory in a descendant. If now we take the descendant shape trajectories for w/x and z/x as a function of the factor α :

$$\left(\frac{w}{x}\right) = a_w x^{(k_w - 1)/\alpha} \quad \text{and} \quad (21)$$

$$\left(\frac{z}{x}\right) = a_z x^{(k_z - 1)/\alpha}. \quad (22)$$

The following shape versus shape trajectory is obtained

$$\begin{aligned} \left(\frac{w}{x}\right) &= a_w(a_z)^{-[(k_w-1)/\alpha]/[(k_z-1)/\alpha]} \left(\frac{z}{x}\right)^{[(k_w-1)/\alpha]/[(k_z-1)/\alpha]} \\ &= a_w(a_z)^{-(k_w-1)/(k_z-1)} \left(\frac{z}{x}\right)^{(k_w-1)/(k_z-1)}, \end{aligned} \tag{23}$$

which is similar to the equation for the ancestor (the same is true for shape indexes w/z and r/s , where w , z , r , and s are any body measurements). Therefore, contrary to allometric relationships and to correlations between shape and body size, the relationship between any two developing morphometric shapes is not affected by the scaling factor α , and the total n -dimensional shape trajectory in the subspace σ_j remains the same. Consequently, if bivariate projections of the total shape trajectory (i.e., plots of shape vs. shape) are not coincident in putative ancestor and descendants (except for the terminal regions supposed to undergo extension or truncation), the hypothesis of heterochrony by developmental retardation (paedomorphosis) or acceleration (peramorphosis) must be discarded.

Notice that somatic retardation was defined by Gould (1977) as a delay of development relative to both growth and maturation, and for this reason the same conclusions hold for shape-time dissociations. For negatively allometric traits, the hypothesis implies an increase in growth coefficient (so that trait growth catches up with body growth, thus approaching isometry), and a decrease for positively allometric traits. However, the correlations between shapes remain the same. It follows that if the shape versus shape trajectory is preserved, it will be invariant under linear transformations in the time axis. In other words, Rice's (1997) definition of heterochrony resembles our criterion, insofar as polynomial functions are used to describe trait growth and shape development.

The two possibilities outlined in this section (ontogenetic scaling and dissociation between shape and size or time) also circumscribe the descriptive scope of paedomorphosis and peramorphosis and therefore of morphometric heterochrony. If a disruption of the shape correlation structure occurs, ancestral ontogenetic stages are no more recognizable in a putative descendant, and the global shape trajectory of the latter follows new paths. This excludes from heterochrony the dissociations of growth fields proposed by Godfrey and Sutherland (1996) and any other changes in patterns of relative growth. Huxley (1932, pp. 239–240) had already “grasped that most examples of recapitulation [peramorphosis] constitute simply one side of a more general problem—the problem of altering the relative rates of growth and of other processes within the body.” Despite the explicit reference to Haeckel's biogenetic law, the comment applies perfectly to heterochrony.

Finally, we stress that the criterion defined above—preservation of all shape-shape correlations and differential extension of the shape trajectory—is a necessary but not sufficient condition for heterochrony. Even if a common shape trajectory is conserved in two taxa supposedly connected by a heterochronic process, there still remains the possibility that the two trajectories are just coincident. But the primary criterion deduced (or made explicit) in this section is useful because it suffices to refute some key arguments for heterochrony as a main player in evolution.

For example, we show in the following sections that neither human evolution nor human encephalization can be described by the morphological markers of heterochrony.

Somatic retardation and sequential hypermorphosis

Many distinctive characteristics of modern human morphology have been repeatedly attributed to some form of heterochronic process. One such suggested process is the mechanism of somatic retardation discussed above (Gould 1977; Godfrey and Sutherland 1996), where developmental retardation is more intense than maturation retardation thus giving rise to paedomorphosis (Fig. 3). One of its manifestations would be human neoteny, or $\Delta = (+\delta_s, -\delta_\sigma, +\delta_t)$ in the clock model description, supposedly engendering paedomorphosis, large body size (derived from a longer ontogeny), and late maturation, three key features of humans in comparison with its closest evolutionary relatives. According to the hypothesis, the large adult brain of *Homo sapiens* is an example of paedomorphosis (a retention of an ancestral infantile or juvenile brain size/body size ratio).

The argument originally relied on the existence of distinct phases of brain growth, in particular, a fetal phase of intense growth versus a late phase of modest growth (Gould 1977). Based on the results demonstrated above, it is possible to predict what should happen to brain allometry under a general retardation of development relative to maturation and growth (Figs. 4, 5). Somatic retardation implies flatter shape curves and allometric coefficients closer to isometry, or in the particular case of traits with distinct growth phases such as the brain, flatter shape curves and allometric coefficients closer to isometry in each of the proportionally extended growth phases. Additionally, somatic retardation generates paedomorphosis because maturation is not equally delayed and development is truncated, meaning that either shortening or complete disappearance of terminal developmental phases should be observed in putative descendants.

Before testing the predictions of human neoteny against the known evidence for human brain growth ontogeny and evolution, it is necessary to define the alternative hypothesis of sequential hypermorphosis (McKinney and MacNamara 1991), or a delay in offset time of each growth phase while preserving their original allometric coefficients and shape slopes. The distinction between the two models is important (Godfrey et al. 1998), because they imply different interpretations of developmental delay. When the distinction is made, all the evidence originally presented for human neoteny is in fact evidence for sequential hypermorphosis. For example, Gould (1977; P. 365) postulated that “[human] retarded development carried a set of potential consequences with it: prolongation of fetal growth rates leading to larger sizes and the retention of juvenile proportions [in adults],” which does not correspond to the requirements of changes in growth rates toward isometry and the loss of late growth stages.

The same is observed in the particular case of human encephalization. Based on the well-known allometric curve for the primate brain (Count 1947) characterized by a first phase of positive allometry and a second period of negative allometry, and relying on the comparative study of human and

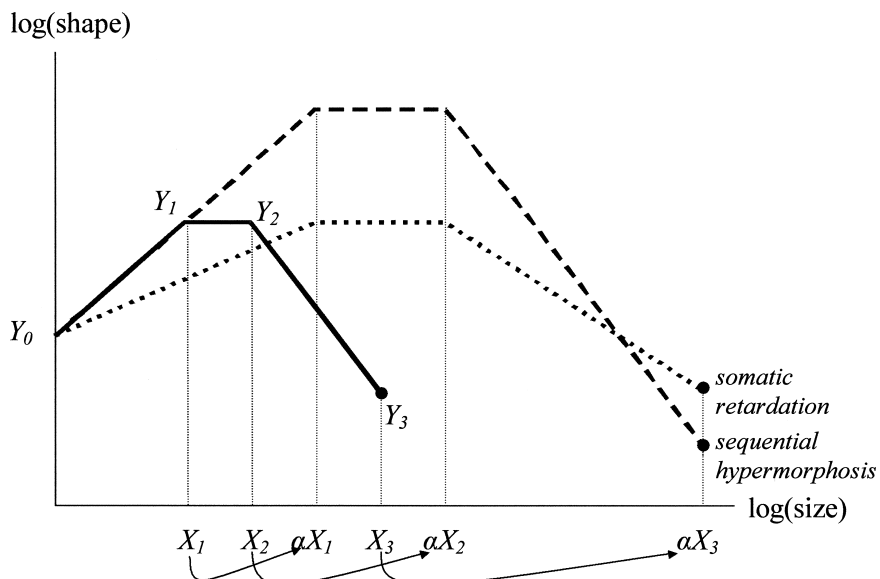


FIG. 4. Somatic retardation and sequential hypermorphosis generate distinct morphometric outcomes. The ancestral ontogeny (Y_{0123}) is represented by the solid line. Somatic retardation implies the displacement of each shape landmark Y_i from size X_i to αX_i where Y is $\log(\text{shape})$, X is $\log(\text{body size})$, and α ($= 2$ in the example) is the retardation factor. Therefore, shape slopes for each phase change and approach zero. Sequential hypermorphosis is the delay in the offset point of each phase, with shape slopes remaining the same.

chimpanzee brain growth by Rice (1997) and Vrba (1998), Gould (2000, p. 246) concluded that if “all descendant growth phases are extended by the same factor while each remains at the ancestral growth rate,” and “if an admittedly paedomorphic result occurs by a prolongation of juvenile growth rates into later stages of a descendant’s ontogeny . . . then the heterochronic result can only be called paedomorphosis by neoteny.” But as seen above, and however counterintuitive it may sound, encephalization by somatic retardation implies both a decrease in the positive fetal brain growth rates and an increase of negatively allometric later growth rates. The empirical studies of primate and human

growth patterns by Count, Vrba, and Rice seem to refute the hypothesis of human paedomorphosis and encephalization by somatic retardation, therefore strengthening the case for the alternative model of sequential hypermorphosis. As seen below, this implies another difficulty for the theory of heterochrony, because sequential hypermorphosis does not produce heterochronic morphologies.

Sequential hypermorphosis and human encephalization

While McKinney and McNamara never used sequential hypermorphosis to make exact quantitative predictions about

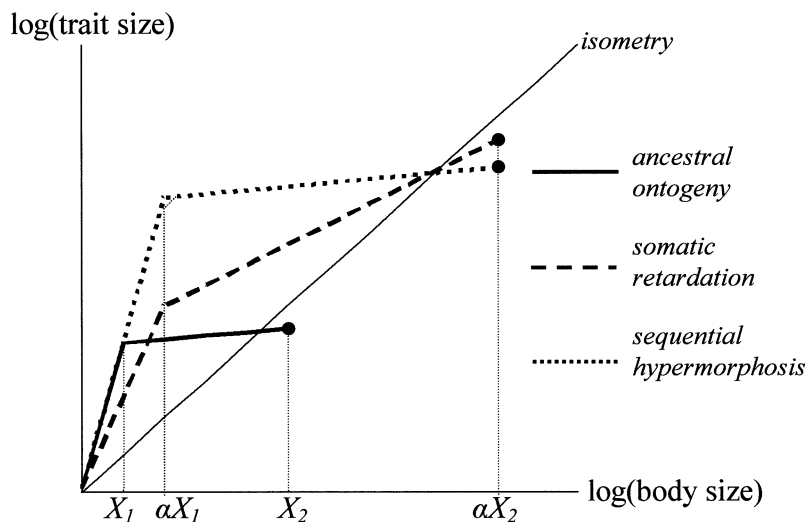


FIG. 5. Distinct behavior of allometric coefficients and adult trait sizes under somatic retardation or sequential hypermorphosis. In the example, trait y is positively allometric ($k_y > 1$) in the first growth phase and negatively allometric ($0 < k_y < 1$) in the second. Gould’s (1977) somatic retardation implies a movement of both early and late allometric coefficients toward isometry, so that the first becomes less positive and the second less negative. Sequential hypermorphosis is the simple extension of original trait allometries.

the evolution of human size and shape, Vrba (1994, 1998) formulated a specific hypothesis where all brain growth phases of a putative chimplike human ancestor are prolonged linearly. This more stringent version of sequential hypermorphosis was proposed as an explanation for a supposed mosaicism of paedomorphic and peramorphic traits and punctuated evolution of *H. sapiens* from remote australopithecine ancestors. Vrba showed that a multiphasic polynomial curve with four stages gives the best linear fit to human brain growth data (in comparison with exponential or logistic models). Vrba’s proportionate or linear sequential hypomorphosis was then simulated, and the resulting transformed human curve was shown to match adequately the partially available data on chimpanzee brain growth, generating an estimate of the magnitude of linear increase in the duration of growth phases. In the following, we reveal some of the additional consequences of Vrba’s model (e.g., that the value of growth phase extension can be calculated a priori) and throw light on its relationship to heterochrony.

Our starting point is the multiphasic polynomial growth curve proposed by Vrba (1998). To simplify the argument, it will be represented in a log-log plot and with two- phases. In this case, final or adult size for trait y can be written as

$$Y_2^A = B_y + k_1^y T_1 + k_2^y (T_2 - T_1), \tag{24}$$

where y is trait size, $Y = \log(y)$, k_i^y stands for the growth coefficient of trait y during phase i , t is time, T equals $\log(t)$, t_1 is the time at breakpoint between the phases, t_2 is the time at the end of phase 2, b_y is the intercept, and B_y equals $\log(b_y)$. Adult or final trait size is then given by the equivalent polynomial function

$$y_2^A = (b_y t_1)^{k_1^y} \left(\frac{t_2}{t_1} \right)^{k_2^y}. \tag{25}$$

Generalizing for an arbitrary multiphasic pattern of n stages, one has

$$Y_n^A = B_y + k_1 T_1 + \sum_2^n k_i^y (T_i - T_{i-1}) \tag{26}$$

that in polynomial form corresponds to

$$y_n^A = (b_y t_1)^{k_1^y} \prod_{i=2}^n \left(\frac{t_i}{t_{i-1}} \right)^{k_i^y}. \tag{27}$$

With $n = 4$, the equation above corresponds to Vrba’s (1998) multiphasic model of human and chimpanzee brain growth. Now it is easy to calculate the effect of a linear extension of growth phases or sequential hypermorphosis by multiplying each stage marker t_i by a factor α (greater than one for prolongation of stages). Adult trait size y_n^D in the hypothetical descendant is then

$$\begin{aligned} y_n^D &= (b_y \alpha t_1)^{k_1^y} \prod_{i=2}^n \left(\frac{\alpha t_i}{\alpha t_{i-1}} \right)^{k_i^y} = \alpha^{k_1^y} (b t_1)^{k_1^y} \prod_{i=2}^n \left(\frac{t_i}{t_{i-1}} \right)^{k_i^y} \\ &= \alpha^{k_1^y} y_n^A \end{aligned} \tag{28}$$

or $Y_n^D = Y_n^A + k_1^y \log(\alpha)$. In other words, one needs only the growth coefficient for the first phase to calculate a priori the effect of linear sequential hypermorphosis on adult trait sizes. It is a mathematical property of composite polynomial curves

that the number and duration of phases does not need to be known to simulate the effects of proportionate sequential hypermorphosis. Now, if we follow Vrba (1998) and take chimpanzees as a model for a human australopithecine ancestor, we can use the equation above to calculate the time increase in the chimpanzee ontogeny needed to generate a *H. sapiens*’ brain size. Using $y_n^A = 410.3$ g as the chimpanzee brain size (Harvey et al. 1988), $y_n^D = 1349$ g as the average human adult brain size, and $k_1^y = 4.74$ as the first coefficient of human brain growth (the two latter estimates from Vrba 1998), we have $\log(1349) = \log(410.3) + 4.74 \log(\alpha)$, or $\alpha = 1.29$ from chimpanzee to man or inversely $\alpha = 0.78$ from man to chimpanzee.

Interestingly, this is exactly the value for α empirically obtained by Vrba (1998) and that produces the best statistical fit between the transformed (hypomorphic) human curve and the chimpanzee curve. Unfortunately, data for the crucial first and fully intrauterine chimpanzee phase are missing, so that the chimpanzee curve is a hybrid borrowing the first phase from the human curve. Despite the match between predicted and observed values, the hypothesis of sequential hypermorphosis from chimpanzee to man cannot be fully corroborated without the confirmation of the existence of this first phase in chimpanzees. Rice (2001) used a different method and obtained the also similar value of $\alpha = 1.22$, but except for two points the data used in his study do not cover the fetal phase of chimpanzee brain growth. Both empirical and theoretical results seem to indicate that sequential hypermorphosis is the correct model of human brain size evolution.

A final and important question is whether sequential hypermorphosis generates paedomorphosis or peramorphosis. Vrba (1994, 1998) tried to prove that traits whose growth rates decrease during ontogeny (such as the brain) become paedomorphic (i.e., both absolutely and relatively larger) following sequential hypermorphosis, whereas traits showing increasing growth rates during ontogeny (such as the legs) would be peramorphic (absolutely larger but relatively smaller). A difficulty in such argument is that paedomorphosis or peramorphosis refer to relative size and therefore cannot be assessed purely from trait growth curves. For that reason, it is necessary to introduce body growth curves into the analysis and to calculate the effect of sequential hypermorphosis not only on brain size but also on a measure of encephalization (e.g., the ratio of brain size to body size). To simplify the argument, we now define a multiphasic polynomial function to describe body size growth (instead of using logistic or Gompertz curves as is usually done):

$$x_n^A = (b_x t_1)^{k_1^x} \prod_{i=2}^n \left(\frac{t_i}{t_{i-1}} \right)^{k_i^x}. \tag{29}$$

Our measure of encephalization can be defined simply as the ratio of brain size to body size y_n^A/x_n^A , or $Y_n^A - X_n^A$ in logarithmic form. We want to know what happens to this ratio in a descendant. As already shown, following sequential hypermorphosis we expect (no matter the number of growth phases) adult values of brain size and body size to become respectively $Y_n^D = Y_n^A + k_1^y \log(\alpha)$ and $X_n^D = X_n^A + k_1^x \log(\alpha)$. Therefore, the descendant value $Y_n^D - X_n^D$ will be

$$Y_n^D - X_n^D = Y_n^A - X_n^A + (k_1^y - k_1^x) \log(\alpha). \tag{30}$$

In other words, the change in relative brain size is given by the factor $(k_1^y - k_1^x \log(\alpha))$. The equation above shows that sequential hypermorphosis ($\alpha > 0$) produces a relatively larger brain because $k_1^y > k_1^x$, i.e., because there is a phase of positive allometry early in ontogeny so that the brain grows more rapidly than the body.

It is easy to show that an opposite outcome results from classic hypermorphosis (or extension of the last growth phase). In the simplest case where there are two growth phases and a common breakpoint for both brain and body size curves, one has the same ancestral brain growth and body growth curves calculated above

$$Y_2^A = B_y + k_1^y T_1 + k_2^y (T_2 - T_1) \quad \text{and} \quad (31)$$

$$X_2^A = B_x + k_1^x T_1 + k_2^x (T_2 - T_1). \quad (32)$$

But under classic hypermorphosis, t_1 remains the same in the descendant, whereas t_2 only is multiplied by a factor α (so that T_2 becomes $T_2 + \log(\alpha)$ in log form). Therefore, adult brain size and body size become, respectively,

$$\begin{aligned} Y_2^D &= B_y + k_1^y T_1 + k_2^y [T_2 + \log(\alpha) - T_1] \\ &= B_y + k_1^y T_1 + k_2^y (T_2 - T_1) + k_2^y \log(\alpha) \quad \text{and} \quad (33) \end{aligned}$$

$$\begin{aligned} X_2^A &= B_x + k_1^x T_1 + k_2^x [T_2 + \log(\alpha) - T_1] \\ &= B_x + k_1^x T_1 + k_2^x (T_2 - T_1) + k_2^x \log(\alpha). \quad (34) \end{aligned}$$

Consequently, the ratio y/x in the adult descendant will be

$$Y_2^D - X_2^D = Y_2^A - X_2^A + (k_2^y - k_2^x) \log(\alpha). \quad (35)$$

The factor $(k_2^y - k_2^x) \log(\alpha)$ indicates that the change in relative brain size following hypermorphosis depends only upon the second coefficient, contrary to the case of linear sequential hypermorphosis where the effect depends only on the first growth coefficient. Late in human ontogeny, body growth predominates over brain growth (i.e., brain allometry is negative), which is why classic (versus sequential) hypermorphosis would cause a decrease in the ratio of brain size to body size. The conclusion also holds for any two traits y and w (not necessarily body size) and for growth curves with more than two phases (because only the coefficients of the first and last phases, respectively, are used in the calculations).

There is a final difference between classic and sequential hypermorphosis—their relationship with heterochrony. From the very definition of sequential hypermorphosis, it can be seen that it does not preserve a global ontogenetic trajectory in the descendant, given that the extension of ontogenetic stages is not accompanied by a trend toward isometry. In Figure 6 the effect of sequential hypermorphosis on correlations between shapes is compared with the alternative heterochronic hypotheses of somatic retardation and classic hypermorphosis. Such effect cannot be described by either pedomorphosis or peramorphosis, being “something new” (sensu Godfrey and Sutherland 1996). Therefore, sequential hypermorphosis is an example of how morphometric evolution can be modeled without any necessary reference to heterochronic categories.

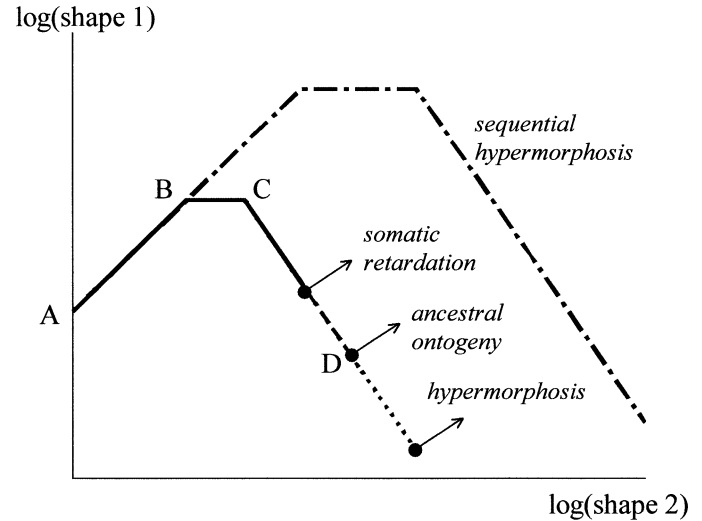


FIG. 6. Sequential hypermorphosis generate neither pedomorphosis nor peramorphosis. The ancestral shape trajectory is represented by the sequence ABCD. Somatic retardation produces pedomorphic descendants as it leads to a truncation of the ancestral trajectory. Hypermorphosis is an example of ontogenetic scaling, and as such it gives rise to peramorphic descendants by extension of ancestral shape trajectories. Sequential hypermorphosis does not generate heterochronic descendants because the ancestral trajectory shifts to a different region of the shape space.

DISCUSSION

The merit of morphometric heterochrony was to recover growth as a key to evolutionary analysis, which involved a shift toward measurable traits, thereby supplying morphometric studies with the theoretical framework they had been partially lacking. Morphometric heterochrony was originally formulated as a challenge and alternative to mainstream evolutionary biology, as observed in its definition as “one particular kind of evolution operating with a relative frequency . . . change by shifts in developmental timing for features already present in ancestors, thus illustrating evolution within constraints or channels of past inheritance” (Gould 2000, P. 244). Many believe that the claim for this kind of evolution does not rest upon firm empirical ground, but the use of allometry as a descriptive metric for growth has led to more widespread acceptance. Ironically, a full account of this metric, embodied in the formalism of ontogenetic trajectories, shows that the morphological phenomena described by heterochrony (pedomorphosis and peramorphosis) are a limited subset of the possible outcomes of changes in growth patterns, as recognized by Gould (1977, 2000), Godfrey and Sutherland (1996), and Rice (1997).

We attempted to establish mathematically such limits from the point of view of homology in the context of morphometric traits and allometric trajectories. In cladistic analysis, it is generally recognized that the assessment of homology involves two steps (de Pinna 1991; Brower and Schawaroch 1996). Primary homology is a conjecture that the similarity between two characters is evidence of phylogenetic kinship. The conjecture becomes secondary homology if it is corroborated by other homologies in a phylogenetic analysis. After excluding phenetic similarity between adults as a valid cri-

terion, we offered an explicit definition of primary homology for heterochrony as a coincidence of the shape trajectory in putative ancestors and descendants, followed by its truncation or extension in the descendant. As an example of how the criterion can be applied, we showed that Gould's conjecture of human neoteny stands the test of primary homology but not of empirical evidence.

The limited descriptive scope of paedomorphosis and peramorphosis indicates that morphometric evolution can be studied without reference to heterochronic markers, as shown in the analysis of the hypothesis of human encephalization by sequential hypermorphosis. Vrba's (1998) linear version of sequential hypermorphosis seems to be a convincing model of how human brain size could have evolved from an australopithecine-like ancestor, as shown independently by Rice (2001). But, from a morphological point of view, sequential hypermorphosis produces something new rather than heterochronic descendants, and as such, the model does not need either to rely on putative similarities between adult humans and juvenile apes (e.g., in brain size/body size ratio) or to postulate any claims of primary homology.

Satisfying a criterion of primary homology also implies that ontogenetic trajectories should be incorporated into comprehensive phylogenetic contexts. Grounded on the primary homology between the human and chimpanzee brain growth curves (but not between their relative brain sizes) and on the derived character of such curve in a phylogenetic analysis (Rice 2001) including also nonhominoid primates (secondary homology), sequential hypermorphosis becomes a successful ontogenetic model for how changes in growth parameters can generate a large (but neither paedomorphic nor peramorphic) human brain. As such, it illustrates Huxley's (1932) point that the evolution of growth patterns generates more outcomes than heterochronic markers can possibly describe. We believe that the acceptance of this view would help morphometric studies to address more precisely and broadly the relationship between growth, development, and evolution.

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