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**Chapter 7**

# The Role of Heterochrony in Primate Brain Evolution

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Nearly everyone agrees that heterochrony has played some important role in human evolution. The exact nature of this role and, in particular, the type or types of heterochrony involved are not so well agreed upon. The notion of humans as neotenic apes has a certain poetic resonance (as depicted, e.g., in Aldous Huxley's *After Many a Summer Dies the Swan* [1939]) and makes sense of the physical resemblance between infant apes and humans. On the other hand, humans are larger and longer lived than our closest relatives, the chimpanzees, implying some sort of hypermorphosis (Shea 1988; McKinney and McNamara 1991). Distinguishing between these options has been made difficult by the fact that the concept of heterochrony is used alternately as a catch-all description for any morphological change that involves a change in rate or timing (i.e., any morphological change) and as a specific theory that identifies a well-defined set of mechanistic transformations that may underlie some, but not all, morphological evolution.

Theoretical concepts in science allow us to represent the world in ways that draw our attention to fundamental relationships between different phenomena and give us a way to bring rigorous logic or mathematics to bear so as to discover new phenomena and direct further empirical research. If the concept of heterochrony is to serve as a theory in this sense, and I think that it should, then we must be able to distinguish between what is and is not heterochrony and have some notion of what it means biologically to make such a distinction. I addressed this elsewhere (Rice 1997) by showing that, if we define heterochrony as a uniform change in the rate or timing of some developmental process, with no other internal change to





that process, then the traditional categories of heterochrony correspond to meaningful biological transformations that we can test for by comparing ontogenetic trajectories.

In this chapter, I develop a statistical test for heterochrony based on this definition and then apply this test to the trajectories for brain growth in humans and some other primates. The differences between human and chimpanzee brain growth are largely a result of uniform changes in rate and timing, thus heterochrony. Compared to other primates, though, humans and chimps show a novel phase of brain growth that is not a simple heterochronic modification of an ancestral trajectory. I also compare the overall growth of the body in humans and chimpanzees and show that heterochrony seems to be a factor here also, but with different kinds of transformations acting at different stages of growth.

## Analyzing Ontogenetic Trajectories

Figure 7.1 shows the transformations that correspond to different types of heterochrony (see Rice 1997 for justification and derivation). I refer to two trajectories as being *commensurate* if we can superimpose one on the other by applying some combination of these transformations. If two trajectories are commensurate, then we can infer that the difference between the two growth processes *could* be accounted for by a uniform change in rate or timing.

By contrast, if two trajectories cannot be related by some combination of these transformations, then we can infer that there must have been some change in the nature of the interactions underlying the growth process, not just a change in the rate or timing of that process. This definition of the types of heterochrony is compatible with that of Alberch et al. (1979), with one modification. In Alberch et al., the endpoint of the trajectory was held fixed in time unless there was progenesis or hypermorphosis. I am allowing the endpoint to shift if the entire growth process is slowed down (neoteny) or sped up (acceleration). This will be important in the discussion of whole-body growth.

Because this definition assigns so much significance to the superposition of trajectories and because data for actual trajectories are likely to be noisy, we seek a statistical test to compare trajectories and potentially reject a hypothesis of heterochrony. Often, ontogenetic trajectories must be inferred from clouds of points, each of which represents a separate individual (Fig.

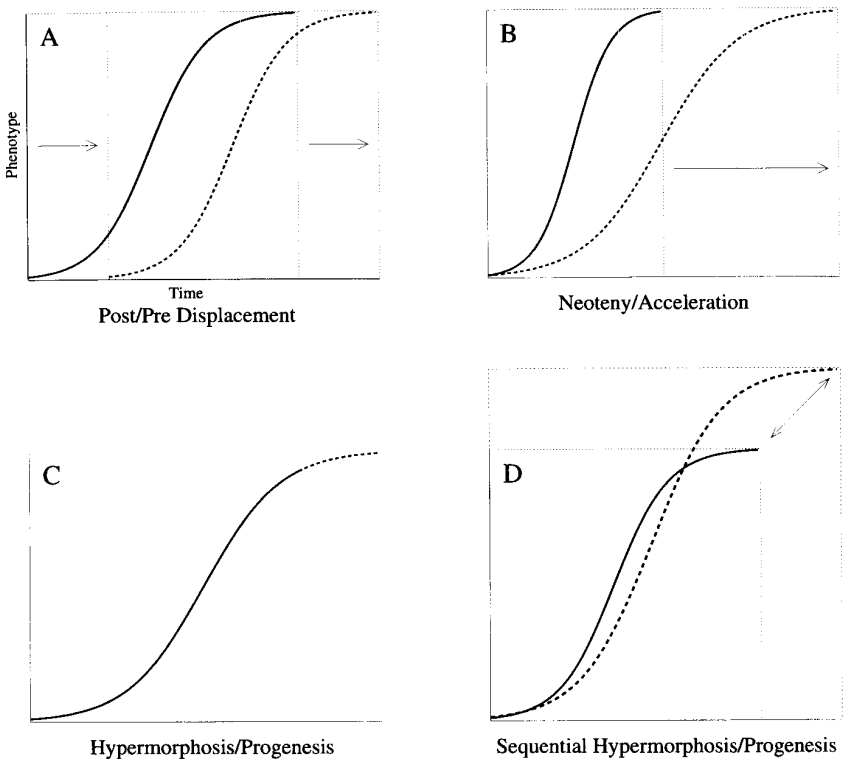


**Fig. 7.1.** Transformations of ontogenetic trajectories corresponding to different types of heterochrony. *A:* Shifting the entire trajectory to the *right* constitutes *postdisplacement;* the converse is *predisplacement. B:* A uniform stretching of the time axis, as if each value on that axis were multiplied by a constant greater than 1, constitutes *neoteny.* Contracting the time axis by multiplying each time value by a constant between 0 and 1 produces *acceleration. C:* Allowing the trajectory to continue beyond its ancestral termination is *hypermorphosis.* Stopping it before this point is *progenesis. D: Sequential hypermorphosis* involves stretching both the time and phenotype axes by the same amount.

7.2). After overlapping the trajectories by applying some combination of the transformations in Figure 7.1 to one of them, we need to decide whether they are commensurate. One way to do this is to fit a curve through one of the sets of points (species A), making sure that the points representing species A are symmetrically distributed around this curve, then to check the distribution of points representing species B around the same curve. If the two trajectories are commensurate, implying heterochrony, then each point representing species B should have a probability of 0.5 of lying above (or below) the line. If the points represent different individu-



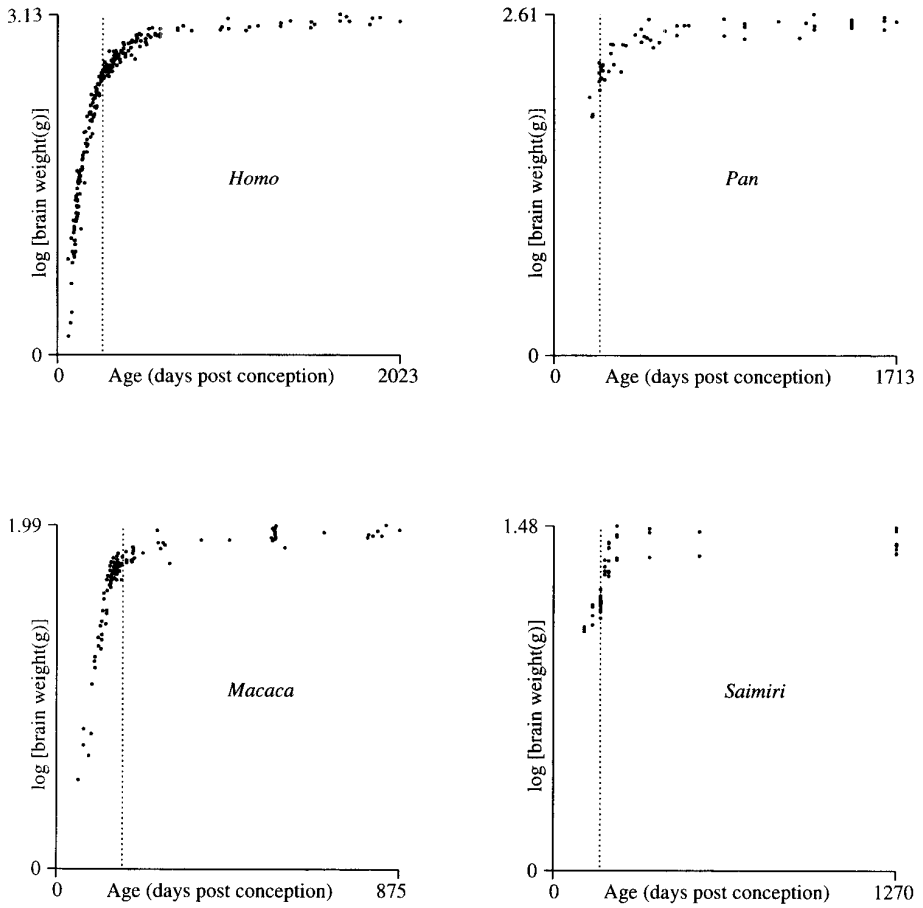


**Fig. 7.2.** Ontogenetic trajectories for log of brain weight (in grams) with time for human, chimpanzee (*Pan paniscus*), macaque (*Macaca mulatta*), and squirrel monkey (*Saimiri sciureus*). The bimodal adult trajectory for squirrel monkeys is a result of sexual dimorphism. The *dotted lines* represent age at birth. *Sources:* Human data are from Dobbing and Sands 1973 and Blinkov and Glezer 1968. Chimpanzee data are from Vrba 1998 and Shantha and Manocha 1969. Macaque data are from Cheek 1975. Squirrel monkey data are from Manocha 1978. Except for Dobbing and Sands 1973 and Manocha 1978, the data were calculated from digitized images of plotted points.

als, then each is an independent event, and we can think of the sequence of points lying above or below the line as analogous to a sequence of coin flips.

This suggests a test: define a run of length $x$ as a set of $x$ points for one species, adjacent to one another along the time axis, that all lie on the same

side of the fitted curve for the other species. We seek the probability of encountering a run of length $x$ or longer if the two trajectories are in fact commensurate.

Let $\Pi_{x,n}$ be the probability that there is *no* run of length $x$ or longer in a set of $n$ residuals. Then,

$$\Pi_{x,n} = 1 \text{ for } n < x, \Pi_{x,x} = 1 - \frac{1}{2^{x-1}}$$

Values of $\Pi_{x,n}$ can be found from these relations and the recursion:

$$\Pi_{x,n} = \Pi_{x,n-1} - \frac{1}{2^x}\Pi_{x,n-x}$$

(see the appendix for the derivation). Thus, if we observe a run of $x$ in set of $n$ points, then

$$p = 1 - \Pi_{x,n}$$

gives the probability of observing such a run (or a longer one, since a run of $>x$ contains a run of $x$) if the trajectories were commensurate.

The most problematic step in this process is that of fitting a curve to one of the datasets. The best way to do this depends on the nature of the data. In the analysis that follows, I first decide which dataset will be transformed and then find the distance between each point in this set and the regression line through the ten points in the other dataset that surround it (five on each side along the time axis); only those points for which this can be done are considered. This gives the necessary result that, when any of the datasets is compared with itself in this way, the residuals are evenly distributed around zero. I refer to this distance, from a point for one species to the local regression line for the other species, as the *between-species residual* for that point.

### Brain Growth

Figure 7.3*A* shows growth trajectories for humans and chimpanzees (*Pan paniscus*) and, below these, a plot of the between-species residuals representing the distance between each chimpanzee data point and the mean of the human points at that age (calculated by taking the regression through ten



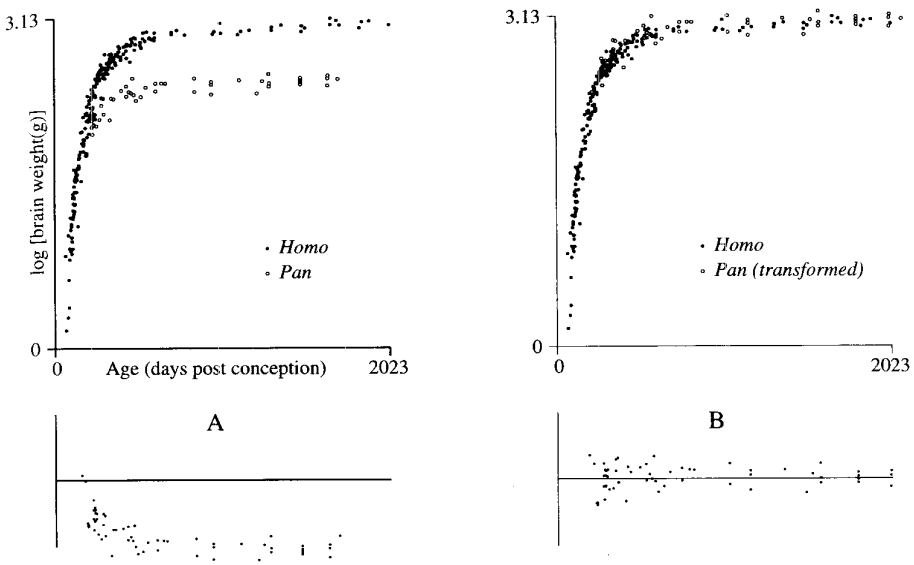


**Fig. 7.3.** Comparison of human and chimpanzee brain growth trajectories. The plots below the trajectories show the distribution of residuals representing the distance between each point on the chimpanzee curve and the local best-fit regression line through human data. *A:* Untransformed trajectories. *B:* The chimpanzee trajectory has been transformed by sequential hypermorphosis, multiplying both the phenotype and time axes by 1.22. With this transformation, the residuals come to be evenly distributed around the human curve. There are fifty-four residual points, and the longest run on the same side of the human line is four.

points of the human data, five on either side of the chimpanzee point being considered). Figure 7.3*B* presents the same comparison after the chimpanzee data have been transformed by sequential hypermorphosis. The between-species residuals are now evenly distributed around the human mean. Out of fifty-four points, the longest run of points on the same side of the regression line is four. Since the probability of getting a run of four or more if the transformed chimpanzee points were drawn from the same distribution as the human points is >0.9 and since the common ancestor had a brain closer in size to that of a chimpanzee, we conclude that the difference between these two trajectories is explained by sequential hypermorphosis along the human lineage. Vrba (1998) arrives at the same conclusion.

Showing that two ontogenetic trajectories are not commensurate is sufficient to show that there must have been some change in the nature of the developmental process underlying the characters under study, not just a uni-

form rate change. The converse, however, is not true. Commensurate trajectories *could* be related by some sort of heterochrony, but it is always possible that changes internal to the developmental process might not significantly alter the overall trajectory. This said, the match between the human and chimpanzee trajectories indicates that there have been no large-scale changes in growth of different parts of the brain since our mutual common ancestor.

The same analysis applied to humans and macaques (*Macaca mulatta*) tells a different story (Fig. 7.4). Though sequential hypermorphosis applied to the macaque data brings it into line with the fetal stage of human growth and the later stage after about one year after birth, the two curves are clearly not commensurate over the period from birth to about one year of age in humans. This is particularly apparent in the plot of between-species resid-

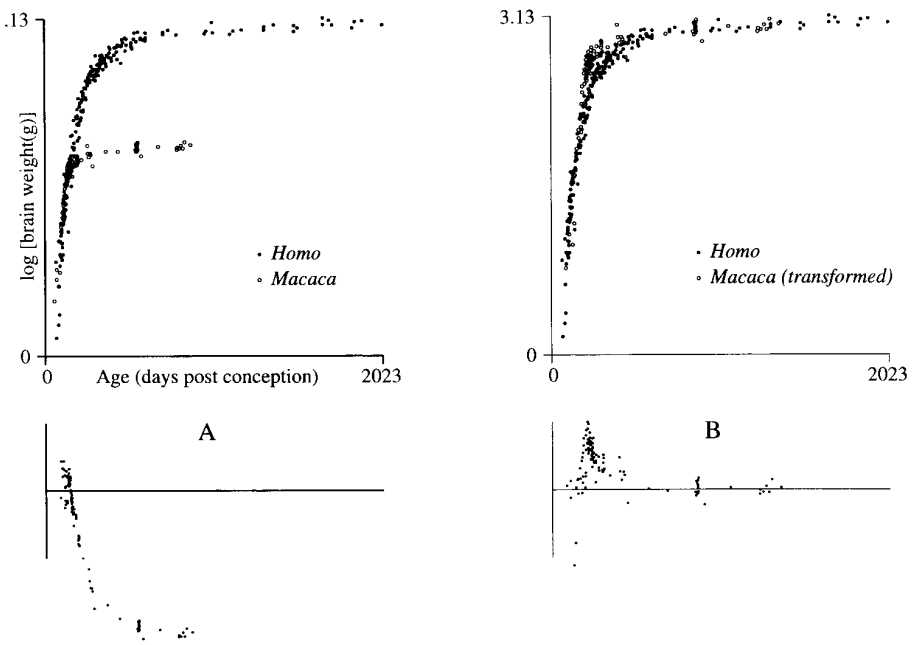


**Fig. 7.4.** Comparison of human and macaque trajectories. *A:* The macaque trajectory closely follows that for humans initially and then deviates at about the time of birth in macaques. *B:* Expanding the macaque curve by a factor of 1.56 produces a good match for the early and late parts of the trajectories but leads to an overshoot at around the time of birth. This is apparent in the plot of residuals, which has a run of 66 points (out of 101) above the human curve. The probability of a run of this length or longer arising by chance if the curves were actually the same is less than $10^{-6}$.



uals, which shows a distinct bump during this stage. The longest run of residuals on the same side of the line is 66 (out of 101 points); the probability of such a run (or a longer one) is less than $10^{-6}$. Further transforming the macaque data with neoteny improves the fit but not to the degree that we would accept heterochrony as an explanation of the differences between these two species.

Considering just three species, we cannot say whether the human/chimp brain growth trajectory is ancestral or derived within the primates. To resolve this, we need another outgroup (Maddison et al. 1984). The squirrel monkey (*Saimiri sciurius*) is a New World monkey that is an outgroup to the clade containing the Old World monkeys (including the macaque) and the apes. The ontogenetic trajectory of the squirrel monkey brain differs from all those considered thus far in that the growth spurt is delayed until after birth. When it occurs, though, the growth spurt and subsequent leveling off of brain size are commensurate with the trajectory for macaques (Fig. 7.5). Thus, the kind of trajectory exhibited by chimpanzees and humans seems to be derived within the primates.

It is generally argued that postnatal growth of the human brain is simply an extension of the fetal pattern of growth beyond our premature (relative to other primates) birth date (Deacon 1997; Martin 1983), either with a simple extension or through sequential hypermorphosis (McKinney and McNamara 1991). The data considered here suggest that this is not the case. When we plot brain size against time, the initial growth phase following birth clearly shows a slope different from that of the prenatal phase (Fig. 7.6*A*). When we plot brain size against body size (as is usually done), the difference in slopes is less pronounced but still significant (Fig. 7.6*B;* using an $F$ test for equality of slopes [Sokal and Rohlf 1995]). Thus, the distinctive growth phase seen in humans and chimpanzees does not seem, in and of itself, to be a heterochronic modification of any phase in the growth of other primates. Count (1947) noted this pattern also, defining a "transitional" growth phase between the "fetal" and "post-infantile" phases (see Count's Figs. 1 and 2).

Putting all of these results together on a phylogeny (Fig. 7.7) suggests that, sometime between the common ancestor of Old World monkeys and apes, around 25 million years ago (Goodman et al. 1982), and that of chimpanzees and humans (around 5 million years ago), a novel phase of brain growth appeared in the hominoid line. This growth phase begins at around the time of birth and continues for approximately nine months in chimpanzees and one year in humans.

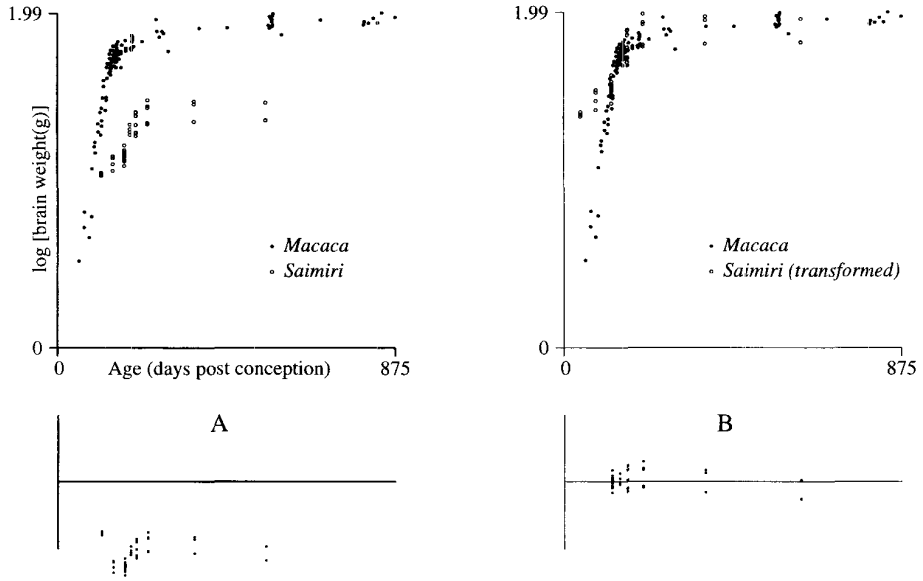


Fig. 7.5. Macaque and squirrel monkey trajectories. *A:* The growth spurt of the squirrel monkey brain occurs much later than that of the macaque. *B:* Transforming the squirrel monkey trajectory with a combination of sequential hypermorphosis (× 1.35) and predisplacement brings the growth spurt up to a fairly good match with that of the macaque. The maximum run of residuals is six in a total of forty-two points ($p$ = 0.477).

This notion that the brains of humans and chimpanzees are developmentally similar to one another and different from those of other primates is consistent with the observation that chimpanzees show a humanlike pattern in the asymmetry of the planum temporale, a region of the brain associated with language in humans (Gannon et al. 1998). Thus, whatever internal modifications of brain growth set the stage for the evolution of the human brain, they seem to have been in place by the time of the most recent common ancestor of humans and chimpanzees.

Finally, a note on hypermorphosis. Although it seems to be one of the commoner types of heterochrony, hypermorphosis is, from the standpoint of analysis, the most problematic because we cannot know where a trajectory would go if "allowed" to continue. The test for sequential hypermorphosis discussed here is applicable only if the trajectories in question are composed of linear segments (which seems to be the case for log-transformed brain trajectories [Vrba 1998]).



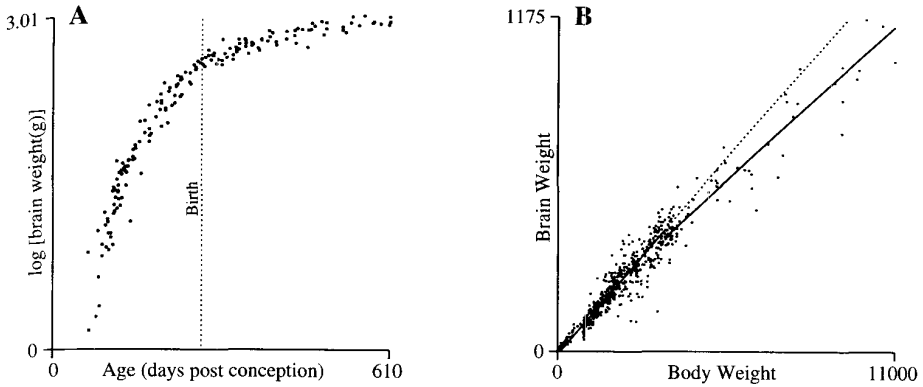


Fig. 7.6. *A:* Human brain growth trajectory up to the age of about one year after birth. The postnatal growth phase is not a hypermorphic extension of prenatal growth. *B:* Brain weight in relation to body weight for humans up to about one year after birth. The *solid lines* are the reduced major axis regression lines through the points up to 3,500 g body weight (around birth) and after this point. The *dotted line* is the extension of the fetal regression line. The longest run of points on one side of this extended fetal line is fifteen ($p < 0.01$ by the test described earlier). *Sources:* Data are from Dobbing and Sands 1973, Blinkov and Glezer 1968, Larroche 1967, and Burn et al. 1975. Estimates of age by weight are from Dobbing and Sands 1973.

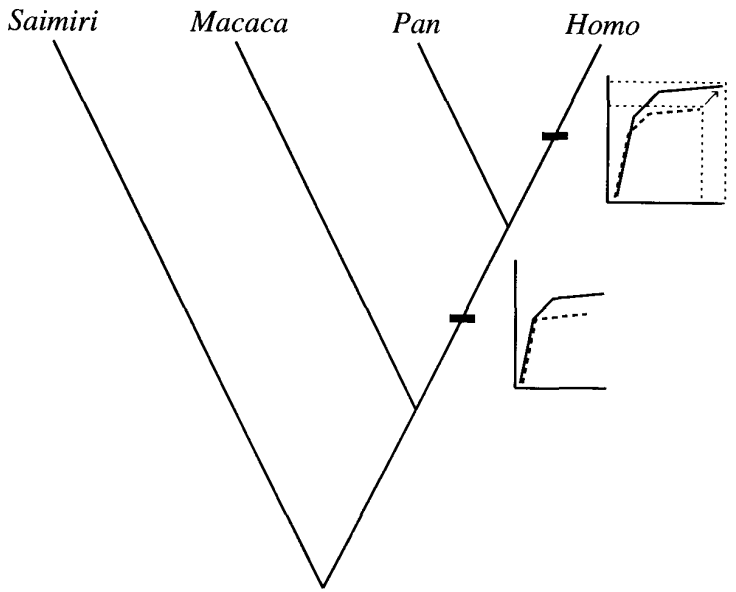


Fig. 7.7. Phylogeny of anthropoids showing changes in the ontogenetic trajectory for brain size.

### *Body Size*

Figure 7.8 shows trajectories of body size for humans and chimpanzees (males in each case). A combination of neoteny and sequential hypermorphosis applied to the entire chimp trajectory yields a reasonable overlap (Fig. 7.8*B;* Rice 1997 considered only this comparison). If, however, we consider separately the curves before and after the onset of the adolescent growth spurt, we find that a combination of sequential hypermorphosis, neoteny, and predisplacement explains the differences between the trajectories up to the onset of the growth spurt (Fig. 7.8*C*), while the same amount of sequential hypermorphosis combined with slight acceleration yields a striking match for the trajectories after this point (Fig. 7.8*D*).

The need for some predisplacement to align the trajectories is probably an artifact caused by measuring age since birth, which in chimpanzees occurs after a shorter gestation than in humans. The apparent postdisplacement in Figure 7.8*D* is a consequence of anchoring the trajectories at the point at which they diverged in Figure 7.8*C*. Because some of these data are derived from following the growth of particular individuals, we cannot treat each point as an independent event and thus cannot apply the statistical test discussed above. The close match of the trajectories, though, especially over highly nonlinear regions, makes a strong case for heterochrony.

Both neoteny and hypermorphosis thus seem to have played roles in the evolution of human growth. More significantly, this analysis draws our attention to the onset of the adolescent growth spurt in humans as a point at which there has been a change in the nature of the growth process. This is consistent with the arguments of Bogin and Smith (1996) that the adolescent growth spurt in humans is a novel character distinct from the small increases in growth seen in some other primates. Here, this novelty is seen to be a consequence of slowing growth (neoteny) until the onset of puberty.

Partitioning an ontogenetic trajectory in this way and then looking for heterochrony in the different parts is dangerous. We can render any trajectory into a large number of approximately linear segments; if we are then allowed to apply any kind of heterochrony independently to each segment, we can make the resulting trajectory look like whatever we want it to. Breaking up the trajectory is appropriate in this case, since the two parts have distinct, nonlinear forms and the breakpoint corresponds to a developmentally distinguishable event, the onset of puberty.



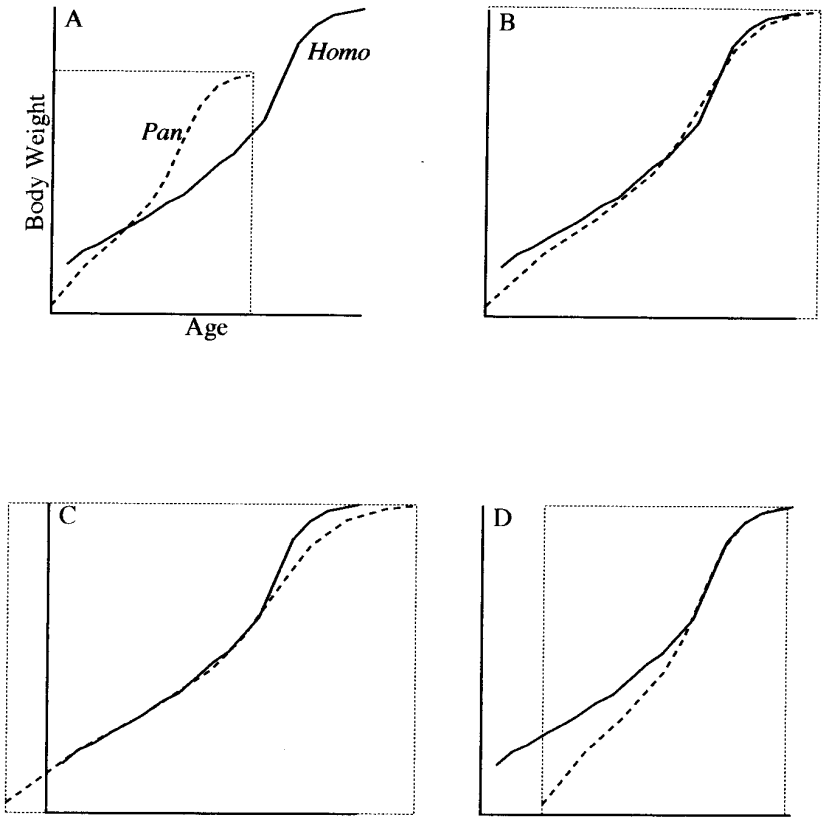


Fig. 7.8. Ontogenetic trajectories for body weight as a function of age for humans and chimpanzees (*Pan*). *A:* Untransformed trajectories. *B:* The best match for the entire trajectories obtained with a combination of sequential hypermorphosis and neoteny applied to the chimpanzee curve. *C:* Chimpanzee curve transformed by sequential hypermorphosis (both axes stretched by a factor of 1.28) and neoteny (age axis stretched by a factor of 1.6) combined with predisplacement. *D:* The same amount of sequential hypermorphosis combined with a slight amount of acceleration (untransformed trajectory compressed by a factor of 0.96). The chimp curve here is positioned so that the onset of the growth spurt occurs at the same point as in *C. Sources:* Human data are from Tanner 1978; chimpanzee data are from Grether and Yerkes 1940.

## Conclusions

Even using a definition that would reject most changes in development, one can still conclude that heterochrony has played an important role in human evolution. In particular, the human brain follows a growth curve that is al-

most exactly what would be expected from sequential hypermorphosis applied to the chimpanzee brain. This said, the case in which heterochrony is rejected as an explanation (the comparison of human/chimp brain growth with that of other primates) is in some ways the most interesting. Rejecting heterochrony as a mechanism here highlights the appearance of a novel phase of brain growth that arose before the common ancestor of humans and chimpanzees. Along with the results of Gannon et al. (1988), who showed that chimpanzee brains are morphologically similar to human brains even in a character that (in humans) functions in language processing, the results presented here imply that much of the important rearranging of brain growth that led to the evolution of the modern human brain had already taken place before the split with our closest living relative.

The potential to reject a hypothesis of heterochrony is also important in the comparison of overall growth in body size. We can only accept or reject a hypothesis of heterochrony with respect to a particular trajectory or segment thereof. Rejecting heterochrony at a particular level may direct our attention to other levels. In this case, rejecting heterochrony at the level of the entire body size trajectory leads to the observation that different kinds of transformations are involved at different stages in the growth process. This, in turn, leads to insight into the evolution of another novel character, the human adolescent growth spurt. Thus, rather than rendering the concept too restrictive to be useful, narrowing and clarifying the definition of heterochrony makes it a more useful theoretical tool for the analysis of morphological evolution.

## APPENDIX

### Sequential Hypermorphosis

The transformations shown in Figure 7.1, *A* through *C*, have the property that the same transformation of the phenotype axis will linearize both trajectories (Rice 1997). They thus correspond to the traditional types of heterochrony discussed by Alberch et al. (1979). Figure 7.1*D* shows a transformation that does not meet this criterion but is still reasonably interpreted in terms of heterochrony. Because this particular transformation will play a major role in the discussion of primate evolution and because it strictly lies outside the classical categories of heterochrony, I discuss it in a little more detail.



Consider a phenotypic character, $\phi$, present in both an ancestor and a descendant, that follows ontogenetic trajectories $\phi_A(t)$ and $\phi_D(t)$ in the ancestor and descendant, respectively. We can write the derivatives of these trajectories as

$$\frac{d\phi_A}{dt} = \omega\,(\phi,p) \text{ and } \frac{d\phi_D}{dt} = \omega'\,(\phi,p')$$

where $p$ and $p'$ are sets of parameters that include any other factors influencing the development of the character. The transformations shown in Figure 7.1*A–C* are cases in which, for a particular value of $\phi$, call it $\phi^*$, that is visited by both trajectories,

$$\omega(\phi^*,p) = C\omega'(\phi^*,p')$$

where $C$ is a constant. In other words, the growth process has been sped up ($C > 1$) or slowed down ($C < 1$) or shifted as a unit, with no change in the structure of the equation describing growth. By contrast, the transformation in Figure 7.1*D* corresponds to

$$\omega(\phi^*,p) = \omega'(C\phi^*,p')$$

Here, the constant $C$ is moved inside the function and multiplies $\phi$ wherever it occurs. This corresponds to a case in which, for each value of the phenotype, the developmental process of the descendant behaves as that of the ancestor would at an earlier ($C < 1$) or later ($C > 1$) point in development. Geometrically, if we think of the trajectory as being made up of many small linear segments, then this is the same as extending ($C < 1$) or contracting ($C > 1$), each segment by the same amount. This corresponds to what McNamara has called sequential hypermorphosis (or progenesis). However, sequential hypermorphosis of each segment of the trajectory will look like Figure 7.1*D* only if those segments are linear (Vrba 1998).

### Statistics

Consider a string of points each designated either *A* (above the line) or *B* (below). The state of each point is independent of the others, and each has

a probability of 0.5 of being $A$ (or $B$). A run of length $x$ is an uninterrupted sequence of $x$ points with the same state.

To derive the recursion given in the text, we seek the probability that adding the $n$th point onto a string does not complete the first run of length $x$. This is equal to the probability that there was no run of $x$ in the first $n - 1$ points, $\Pi_{x,n-1}$, multiplied by the probability that, given that there was no such run, the $n$th point does not complete one.

First, we calculate the probability that the $n$th point does complete the first run of length $x$. For this to be the case, the last $x - 1$ points [counting backward from the $(n - 1)$th point] must have been the same and the one just before these different. There are only two ways to achieve this, $(x - 1)$ $A$s followed by a $B$ or the reverse. The probability that the last $x$ points have this property is thus $2(2^{-x}) = \frac{1}{2}^{x-1}$, and the number of possible sequences of $n - 1$ points with just the last $x - 1$ the same is $2^{n-1}/2^{x-1}$. The probability that there was no run of length $x$ in the previous $(n - 1) - (x - 1)$ points leading up to these is, by definition, $\Pi_{x,n-x}$, so the total number of possible sequences with no run of length $x$ but the last $x - 1$ constituting a run is

$$\frac{2^{n-1}}{2^{x-1}}\Pi_{x,n-x} \tag{7A.1}$$

The conditional probability, given that there was no previous run of $x$, of the last $x - 1$ points being the same is simply Equation 7A.1 divided by the total number of sequences of $n - 1$ with no run of $x$, which is

$$2^{n-1}\Pi_{x,n-1} \tag{7A.2}$$

There is a probability of 1/2 that the next ($n$th) point has the same value as the previous $x - 1$ points, so the probability that the $n$th point completes a run of $x$, given that there is no such run in the first $n - 1$ points, is ½ times Equation 7A.1 divided by Equation 7A.2, or

$$\frac{1}{2}\frac{\Pi_{x,n-x}}{\Pi_{x,n-1}} \tag{7A.3}$$

The probability that the $n$th point does not complete the first run of $x$ is then $\Pi_{x,n-1}$ multiplied by [1 − (7A.3)], which gives the result presented in the text.



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