ARTICLES

Evolution of Human Growth

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The human pattern of growth and development (ontogeny) appears to differ markedly from patterns of ontogeny in other primate species. Humans present complex and sinuous growth curves for both body mass and stature. Many human proportions change dramatically during ontogeny, as we reach sizes that are among the largest of living primates. Perhaps most obviously, humans grow for a long time, with the interval between birth and maturation exceeding that of all other primate species. These ontogenetic traits are as distinctive as other key derived human traits, such as a large brain and language. Ontogenetic adaptations are also linked to human social organization, particularly by necessitating high levels of parental investment during the first several years of life.

Despite the importance of these adaptations, our understanding of precisely how and why human ontogeny differs from ontogeny in other primate species remains qualitative and rather speculative.[1–6] Consequently, this study builds on previous analyses in fields such as allometry, heterochrony, human and primate growth studies, and life-history theory[7–10] to address several issues regarding human growth. First it develops an interspecific allometric perspective to determine how human growth compares to trends established by other anthropoid primates. Because humans are comparatively large primates, we may simply follow a growth pathway that is consistent with expectations based on body size. Second, this allometric foundation enables evaluation of two competing models that seek to account for the pattern of human growth prolongation. Prolongation could represent extension or "retardation" of all phases of growth. Alternatively, the overall prolongation of human growth could represent extreme lengthening of a single growth phase. Obviously, a blend of these possibilities, such as prolongation of a few growth periods coupled with abbreviation of others, may exist. Third, this study reviews alternative life-history models that seek causal explanations for prolonged human growth. Allometric results are applied to qualitative evaluations of these models with the objectives of clarifying the focus of these models and directing theoretical attention to new areas. For example, life-history models often emphasize certain variables, such as age at maturation, while looking past others, such as neonatal growth rates. Exploring human growth in light of alternative life-history models has the potential to refine and redefine these models with the ultimate goal of providing an integrated explanation for the evolution of human ontogeny.

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THEORETICAL PERSPECTIVES ON HUMAN ONTOGENY

Comparative and Historical Questions

Climbing Schultz's ladder: traditional perspectives on human growth

The fact that human ontogeny stands out among that of other mammals has been recognized for centuries, if not millenia.[11–13] However, comparative analyses of human ontogeny have only recently attracted attention in biological anthropology. Previous research generally conveyed the impression that uniform extensions in the duration of various growth phases account for the long period of human ontogeny. This view stems mainly from Adolf Schultz's[14] classic diagram of primate ontogenies (Fig. 1). This model, regularly figured in introductory biological anthropology textbooks, is based on several ontogenetic variables, including gestation length, dental eruption timing, reproductive maturation, and total life span. It predicts that the evolution of primate ontogeny involves "progressive" and regular increments in the duration of various growth stages, reminiscent of a *scala natura* concept of primate evolution. As noted by subsequent researchers,[15,16] Schultz's view implies that the evolution of primate ontogeny may have involved a regular, highly predictable, and perhaps orthogenetic process that culminated in the evolution of the human pattern. In effect, Schultz's view reflects a predictable, if rather uninspiring, mechanism for the production of ontogenetic diversity among primates.

Schultz's scheme, despite its simplicity, promoted significant advances in our understanding of human evolution. For example, Lovejoy[17] interpreted Schultz's framework as a reflection of "progressive prolongation of life phases and gestation in primates" (p. 342). According to Lovejoy, Schultz's model provides powerful evidence that a *scala natura* is an adequate metaphor describing developmental variation within the primate order. Lovejoy's perspective implies that prolongation of human growth

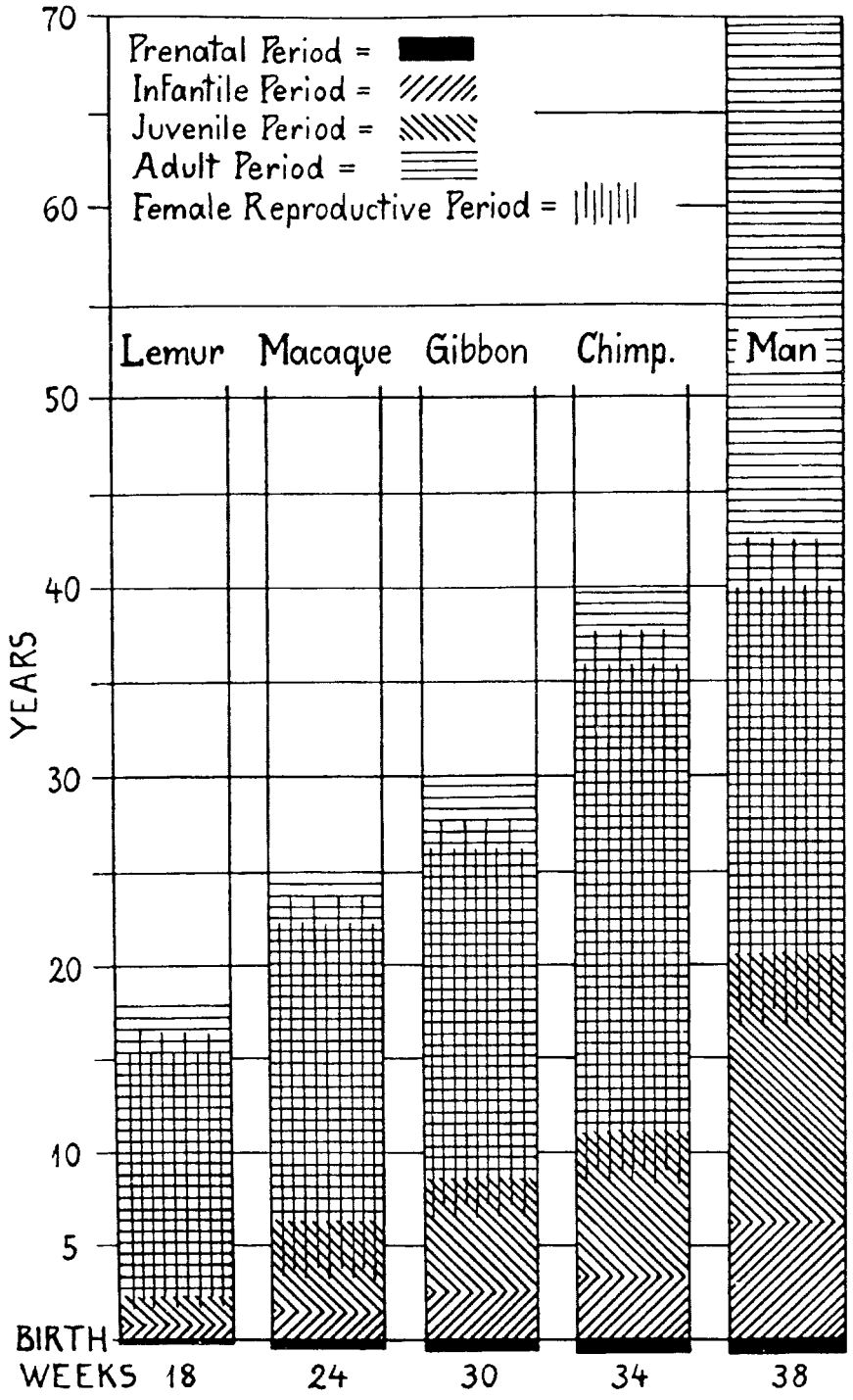

Figure 1. Adolf H. Schultz's rendition of primate life histories in comparative perspective.[14] This figure has been used repeatedly to demonstrate the prolonged period of human ontogeny.

was central to the divergence of hominins, requiring models of hominin evolution to include ontogenetic and life-history components. Lovejoy's interpretations have been controversial. Egregious errors can follow from applying a *scala natura* to ontogenetic variation within primates (Box 1). Nevertheless, he placed life history and ontogeny within the mainstream of biological anthropology while providing an impetus for critically evaluating Schultz's model.

Allometric approaches

Research during the 1970s and 1980s, particularly interspecific allometric studies, brought a quantitative comparative perspective to studies of primate ontogeny.[18,19] Unfortunately, few of these studies focused exclusively on human life histories. However, these studies illustrated significant variation in primate life-history parameters that could not be accommodated solely by body size; that is, they documented significant residual variation.[18–20] This finding implied less regularity in the evolution of primate ontogeny than could be encompassed by Schultz's model.

Building on this research, Gould[21] pursued themes that emerged from both Schultz's observations and allometric analyses to present the first major attempt to evaluate ontogenies as adaptations.[21] Gould's theoretical framework moved well beyond Schultz's model by emphasizing variables that are important in the analysis of heterochrony. These variables include size and shape, as well as the timing of events in ontogeny. Gould promoted his perspective by exploring the adaptive bases of a "matrix of retardation" in human ontogeny. According to Gould[21]:

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> "A general, temporal retardation of development has clearly characterized human evolution. This

Box 1. Primate Life History and Race

Schultz's diagram is commonly used as a representation of primate life histories. Lovejoy's deployment of the figure in his 1981 article undoubtedly popularized the model.[17] Unfortunately, Lovejoy also explicitly brought the notion of a *scala natura* into this framework, transcending and formalizing Schultz's general comments about the "progressive" nature of primate life histories. Lovejoy obviously mentioned the *scala natura* in a metaphorical sense to simplify variation within the primate order. However, this metaphor has been taken literally in attempts to associate "racial" variation in IQ with developmental scheduling. Specifically, J.-P. Rushton[73] used Schultz's diagram coupled with explicit reference to Lovejoy's *scala natura* in constructing a "racial" ranking system. Thus, Rushton's "relative ranking of races on diverse variables" is determined by differences among "races" in developmental scheduling. This simply amounts to extending Schultz's figure along the *X* axis to accommodate "racial" categories of humans.[73] More explicitly, Rushton grounded his idea of "an inverse relation . . . between brain size and speed of physical maturation" (p. 214) on Schultz's model.

Ranked differences stem from variation in maturation rates, which are said to affect brain morphometrics and IQ. Rushton's ideas are problematic at a number of different levels.[74,75] However, Schultz's model can no longer be used as an adequate description of primate life-history variation. Moreover, by relying on a scala natura, Rushton forfeits any insight into human variation that could be gained from evolutionary theory.[76] Simply put, and as is clearly reflected by Fleagle's updated version of this figure,[77,78] a better understanding of primate ontogenetic variation fails to illustrate the progressive climb so crucial to Rushton's narrative.

> retardation has established a matrix within which all trends in the evolution of human morphology must be assessed" (p. 365).

Gould also attempted to explain ontogenetic variation through the concept of an "r" and "K" selection continuum. Despite the fundamental deficiencies of r-K selection theory,[10] Gould's framework stimulated adaptive explanations of variation in age at maturity in primates[20] and showed that life-history theory could play a role in explaining differences in morphological ontogeny.

Gould's views were consistent with what was known about comparative growth and development during the 1970s. However, we still lack a clear understanding of whether or not this "matrix of retardation" adequately accounts for the human pattern. Consequently, this analysis evaluates Gould's idea along with alternative hypotheses that seek to define and explain derived changes in human growth. Addressing these issues involves consideration of several contemporary theoretical perspectives that explore complementary, but often nonoverlapping aspects of this problem. Specifically, researchers have either focused exclusively on distinguishing how the pattern of human morphological ontogeny compares to that of other primates or have applied general life-history models to humans, concentrating mainly on maturation age. Thus, a secondary goal of the study is to achieve better integration between these fields.

Allometry and Life History of Ontogeny

Redefining allometric hypotheses

Allometric analyses have great potential to increase our understanding of how human ontogeny compares to that of other primates. It is important to emphasize that Schultz's scheme is insufficient for this purpose for at least three reasons. First, as noted, he used data from several developmental systems. We now know that each of these traits may evolve in response to differing selective pressures.[22] Consequently, comparisons of ontogeny require investigations that isolate specific variables or use multivariate measures that capture variation in numerous traits. Second, the variation Schultz observed could easily be explained by simple size differences among species. In effect, the X-axis in Schultz's diagram (Fig. 1) represents a size axis, with each species spending the same proportion or percentage of its total growth period at each developmental phase. Third, allometric analyses lead to quantitative estimates of the degree to which human growth differs from expectations. Such estimates may be extremely important for refining life-history models.

An allometric perspective reveals two major possibilities to explain how prolongation of human growth may have occurred. The first is a "general extension" model. In this model, prolongation of growth could be a result of evolutionary extension in all growth periods (for example, prenatal, neonatal, and infant). This model emerges from both Schultz's model and Gould's views on human temporal retardation. For each growth period, human values should either meet expectations based on size (reflecting maintenance of proportions), or deviate from expectations in a consistent direction. The second model, differential extension, involves extension of only one or a few specific periods. For example, a long period of infancy can result in delays in maturation, but this may not be accompanied by increases in the length of other developmental periods. Obviously, the lengths of various phases could be altered in any direction and still lead to an overall delay in age at maturation. With differential extension, certain periods of human growth should show deviations from expectations, and the pattern of deviations should illustrate at least one period of ontogeny that is longer than expected. Deciding which

of these two models applies to humans is critical to evaluating how well life-history models explain human growth.

A complicating factor in choosing between these possibilities concerns alternative mechanisms for differential extension. For example, an extension of the total preadult period could be produced either by increases in specific growth phases or through "insertion" of novel growth periods not present in ancestral forms. These possibilities are very difficult to distinguish from one another. In each case, growth phases are defined based on inflections in growth rate curves.[23–25] Thus, Bogin[23] has suggested that human statural growth is prolonged through "insertion" of novel growth periods representing childhood and adolescence. His idea relies on evidence that human statural growth spurts are unique to primates. In addition, Bogin employs both behavioral and hormonal definitions to define these different traits. Unfortunately, testing these hypotheses for other primates requires separate investigations. On the other hand, mass growth spurts are very common among primate species, including humans[15,26] and African apes,[15,25] which enables comparative analyses. While it may not be possible to distinguish between these hypotheses, this distinction may be unnecessary for mass growth, given the qualitative similarities between mass growth-rate curves for humans and other primates.[25]

Life-history approaches

Explanations of why human growth differs from the growth of other primates must be sought in terms of life-history models. Causal explanations rest on a strong foundation of demographic and life-history studies of human populations that has developed in the last two decades.[27,28] Integration of allometric and demographic approaches has considerable potential to refine life-history models, but not all models recognize precisely the same classes of allometric variables (for example, growth rates and growth phase durations). Thus, most life-history models concentrate on explaining maturation age variation[10] without attempting to subdivide the time span prior to maturation. This emphasis has repeatedly proven its value, but understanding how human ontogeny compares to that of other primates should point life-history theories to new research areas. For example, either a protracted infancy or adolescence may retard human age at maturation, but these scenarios carry very different implications for parental effort,[29] functional differences among age classes,[30] mortality, and social organization. Therefore, this analysis briefly reviews alternative life-history models and applies allometric results to these models. Allometric findings provide the opportunity for qualitative assessment of how well life-history models account for ontogenetic differences between humans and other primates. The life-history models considered include a brain growth model, an adult mortality risk model, a juvenile metabolic risk aversion model, and an investment model.[9,28] Although the present analysis approaches these questions qualitatively, it can be noted that quantitative tests of life-history models with primate interspecific data show both advantages and disadvantages to several of these models.[31]

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Brains and Learning. Attempts to interpret the long period of human growth by reference to either learning or brain ontogeny have a deep history. As early as the mid-1700s, Henry St. John (Lord Bolingbroke)[32] proposed that humans require long periods of development "because they have more to learn and more to do" than other animals (p. 383). This idea was reformulated by numerous authors, then cast into Darwinian framework by John Fiske[33] and Herbert Spencer.[34] Subsequent authors amplified this notion by relating growth prolongation to adaptive flexibility.[21] As with many historical ideas, claims about learning, plasticity, and growth prolongation often rely on untested assumptions. Recent formulations of this hypothesis suggest that human growth periods are lengthened because we must assimilate considerable information prior to adulthood.[1,9,21,23,28,31–40] Learning, behavioral-flexibility, and brain-growth models of life histories imply that the benefits of what is learned during the preadult period offset the reproductive opportunities lost by prolonged growth. These models predict that the earliest periods of growth have undergone the greatest prolongation because of ties between somatic and brain ontogeny. It can also be suggested that brain growth and development necessitates slow rates of growth in body mass in order to accommodate the metabolic costs of total brain size growth.

Adult Mortality. A compelling alternative general explanation of primate growth prolongation is an adult mortality model developed by Charnov.[7,41] This model predicts that extrinsic mortality determines the timing of maturation. It relies on the observation that the amount of energy available for growth and reproduction (the "production function") for primates is lower than that of other mammals. The relatively low primate production function affects both growth and reproduction, with optimal adult body size set by a balance between mortality and production. Selection for larger body size, given the low primate production rate, increases the time necessary for growth, but the associated delay in maturation increases mortality risk before maturation. Large body size increases production, but only if the increase in

size offsets increased mortality. Decreased adult mortality is critical because it extends the expected adult reproductive life span enough to offset costs of delayed maturation while providing production benefits as a result of larger adult size. Maturation delays may not be related to selection directly on juveniles. Long juvenile periods may have no special significance, and thus may "arise automatically as a result of selection for larger size"[42] (p. 37).

A mortality risk model has difficulty accommodating differences in growth rates. Specifically, Charnov and Berrigan's[7] version of this model assumes that size differences among species are driven by differences in growth duration rather than growth rates, with growth rates determined by the equation:

$$dw/dt = A * (W^{.75}),$$

where growth rate (dw/dt) is equal to the production value (A) times mass at maturation age (W) scaled to the 0.75 power (or 0.7 power) (see Charnov and Berrigan[7] and Charnov,[41] respectively). Charnov and Berrigan[7] report an A value for primates at about 0.4, which is substantially below the value of 1.0 for other mammals. However, differences among species in growth rates may pose problems for the model simply because if rates vary, then either A must vary or mass must be scaled by a different exponent. Increases in growth rates can reduce the amount of time to reach adult size, thereby reducing time-related mortality risks during the growth period. On the other hand, this model cannot explain why growth rates may decrease, because this theoretically extends the period of preadult mortality in the absence of compensatory increases in growth rates during other phases of ontogeny. Insufficient knowledge about rate variation does not, however, diminish the value of exploring correlations between adult mortality and growth duration.

Metabolic Risk Aversion. The third model makes a very different set of predictions from the adult mortality model, but can be seen as an alternative mortality risk model. Specifically, Janson and van Schaik's[43] metabolic risk aversion model suggests that slow growth rates necessitate a lengthy period of ontogeny. The premise for this position is that low growth rates (small changes in mass per unit of time) can be selectively favored. Janson and van Schaik suggest that primates generally encounter conditions that increase metabolic risk as a result of feeding competition within groups, thus selectively favoring reduced growth rates. Feeding competition, which stems from predator pressure for group formation, may affect growing individuals more drastically than adults. Young animals, because of their small size, should face relatively high risks of mortality through predation. Feeding at or near the center of a group minimizes predation risks, but elevates feeding competition and, therefore, the risk of mortality through starvation. This, coupled with the fact that juveniles have less foraging efficiency than adults do, means that juveniles may encounter selection against high growth rates, reducing metabolic costs per unit of time. Maturational delays essentially arise as byproducts of selection against high growth rates. On the other hand, increases in growth rates could be explained in terms of reduced metabolic risks.

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Although Janson and van Schaik emphasize differences between primates and nonprimates, they also apply their model to explanations of differences among primates (for example, anthropoids versus nonanthropoids and folivores versus nonfolivores). The relationship between reduced growth rates and increased growth duration may not be obvious in this model. However, this model can accommodate prolongation of growth if selection for optimal adult size occurs late in ontogeny to balance early selection for reduced growth rates. Thus, they suggest that selection for slow growth rates can "force" delayed maturation as a "correlated response"[43] (p. 72).

Future Investment. Kaplan and colleagues[9] have recently proposed an integrative model to explain derived attributes of human life histories. They relate human growth prolongation to slow rates of growth in body mass and to the period of brain growth. Their model couples reductions in mortality with increases in parental production to offset the costs of subsidizing offspring development. These authors recognize that preadult humans are unable to support their own development but argue that this period of extremely low productivity is effectively an investment in future production. Specifically, large bodies that grow slowly, the acquisition of detailed knowledge, and the ability to learn all pay off by higher production in the adult period. This high payoff enables very high production surpluses that can then be channeled to developing offspring. Kaplan and colleagues note that this life-history pattern requires the ability to offset three important investment costs during the adult period, including "low productivity early in life, delayed reproduction, and a very expensive brain to grow and maintain" (p. 161). Delays in maturation enable the investment necessary to counterbalance these costs.

Ontogenetic data and attention to growth phases are potentially very important to this model. Distinguishing between general and differential extension hypotheses can lead to hypotheses about which costs are likely to be most important at various phases in development. For example, prolongation of all growth phases suggests an equal spread of investment costs throughout ontogeny. Earliest phase extensions imply that the costs of brain growth and basic knowledge acquisition are highest, while extension of later phases suggests that the most costly knowledge pertains to adult behaviors, including hunting, foraging, and social relations.

Testing Human Life-History Models. Evaluations of life-history models require data regarding ontogeny, demography, feeding competition, predation, and socioecology. However, the performance of these models with respect to the human case can be qualitatively evaluated by assessing how well they explain the observed pattern of human growth prolongation. First, a brain growth model predicts protraction of the earliest phases of human ontogeny. Exceptionally long later growth phases, such as the period encompassing the subadult growth spurt, may not be consistent with this model because brain size growth has been completed by this life phase. Second, an adult mortality model is consistent with prolongation of all growth periods at size-expected rates of growth. Here, mortality reductions produced by growing at a size-expected growth rate to a large size, with attendant increases in production, can explain the human pattern. Differences in growth rates during ontogeny are problematic for the adult mortality model.[22] Third, a metabolic risk-aversion model can accommodate differences in growth rates. However, heavy subsidies for human juveniles[9] complicate assessments of this model. Fourth, the investment model of Kaplan and colleagues fits best with prolongation of early growth phases, given its emphasis on learning, but can accommodate changes in the duration of later phases in any direction.

INVESTIGATING HUMAN GROWTH PROLONGATION

These models attempt to explain extremely important aspects of human evolutionary biology. However, they are difficult to test because of limitations on comparative growth data. Several factors account for this problem. Comparative ontogenetic data from known-age individuals of species other than macaques and chimpanzees[44] are extremely rare. Even the available data are inadequate for many comparisons, having been collected from small captive samples many decades ago, then analyzed without the benefits of new scatterplot smoothing techniques.[45,46] Growth studies often failed to include sufficient numbers of adult animals for gauging later growth. Certainly, new data are emerging, particularly from noncaptive populations,[46–49] and such data will be critical to future analyses. Unfortunately, chronological developmental data for traits other than body mass are virtually nonexistent. The only currently viable approach to comparing primate growth patterns involves studying growth in body mass based on data derived from captive sources. These data have certain limitations,[50,51] but these should not obscure broad-scale interspecific trends.

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Interspecific Comparisons of Growth Trajectories

This study uses human growth data representing Western populations, which can be assumed to represent circumstances that do not include nutritional stress. Information regarding early aspects of growth are derived from Tanner, Whitehouse, and Takahashi's British growth standards.[52] Because the attributes of growth spurts are important in comparisons across taxa, detailed data for human growth spurts reported by Buckler's[26] study of children in Leeds, UK are used. Body mass and chronological age data are available for nearly fifty nonhuman primate species, all from captive sources.[50] For the purposes of this study, only those species with late growth spurts will be quantitatively evaluated, reducing the sample to twenty-one species with male spurts and only twelve with female spurts.

This protocol has been employed previously[6,25] in comparisons between noncaptive and captive adult masses.[53] The analytical protocol involves several steps. First, mixed-longitudinal mass data collected from captive individuals are plotted against age. Second, nonparametric lowess regressions[45,46] are calculated for males and females. These regressions are desirable because they make no assumptions about functional form and can be applied across species. However, lowess regressions have a tendency to "overfit" some ranges of the data. Consequently, in a third step, data were resmoothed using nonparametric spline regressions.[54] Splines fit the data using a broader or global criterion, resulting in smoother curves that reflect less sensitivity to localized differences in data abundance. Fourth, values predicted by spline regression are used to calculate velocity or rate curves. These "pseudo-velocity" curves[55] portray changes in rates of growth (kg/yr).[6,25] A pseudo-velocity curve approximates the first derivative of a parametric curve.

The next step is to define various growth periods based on clearly defined inflection points in rate curves (Fig. 2). Subdividing body mass growth rate curves enables the analysis of several different variables. These are broadly divided into variables that describe either the time course or the rate of ontogeny. Additional comparisons are based on "size-for-age," which represents size (kg) at various points during ontogeny. All variables are assessed allometrically (that is, relative to size) in two ways. First, timing and velocity variables can be gauged relative to size-for-age. Second, these variables can be regressed on adult mass. Literature sources are used for investigating human gestation period and neonatal mass.[56,57]

Time components can be extracted from rate curves by defining segments along the X (age or time) axis. This study examines only three time segments, defined by the age at growth

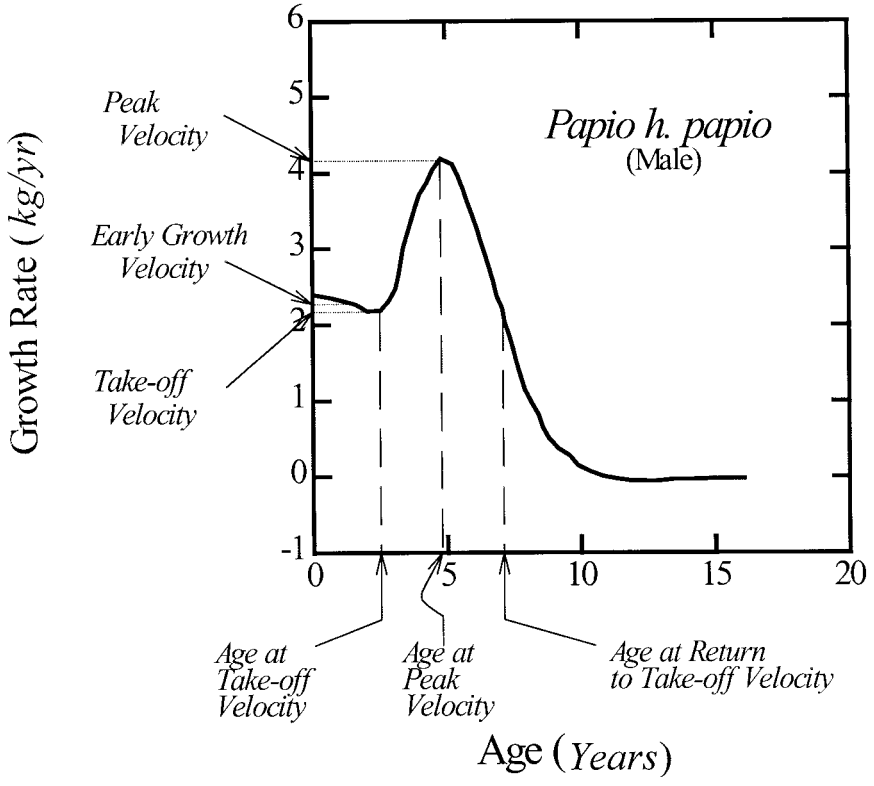

Figure 2. Attributes of growth velocity (pseudo-velocity) curves measured in the present study. The X axis represents age (in years), and the Y axis calibrates velocity (kg/yr) for male red baboons. Lines connecting the velocity curve to the Y axis measure velocities analyzed. Dashed vertical lines connecting the velocity curve to the X axis measure the age at specific periods of growth.

spurt initiation, the age at peak velocity, and the age at return to take-off velocity (Fig. 2). In the interest of clarity, time-course results are presented schematically for only a few selected species. These graphs show an expected time course of ontogeny based on adult mass as well as an observed time course of ontogeny (schematics are expressed in natural logarithms to facilitate direct comparisons with previously presented bivariate allometric plots[6]). These graphs show how growth timing differs from allometric expectations, enabling discrimination between the general and differential extension models. Human data points were excluded in the calculation of expectations for all variables.

The second category of variable that can be extracted from rate curves are velocities (Fig. 2), or variables along the *Y* axis. Velocities are especially relevant to tests of metabolic risk aversion hypotheses because Janson and van Schaik's[43] model predicts covariation between growth rates and metabolic risks. The growth rate early in ontogeny (early growth point) is measured at 50% of age at take-off velocity. Finally, analyzing the size of each species at various points in ontogeny (size-for-age) provides additional information. Specifically, sizes both at the early growth point and peak velocity are investigated. Size-for-age values represent species averages at given ages obtained directly from lowess regressions. All of these variables (timing estimates, rates, and size-for-age) have been analyzed in greater detail by previous studies.[6,25] Here, the focus is on the both the time course of human ontogeny and on contrasts between early and later ontogeny. Finally, it can be noted that the effects of phylogeny on these variables have been discussed elsewhere.[6]

HUMAN GROWTH IN COMPARATIVE PERSPECTIVE

Timing of Human Ontogeny

Gestation

The time course of human gestation is consistent with interspecific and size-based expectations. Martin and MacLarnon[56] reported that "humans match the typical pattern for precocial mammals generally, and for primates in particular" (p. 73). Their analysis shows that the protracted human prenatal period reflected by Schultz's diagram may be nothing more than what is expected for a primate of our size. More importantly, comparative data for large-bodied apes suggest that humans do not exhibit obviously extreme departures in the attributes of the prenatal period. These results indicate that the gestation period does not markedly prolong the total duration of human ontogeny.

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Postnatal growth

Schematic diagrams representing the time course of growth point to considerable diversity across primates (Figs. 3 and 4). The expected duration of the first growth period (the pre-spurt period) is independent of size across this sample,[6,25] but individual species differ substantially. Humans of both sexes demonstrate exceptionally long pre-spurt periods (Fig. 4), although protracted early growth periods clearly are not unique among primates. Humans also compress late growth periods relative to expectation. Human females show a more abbreviated second growth phase than do males, but males contract the third

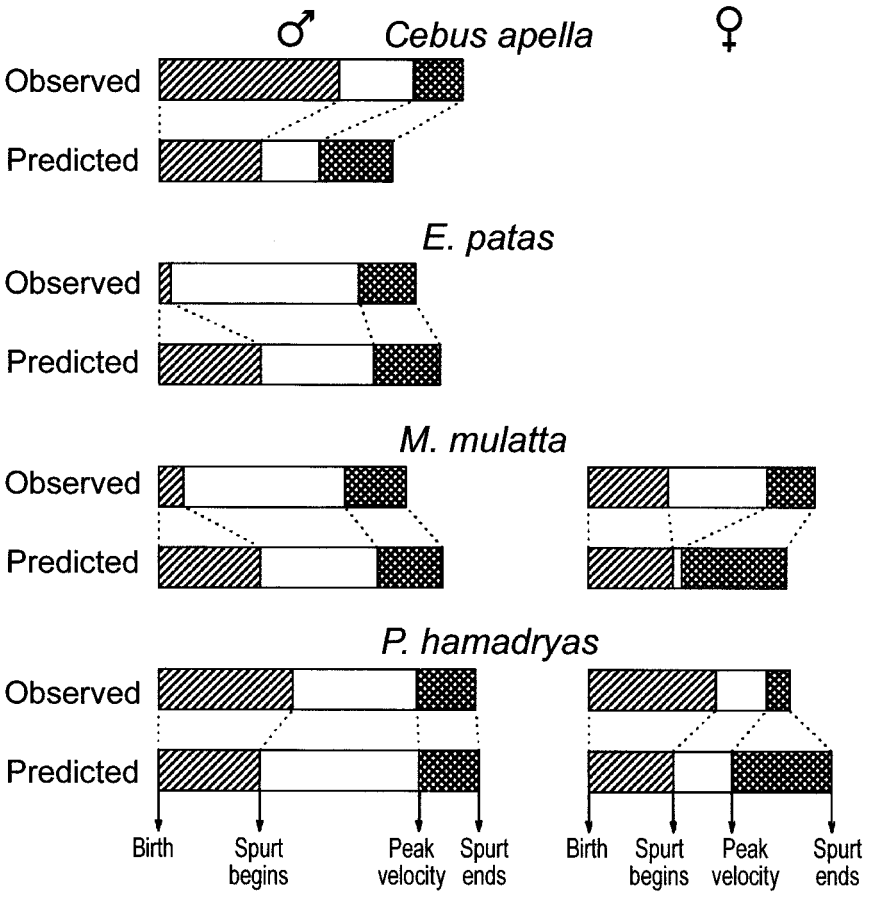

Figure 3. Schematic representation of growth-period durations for selected monkey species. The first filled area represents the period before initiation of the subadult growth spurt (birth to take-off velocity). The second, unfilled area represents the portion of growth from take-off to peak velocity, while the cross-hatched region denotes the period from peak velocity to return to take-off velocity. The upper bar shows observed values; the lower bar for each species is based on reduced-major axis predicted values at values for species average mass. Regressions used to calculate these expected values have been presented previously.[6] Each inch represents one natural logarithm unit.

phase as compared to females. The general human pattern is consistent with that seen in other large-bodied apes. Human growth prolongation seems to be mainly a product of changes in the earliest period of growth; later phases are actually briefer than expected.

Size During Ontogeny and Rates of Growth

Variables that describe size during growth and the velocity of body mass growth illustrate moderate to strong positive correlations as size increases across primates (Figs. 5–7). During early ontogeny, humans generally deviate considerably from interspecific expectations, but these deviations are often minimal during the growth spurt. Thus, humans tend to fall within the range of primate variation as ontogeny progresses. In addition, common chimpanzees (*Pan troglodytes*) typically deviate in the same direction that humans do.

Comparisons of mass at various points in ontogeny (size for age) in relation to adult mass (Fig. 5A–C) illustrate these trends clearly. Correlations between neonatal and adult mass are strong (Fig. 5A). Further, humans deviate from interspecific expectations to a degree not seen in other primates.[57] Size at the early growth point shows a lower correlation with adult mass than does neonatal mass, and humans still deviate considerably from expectations (Fig. 5B). However, during the peak of the growth spurt, humans are nearly the size expected for primates of our size, and fall within the range of residual variation.

Growth rates at different points in ontogeny are strongly correlated with adult size (Fig. 6A–C). Human growth rates are extremely low during early phases, but align with interspecific expectations later in ontogeny. Human growth rates do, however, fall within the range of residual variation even during the early phases of growth (particularly males). Peak velocities for human females and males are highly consistent with interspecific trends.

When growth rates are plotted against size at each growth point, lower correlations are apparent (Fig. 7A–C). Here, the pattern of deviations is closely consistent with the pattern of deviations seen when velocities are regressed against adult mass (Fig. 6). As with previous regressions, humans fall well within the range of residual variation as ontogeny proceeds (Fig. 7C).

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HUMAN GROWTH EVOLUTION

Differential Extension of Human Growth Phases

These analyses point to similarities and distinctions between humans and other primates. The time course of human growth is unexceptional both before birth and after initiation of the subadult growth spurt. In contrast,

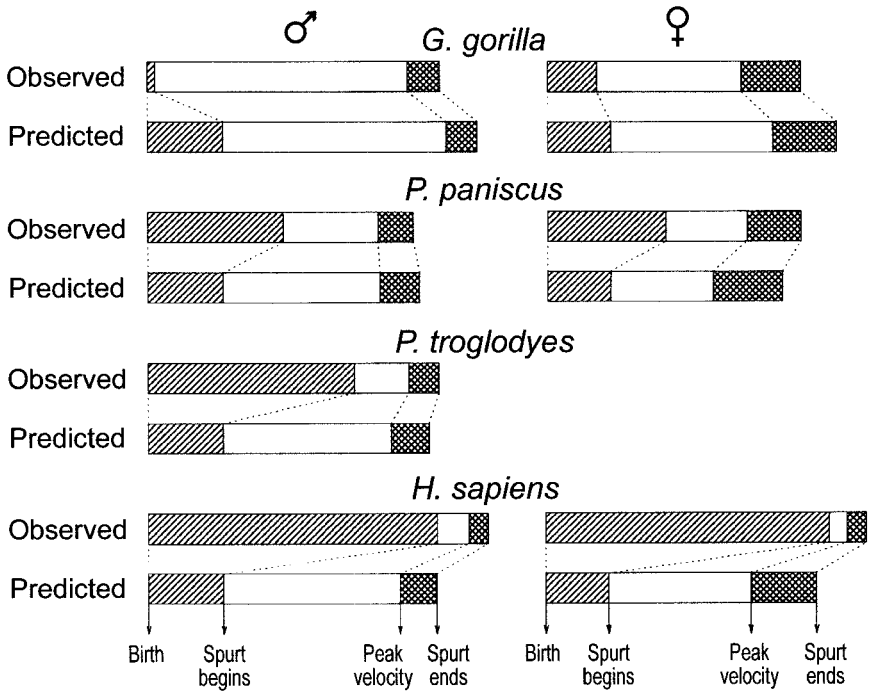

Figure 4. Schematic representation of growth periods for selected ape species. Female common chimpanzees appear to lack a clearly defined subadult growth spurt and are thus not used in this comparison.

the attributes of human growth are quite distinctive during early postnatal phases. These results reject the "general extension" model of human ontogeny, suggesting that Schultz's schematic model is no longer tenable as a description of ontogenetic variation among primates, at least for body mass. Moreover, the answer to the question of how different humans are from other primates depends on what point in ontogeny is being investigated: early periods of postnatal growth deviate substantially from interspecific expectations, but later periods are quite consistent with expectations.

As previous authors[1,15,38,58,59] have recognized, spurts in the growth of body mass occur in many primate species,[25] but in humans are unusual in some respects. We initiate abbreviated growth spurts quite late, but the other properties of the growth spurt are comparable to those of other primates. In effect, we displace an otherwise "standard" but condensed primate subadult growth spurt to a fairly late age. Other things being equal, later human growth periods should reduce the overall growth period because human subadult growth spurts are actually briefer than might be expected. The degree to which humans displace this "standard" subadult primate growth spurt to later ages is quite exceptional, making increases in the early period of human ontogeny all the more remarkable. Many human traits are extraordinary during the period of growth framed by birth and the initiation of the growth spurt. These unusual attributes include large neonatal size, slow growth rates, and deferral of the subadult growth spurt. Large size during this period is probably a product of high neonatal weight. Because the duration of human gestation follows a size-based expectation, it is clear that human gestation reflects a strategy described as "energy expensive" rather than "time expensive."[60] However, just the opposite strategy seems to be pursued between birth and the subadult spurt, with low growth rates distributed over a long period. An energy-expensive approach reflects heavy maternal investment in gestation, while a switch to a time-expensive strategy probably spreads the metabolic burdens of infants over time.

Reconstructing an ancestral condition based on our close relatives (chimpanzees, pygmy chimpanzees, and gorillas) reinforces findings from anthropoid-wide analyses.[60] Humans, gorillas, and pygmy chimpanzees all show both male and female subadult growth spurts, whereas female subadult spurts appear to be absent in chimpanzees.[25,60] If gorillas reflect an ancestral condition for humans, then prolongation of an early human growth phase is quite exceptional because the time span before the subadult growth spurt in gorillas is very short. However, gorilla ontogeny is closely tied to folivorous diets.[61,62] Specifically, gorillas, like other folivores, show elevated growth rates and enhanced growth spurts. Thus, gorilla growth patterns are derived as an adaptation to folivory, manifested as a derived extension of the early growth-spurt period. On the other hand, male chimpanzees and both sexes of pygmy chimpanzees have relatively long growth periods before the subadult growth spurt. Therefore, small differences separate the chimpanzee and human patterns, and the deviations of humans and chimpanzees are consistently in the same direction. Humans and chimpanzees each prolong the earliest phase but abbreviate spurt phases (Figs. 3, 5).

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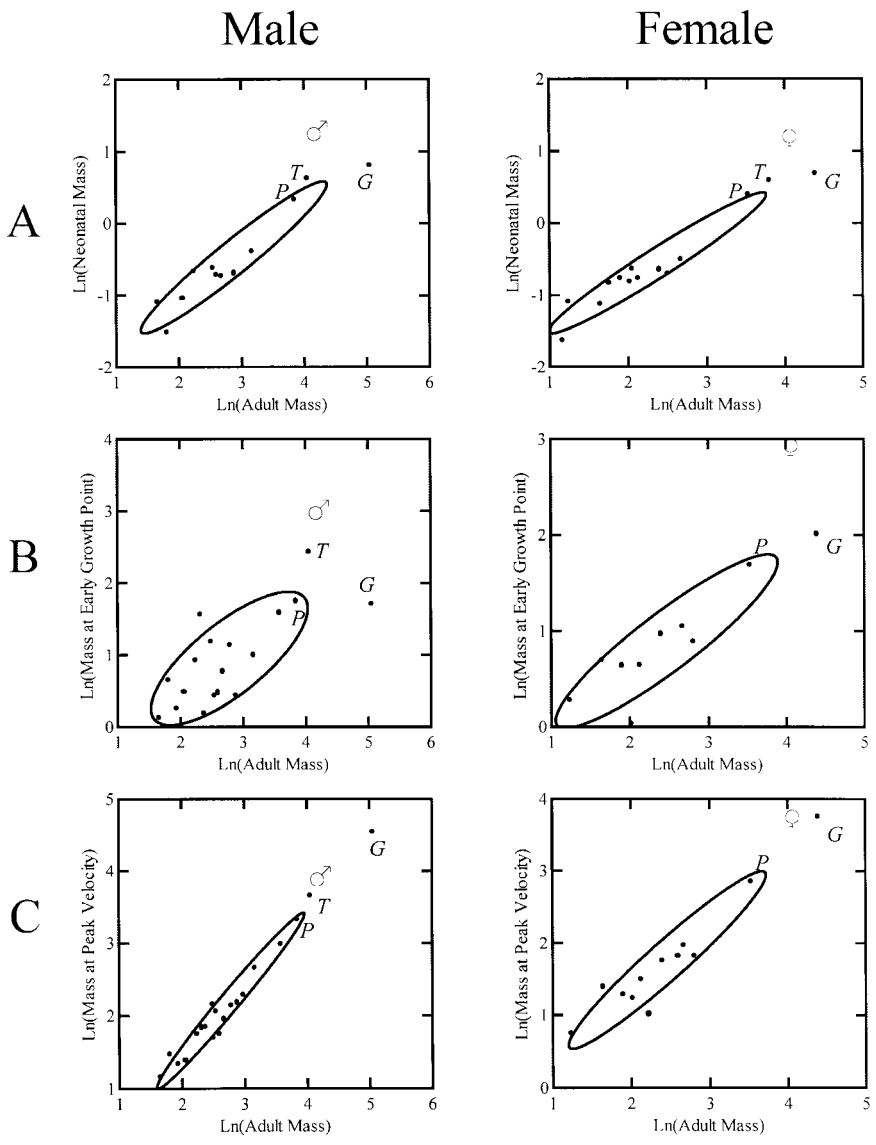

Figure 5. A: Regression plot of neonatal mass against adult mass for male (left) and female (right) anthropoid primate species. Humans are represented by male and female symbols; African ape species are designated by letters (G = gorilla, P = pygmy chimpanzee, T = common chimpanzee). Ellipses represent 60% confidence intervals on the major axis regression for each bivariate relation. All subsequent figures follow these conventions. B: Regression of mass at the early growth point (1/2 age at take-off velocity) plotted against adult mass. C: Regression of mass at peak velocity against adult mass.

At a more general level, comparisons reflect heterogeneity in the ways in which ontogenies can be "assembled." For example, humans grow very rapidly over a run-of-the-mill gestation period, but reverse this pattern after birth and before the growth spurt, growing slowly over a long period. Then, at the spurt, we grow much as primates of our body size should. These observations, coupled with variation across primates, further suggest that the attributes of these phases are highly evolvable.[63] In other words, neonatal mass, early growth rates, and the duration of early growth periods seem to be quite responsive to selection. This point is perhaps underappreciated because Schultz's model conveys such a strong sense of orthogenetic regularity. In contrast, the high level of variation observed in the present study suggests that primate growth periods can respond quite readily, apparently independently of body size, to selection for a delay in the initiation of subadult growth spurts. In addition, the variable presence of subadult growth spurts, plus their wide taxonomic distribution, suggests that later periods of body mass ontogeny are also quite responsive to evolutionary pressures.

Research currently in progress on Old World monkey corroborates these ideas. For example, skeletal growth spurts are evident in the faces, but not necessarily in the postcranial linear dimensions, of baboons. Thus, skeletal growth spurts appear to be "modular" and highly evolvable features of ontogeny: natural and sexual selection[5] can effectively modify the anatomical and chronological "placement" of skeletal growth spurts. These processes mean that there are also different ways of "assembling" ontogenies. Thus, we should expect multifactorial causes of variation at different stages of ontogeny and within different anatomical units or systems. This variation may ultimately be explained by reference to social and ecological selective factors, with potential links to life-history processes.

. . . the high level of variation observed in the present study suggests that primate growth periods can respond quite readily, apparently independently of body size, to selection for a delay in the initiation of subadult growth spurts.

Implications for Life-History Models

Support for the hypothesis that extension of early growth best accounts for human growth prolongation has important implications for life-history models. Comparative analyses of human ontogeny help establish the feasibility of various models and may point to additional hypotheses that can be derived from these models.

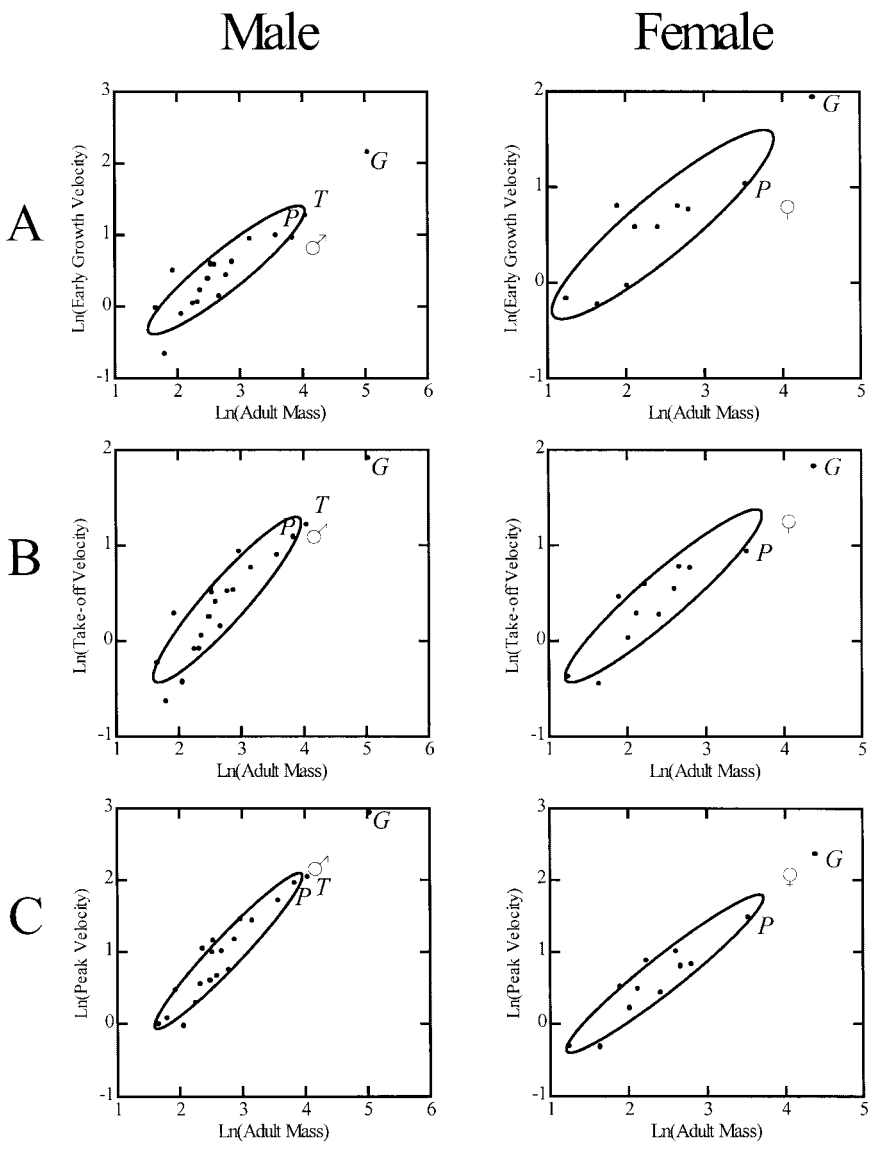

Figure 6. A: Regression plot of velocity at the early growth point (early growth velocity) against adult mass. B: Regression of take-off velocity against adult mass. C: Regression of peak velocity against adult mass.

Brains and learning

Extension of the early growth period is clearly compatible with a traditional brain growth or learning life-history model. Based on analyses of industrialized populations,[64] growth in the size of the human brain occurs only during the first seven to eight years of life. Human body mass growth rates, also from industrialized populations, increase steadily after about two years of age until roughly two years after the cessation of brain growth.[52] Thus, the relationship between brain growth rates and body mass growth rates is not simply an inverse relation, as might be expected based on straightforward metabolic "competition" between brain and body development.[65,66] Consequently, the impact of brain growth on the total duration of growth is uncertain, at least at a morphological level. On the other hand, processes at the cellular level may bear a relation to the total duration of ontogeny. Specifically, studies at the cellular level show two distinct phases of mammalian brain ontogeny, termed the "experience-expectant" and "experience-dependent" periods.[67] During the experience-expectant phase, primary synaptic connections are overproduced, then pruned in response to predictable environmental stimuli that are normally present during critical developmental periods. The subsequent experience-dependent period responds to unpredictable stimuli, leading to the storage of information unique to the individual. This information is encoded by forming new synapses or new connections in response to new knowledge.[68] These periods are crucial to normal brain development, but they take time. Thus, the protracted period of human growth may be related to evolutionary increases in the duration of one or both of these brain development periods. Furthermore, asynchrony in the time courses of these periods is evident when humans are compared to other primates. For example, many regions in the brain of rhesus macaques (*Macaca mulatta*) seem to pass through these stages at about the same age.[69] On the other hand, various regions of the human brain seem to go through experience-expectant and experience-dependent periods at different ages, with the development of some systems even occurring during adolescence.[70]

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These provocative findings suggest a need for future interspecific comparative investigations that evaluate potential correlations between synaptogenesis and growth prolongation. The present study suggests that this is plausible in the case of humans because the early growth period is so protracted. Although a brain-growth-learning model is feasible, we need much more comparative data on both

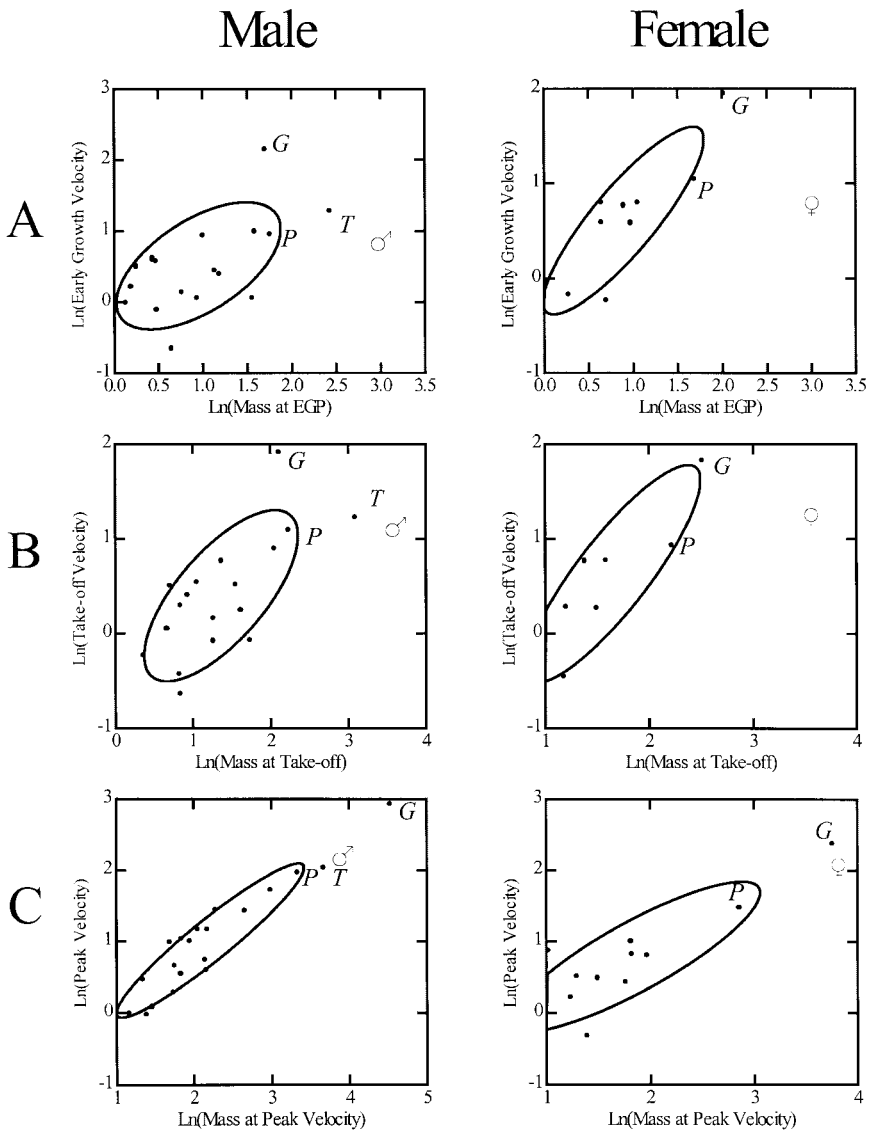

Figure 7. A: Regression of early growth velocity against mass at the early growth point (EGP). B: Regression of take-off velocity against mass at take-off velocity. C: Regression of peak velocity against mass at peak velocity.

size growth of the brain and the development of synapses in the brain before we can test this model further.

Adult mortality models

Localizing human growth prolongation to the earliest periods of ontogeny may have important implications for adult mortality models. Additional evaluation of mortality models requires much more demographic data.[31,71] However, an adult mortality model could suggest that long intervals of early growth require exceptionally low adult mortality. On the other hand, extension of later periods may not have the same requirements, in part because offspring can contribute to their own subsistence. It may be possible to prolong later growth periods, and thus the total growth period, without exceptional reductions in adult mortality.

The present analysis reveals areas of concern for adult mortality models. It is clear that primate growth rates vary considerably, and that growth rates scale to a variety of allometric coefficients,[6] possibly violating assumptions of Charnov's adult mortality model. Growth rate variability could suggest variable production costs, necessitating changes in the assumption of a growth constant. A constant growth rate means that size increase is a product of differing growth period durations, but this is not compatible with empirical evidence showing growth rate variation in primates.[45] Variation in adult size can be reached through many pathways involving timing differences and rate differences. Adult mortality models may require revision in order to accommodate these findings.

Metabolic risk aversion

Models of metabolic risk aversion provide compelling alternatives to both brain growth and adult mortality models.[43] As with demographic hypotheses, the data needed to test metabolic models are currently insufficient.[29,60,63] However, residual growth rate variation across primates may signal consistency with at least some aspects of a metabolic risk-aversion model. This variation could reflect differing profiles of metabolic risk, with periods of high growth rates indicating low risks and periods of low growth rates reflecting high risk.

A constant growth rate means that size increase is a product of differing growth period durations, but this is not compatible with empirical evidence showing growth rate variation in primates. Variation in adult size can be reached through many pathways . . .

Considerations of human growth prolongation suggest that metabolic risk-aversion models are compatible with other life-history models. This is particularly clear for the human case, in which low early growth rates could serve as a metabolic risk-aversion strategy that complements processes involved in learning or brain development. Extended early growth at a slow

rate may offset the risks of developing metabolically expensive structures such as the human brain (although, as noted, the relations between the brain and body growth are far from understood). For example, although adults devote about 25% of resting metabolic energy to brain maintenance,[72] infants devote roughly 85% to the same task.[65,66] Thus, the metabolic risks humans encounter may stem from the costs of a large brain. Evaluating metabolic risk models in light of information about the pattern of human growth prolongation suggests that ontogenetic allometric analyses of brain-body relations during the early period of growth can aid in further testing this model.

Investment model

This model accords well with the observed pattern of human growth prolongation. As anticipated by the model, slow growth for a long period is observed, with the most obvious differences between human- and primate-based expectations occurring during the phase when production is probably lowest. Moreover, a large portion of this stage corresponds to the period of brain growth, which may further increase the gap between production and consumption. Thus, this model seems to have predictive power for slow growth rates. The perspective of an investment model also suggests that early growth prolongation ensures a long period in which the cost of provisioning offspring is high. This raises particularly interesting problems for first offspring because they may necessitate rapid attainment of parenting efficiency. This requirement could help explain the abbreviation of later stages of human growth and rapid increases in production during these periods. An abbreviated human growth-spurt period may reflect rapid acquisition of adult skills during later growth in order to contend with the first offspring.

An investment model explains the pattern of human growth more adequately than does other models in part because it accommodates key elements of other life-history models. For example, an investment model is consistent with a brain and learning perspective, but in a manner that does not require strict adherence to metabolic demands of brain development. Instead, brain development is tied to the acquisition of skill and knowledge. In terms of this model, finding that the earliest phases of human growth are the most prolonged implies that basic knowledge acquisition is more costly in terms of production than is knowledge acquired during later phases of growth. As with a mortality model, a long period of child dependence would require especially low levels of adult mortality and high adult productivity during the earliest years of reproduction. Finally, an investment model is consistent with elements of metabolic risk-aversion models. While slow growth reflects low production, this model suggests that low growth rates should also reduce the costs of subsidizing a developing individual.

An investment model explains the pattern of human growth more adequately than does other models in part because it accommodates key elements of other life-history models.

High metabolic costs of rapid growth clearly raise the costs of subsidizing offspring growth, but slow growth rates may help offset these costs.

CONCLUSIONS

Understanding the evolution of human growth and development requires comparative analyses of other primates and tests of competing life-history models. Comparisons between humans and other anthropoids show that Schultz's influential model of primate ontogeny can no longer be applied to human growth, development, and life-history evolution. Similarly, Gould's general retardation model may not fit the human case. Extensions of early growth periods most clearly distinguish human ontogeny from that of other primates. The later stages of human ontogeny are abbreviated relative to interspecific expectations, while other attributes of later growth periods are compatible with these expectations. Humans and chimpanzees illustrate comparable deviations from interspecific expectations.

These results support some current life-history models while pointing to areas for new research within life-history theory. Brain growth models may be tenable, given that the majority of derived changes in human ontogeny appear to have occurred during the earliest postnatal periods. However, it is apparent that future analyses of brain growth models must incorporate ontogenetic data as well as investigate processes at the cellular level. Adult mortality models may provide further insight into the human problem, particularly if they can shed light on the correlations between extended early development and adult mortality. Comparisons of growth rates across primates may prompt modifications to adult mortality models. Juvenile metabolic risk models also have considerable potential to account for patterns of growth across primates. Finally, an investment model, with a special focus on investment during early growth, is compatible with the apparently derived pattern of human body mass ontogeny.

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REFERENCES

1 Bogin B. 1988. Patterns of human growth. New York: Cambridge University Press.

2 Shea BT. 1990. Heterochrony in primates. In: McKinney MJ, editor: Heterochrony in evolution. New York: Plenum Publishing. p 237–266.

3 Shea BT. 1992. Developmental perspective on size change and allometry in evolution. Evol Anthropol 4:125–134.

4 Smith BH. 1992. Life history and the evolution of human maturation. Evol Anthropol 4:134–142.

5 Leigh SR. 1995. Socioecology and the ontogeny of sexual dimorphism in anthropoid primates. Am J Phys Anthropol 97:339–356.

6 Leigh SR, Park PB. 1998. Evolution of human growth prolongation. Am J Phys Anthropol 107: 331–350.

7 Charnov EL, Berrigan D. 1993. Why do female primates have such long lifespans and so few babies? Or life in the slow lane. Evol Anthropol 1:191–194.

8 Stearns SC. 1992. The evolution of life histories. Oxford: Oxford University Press.

9 Kaplan H, Hill K, Lancaster J, Hurtado AM. 2000. A theory of human life history evolution: diet, intelligence, and longevity. Evol Anthropol 9:156–183.

10 Hill K, Kaplan H. 1999. Life history traits in humans: theory and empirical studies. Ann Rev Anthropol 28:397–430.

11 Lovejoy AO. 1959. Herder: progressionism without transformism. In: Glass BH, Temkin O, Strauss WL, editors. Forerunners of Darwin: 1745–1859. Baltimore: Johns Hopkins University Press. p 207–222.

12 Locke J. 1690. Second treatise on government. Cambridge: Cambridge University Press.

13 Fiske J. 1909. The meaning of infancy. Boston: Houghton-Mifflin.

14 Schultz AH. 1969. The life of primates. New York: Universe Press.

15 Gavan JA. 1982. Adolescent growth in nonhuman primates: an introduction. Hum Biol 54: 1–5.

16 Gavan JA, Swindler DR. 1966. Growth rates and phylogeny in primates. Am J Phys Anthropol 24:181–190.

17 Lovejoy CO. 1981. The origin of man. Science 211:341–350.

18 Harvey PH, Martin RD, Clutton-Brock TH. 1987. Life histories in comparative perspective. In: Smuts BB, Cheney DL, Seyfarth RM, Wrangham RW, Struhsaker TT, editors. Primate societies. Chicago: University of Chicago Press. p 181–196.

19 Harvey PH, Clutton-Brock TH. 1985. Life history variation in primates. Evolution 39:559–581.

20 Ross C. 1988. The intrinsic rate of natural increase and reproductive effort in primates. J Zool (Lond) 214:199–219.

21 Gould SJ. 1977. Ontogeny and phylogeny. Cambridge: Harvard University Press.

22 Periera ME, Leigh SR. 2001. Modes of primate development. In: Kappeler P, Periera ME, editors. Primate socioecology and life history. Chicago: University of Chicago Press. In press.

23 Bogin B. 1997. Evolutionary hypotheses for human childhood. Yearbook Phys Anthropol 40: 63–90.

24 Bogin B. 1999. Evolutionary perspective on human growth. Ann Rev Anthropol 28:109–153.

25 Leigh SR. 1996. Evolution of human growth spurts. Am J Phys Anthropol 101:455–474.

26 Buckler J. 1990. A longitudinal study of adolescent growth. London: Springer-Verlag.

27 Howell N. 1979. Demography of the Dobe !Kung. New York: Academic Press.

28 Hill K, Hurtado AM. Ache life history. Hawthorne, NY: Aldine de Gruyter.

29 Garber PA, Leigh SR. 1997. Ontogenetic variation in small-bodied New World primates: implications for patterns of reproduction and infant care. Folia Primatol 68:1–22.

30 Mayr E. 1963. Animal species and evolution. Cambridge: Harvard University Press.

31 Ross C, Jones KE. 1999. Socioecology and the evolution of primate reproductive rates. In: Lee PC, editor. Comparative primate socioecology. Cambridge: Cambridge University Press. p 73–110.

32 Bolingbroke H St. J. Lord Viscount. 1754. Fragments or minutes of essays. Hildesheim: Greg Olms Verlagsbuchhandlung (1968 facsimile edition).

33 Fiske J. 1885. The destiny of man, viewed in the light of his origin. Boston: Houghton, Mifflin.

34 Spencer H. 1888. The principles of biology, Vol. I. New York: D. Appleton.

35 Dobzhansky T. 1962. Mankind evolving. New Haven: Yale University Press.

36 Wasburn SL, Hamburg D. 1965. Implications of primate research. In: DeVore I, editor. Primate behavior: field studies of monkeys and apes. New York: Holt, Rinehart, and Winston. p 607–622.

37 Poirier FE. 1977. Introduction. In: Chevalier-Skolnikoff S, Poirier FE, editors. Primate biosocial development. New York: Garland Publishing. p 1–42.

38 Tanner JM. 1978. Foetus into man. Cambridge: Harvard University Press.

39 Smith BH, Tompkins RL. 1995. Toward a life history of the Hominidae. Ann Rev Anthropol 24:257–279.

40 Sarnat HB, Netsky MG. 1981. Evolution of the nervous system. New York: Oxford University Press.

41 Charnov EL. 1991. Life history invariants. New York: Oxford University Press.

42 Pagel MD, Harvey PH. 1993. Evolution of the juvenile period in mammals. In: Periera ME, Fairbanks LA, editors. Juvenile primates: Life history, development, and behavior. New York: Oxford University Press. p 28–37.

43 Janson CH, van Schaik CP. 1993. Ecological risk aversion in juvenile primates: slow and steady wins the race. In: Pereira ME, Fairbanks LA, editors. Juvenile primates. New York: Oxford University Press. p 57–74.

44 Watts ES, Gavan JA. 1982. Postnatal growth of nonhuman primates: the problem of the adolescent spurt. Hum Biol 54:53–70.

45 Efron B, Tibshirani R. 1991. Statistical data analysis in the computer age. Science 253:390–395.

46 Moses LE, Gale LC, Altmann J. 1992. Methods for analysis of unbalanced, longitudinal, growth data. Am J Primatol 28:49–59.

47 Pusey A. 1990. Behavioral changes at adolescence in chimpanzees. Behaviour 115:203–246.

48 Strum SC. 1991. Weight and age in wild olive baboons. Am J Primatol 25:219–237.

49 Cheverud JM, Wilson P, Dittus WPJ. 1992. Primate populations at Polannaruwa. III. Somatometric growth in a natural population of toque macaques. J Hum Evol 23:51–77.

50 Leigh SR. 1992. Ontogeny and body size dimorphism in anthropoid primates. Ph.D. dissertation, Northwestern University, Evanston, Il.

51 Leigh SR. 1992. Patterns of variation in the ontogeny of primate body size dimorphism. J Hum Evol 23:27–50.

52 Tanner JM, Whitehouse RH, Takahashi M. 1966. Standards from birth to maturity for height, weight, height velocity, and weight velocity: British children, 1965, Part II. Arch Dis Child 41:613–635.

53 Leigh SR. 1994. The relations between captive and noncaptive weights in anthopoid primates. Zoo Biol 13:21–44.

54 Schluter D. 1988. Estimating the form of natural selection on a quantitative trait. Evolution 42:849–861.

55 Coelho AM. 1985. Baboon dimorphism: growth in weight, length and adiposity from birth to 8 years of age. In: Watts ES, editor. Nonhuman models for human growth and development. New York: Alan R. Liss. p 125–159.

56 Martin RD, MacLarnon AM. 1990. Reproductive patterns in primates and other mammals. The dichotomy between altricial and precocial offspring. In: DeRoussea CJ, editor. Primate life history and evolution. New York: Wiley-Liss. p 47–88.

57 Smith RJ, Leigh SR. 1998. Sexual dimorphism in primate neonatal body mass. J Hum Evol 34:173–201.

58 Bogin B. 1994. Adolescence in evolutionary perspective. Acta Paediatr 40(suppl):29–35.

59 Watts ES. 1982. Adolescent growth and development of monkeys, apes, and humans. In: Watts ES, editor. Nonhuman primate models for human growth and development. New York: Alan R. Liss. p 41–65.

60 Lee PC. 1999. Comparative ecology of postnatal growth and weaning among haplorhine primates. In: Lee PC, editor. Comparative primate socioecology. Cambridge: Cambridge University Press. p 111–139.

61 Leigh SR. 1994. Ontogenetic correlates of diet in anthropoid primates. Am J Phys Anthropol 94:499–522.

62 Leigh SR, Shea BT. 1996. The ontogeny of body size variation in African apes. Am J Phys Anthropol 99:43–65.

63 Houle D. 1992. Comparing evolvability and variability of quantitative traits. Genetics 130: 195–204.

64 Jolicoeur P, Baron G, Cabana T. 1988. Cross-sectional growth and decline of human stature and brainweight in 19th century Germany. Growth, Dev, Aging 52:201–206.

65 Leonard WR, Roberston ML. 1992. Nutritional requirements and human evolution: a bioenergetics model. Am J Hum Biol 4:179–195.

66 Leonard WR, Robertson ML. 1994. Evolutionary perspectives on human nutrition: the influence of brain and body size on diet and metabolism. Am J Hum Biol 6:77–88.

67 Greenough WT, Black JE, Wallace CS. 1987. Experience and brain development. Child Dev 58:539–559.

68 Kleim JA, Vij K, Ballard DH, Greenough WT. 1997. Learning-dependent synaptic modifications in the cerebellar cortex of the adult rat persist for at least four weeks. J Neurosci 17:717–721.

69 Rakic P, Bourgeois J-P, Eckenhoff MF, Zecevic N, Goldman-Rakic PS. 1986. Concurrent overproduction of synapses in diverse regions of the primate cerebral cortex. Science 232:232–235.

70 Huttenlocher PR, Dabholkar AS. 1997. Regional differences in synaptogenesis in human cerebral cortex. J Comp Neurol 387:167–178.

71 Gage TB. 1998. The comparative demography of primates. Ann Rev Anthropol 27:197–221.

72 Lehninger AL. 1982. Principles of biochemistry. New York: Worth Publishers.

73 Rushton J-P. 1995. Race, evolution, and behavior. New Brunswick, NJ: Transaction Publishers.

74 Armelagos G. 1995. Race, reason, and rationale. Evol Anthropol 4:103–109.

75 Harpending HH. 1995. Human biological diversity. Evol Anthropol 4:99–103.

76 Lovejoy AO. 1936. The great chain of being. Cambridge: Harvard University Press.

77 Fleagle JG. 1988. Primate adaptation and evolution. New York: Academic Press.

78 Fleagle JG. 1999. Primate adaptation and evolution, 2nd ed. New York: Academic Press.

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