LIFE IN THE SLOW LANE REVISITED: ONTOGENETIC SEPARATION BETWEEN CHIMPANZEES AND HUMANS

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ABSTRACT  This study investigates the evolution of human growth by analyzing differences in body mass growth trajectories among 3 populations—the Ache of eastern Paraguay, the U.S. (NHANES 1999-2000) and captive chimpanzees. The relative growth statistic "A" from the mammalian growth law is allowed to vary with age and proves useful for comparing growth across different ages, populations and species. We demonstrate ontogenetic separation between chimpanzees and humans and show that interspecific differences are robust to variable environmental conditions. The human pattern of slow growth during the lengthened period from weaning to the beginning of the adolescent growth spurt is found among the Ache (low energy availability and high disease load) and also in the U.S. (high energy availability and low disease load). We suggest that selection has acted to decrease human growth velocities during childhood to allow more time for increased cognitive development with lower body maintenance costs that offset increased metabolic investment to neural and immune function.
Primate life histories are characterized by long juvenile periods with slow growth rates in comparison to other orders (Charnov and Berrigan, 1993; Pereira and Fairbanks, 1993; Charnov, 2004). Chimpanzees and especially humans delay the beginning of the adolescent growth spurt much longer than expected for growth-spurting primates of our size, condensing the growth spurt to later ages (Leigh, 2001). The delayed growth is matched by late reproduction in that average age at first reproduction is about 13.1 years for wild chimpanzee females (Sugiyama, 2004) versus 19.7 years for natural fertility hunter-gatherer females (Kaplan et al., 2000). In terms of a successful and evolving life history strategy, why do humans wait so long to grow fast and to reproduce?

Slow human growth is an essential component of the human life history and its characteristics may have implications for a better understanding of human life history evolution. Leigh (2001) presents various models for the evolution of slow human growth. These include explanations invoking brains and learning, adult mortality (Charnov, 1993), metabolic risk aversion (Janson and van Schaik, 1993) and future investment (Kaplan et al., 2000; Kaplan and Robson, 2002; Kaplan et al., 2003). Following his conclusion we agree that the latter model best fits growth patterns as it incorporates important aspects from other models, including learning, energetic constraints and investments to mortality reduction (see below). An emphasis here is to integrate slow human growth into a life history strategy that emphasizes high neural investment early in life with delayed returns in adulthood. A prerequisite for such a process is adult mortality rates that are sufficiently low to allow returns to be realized.
Explaining differences in growth patterns between chimpanzees and humans is paramount to explaining the evolutionary divergence that has occurred since the common chimpanzee/human ancestor some 6 million years ago (Ruvolo et al., 1991; Shoshani et al., 1996). In this paper we model and graph growth trajectories in Ache horticultural-foragers, industrial humans (NHANES 1999-2000) and captive chimpanzees (Leigh and Shea, 1996). Differences in the growth trajectories between these two closely related sister species at different levels of energy availability can help our understanding of the evolution of the long period of arrested growth during human childhood and juvenility, and its relation to human brain size and mortality regimes.

**Brain size and development**

The head and brain grow at rates that are different than the rest of the body. Cranial size rises to adult levels in humans by around age 7-10 (Mann, 1984; Jolicoeur et al., 1988a; Caviness et al., 1996; Matsuzawa et al., 2001) but the brain is already 80% adult size by age 4 (Cabana et al., 1993; Leigh, 2004). Pruning (synapse elimination) and myelination (neuronal insulation) continues well into adulthood (Hashimoto et al., 1990; Cabana et al., 1993; Pujol et al., 1993; Benes et al., 1994; Pfefferbaum, 1994; Durston et al., 2001). Leigh (2004) has demonstrated that chimpanzee brains grow relatively slower (using specific velocity) than human brains until around 18 months of age, after which relative brain growth is approximately the same in both species. Brain growth slows considerably in both species well before growth spurts in overall body mass.

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1 Unfortunately wild chimpanzee growth data to our knowledge are only available from Gombe (Pusey, 1990) and have not been made publicly available.
Long juvenile periods are often interpreted as being necessary to develop sophisticated cognitive capacities to function competitively as an adult in complex socioecologies. The human brain is over three times larger than expected for an anthropoid primate of our size (Falk, 1980), the neocortex is 3.6 times larger than expected, and the human brain exhibits a more gyrified (measure of the amount of cortical folding) prefrontal cortex (Rilling and Insel, 1999). This encephalization with an elaborate folding structure is probably associated with an increase in higher cognitive functions like social intelligence, symbolic thinking and executive function (Grafman, 1995). Given that strong relationships exist among longer juvenile periods and various measures of relative brain size, especially the non-visual neocortex ratio, in primates (Joffe, 1997), it is likely that encephalization in humans is directly tied to longer subadult periods.

**Provisioning and juvenile dependence**

Whereas weaned primates generally have to provide most of their own energy budget (Pereira and Fairbanks, 1993), an interesting feature of human social systems is that children, juveniles, adolescents (*sensu* Bogin, 1999), and even young adults are routinely provisioned (Kaplan et al., 2003). While brain growth is not energetically expensive (Kuzawa, 1998), the nervous system is metabolically expensive to maintain. However, the high-quality human diet and accompanying decreases in gut size may partially offset the increasing metabolic costs of the brain (Leonardo and Robertson, 1994; Aiello and Wheeler, 1995).
To our knowledge, the adolescent growth spurt does not appear to correspond to an increase in individual production. Forager boys and girls are producing less than they consume throughout development thereby accruing a large energy "debt" that is paid out by parents and other adult members in the community (Kaplan et al., 2000). Some have proposed that human children in traditional societies can produce significant amounts of food in certain ecological contexts and especially during certain seasons (Bleige Bird et al., 1995; Hawkes et al., 1995; Blurton Jones et al., 1997; Tucker, 2002). However, in terms of total calories and macronutrients provided to a hunter-gatherer diet, difficult-to-acquire extracted resources appear to be much more important than easily acquired foods (Kaplan, 1997; Kaplan et al., 2000; Cordain et al., 2002) and the ability to produce is delayed accordingly, especially for hunting (Walker et al., 2002). Therefore we are left wanting for an explanation for delaying a growth spurt that is largely supported by adult provisioning. It is important to explore alternatives of energetic allocation to growth, such as investments in mortality reduction in the form of immune function.

**Mortality regimes**

Humans and chimpanzees demonstrate widely differing patterns of mortality. The age-specific mortality profile among free-living chimpanzees decreases rapidly after infancy to about 4% per year by ages 10-11, with little increase until around age 22 and strong mortality rate increases thereafter (Hill et al., 2001). In contrast, mortality among human foragers, specifically the !Kung (Howell, 1979), Ache (Hill and Hurtado, 1996) and Hadza (Blurton Jones et al., 2002), decreases to a much lower rate (about 1% per year) and remains low with little increase between 10 and 40 years of age. About 35% of
wild chimpanzees live to age 15 with an expected lifespan thereafter of 15 more years to the age of 30, whereas 60% of modern hunter-gatherers survive to age 15 with an expected lifespan thereafter of 39 more years to the age of 54 (Hill et al., 2001).

The contrast between chimpanzee and human mortality regimes is striking and conceivably has accompanied other significant evolutionary changes in life history parameters, namely larger brain size and longer juvenile periods. With decreased juvenile mortality, humans can invest more time and energy (e.g., brain development and body growth) during a less dangerous juvenile period with a higher probability of reaching maturity. With decreased adult mortality, the benefits of large investments as a juvenile payoff in terms of increased ability and lengthened time to produce and provision offspring (Hill and Kaplan, 1999; Kaplan et al., 2000).

Reduced adult mortality from extrinsic causes selects for delayed reproduction and extended life histories (Stearns, 1992; Charnov, 1993). Optimality models that allow the organism to invest in mortality reduction (i.e., intrinsic mortality) demonstrate that decreases in extrinsic mortality promote increases in investment to reduce intrinsic mortality that in turn also increase time spent in development (Kaplan et al., 2000). The same autocatalytic process occurs for longer lifespans and brain size (Kaplan and Robson, 2002). However, the empirical reality is that some of the longest lived organisms in the world have small brains or no brains (e.g., trees, sessile worms on the ocean bottom, and tortoises). Thus longer lifespan does not always favor increases in neural investment. Importantly, benefits to investment in brain capital must accrue across the long adult lifespan, leading to positive selection for brain size.
Human sociality has many features that serve as behavioral mechanisms for reducing mortality. Compared to chimpanzees, human invest more in their offspring (Lancaster and Lancaster, 1983). Sickness, morbidity, injury, and accidents are common events in traditional human societies and strongly affect development, mortality and fecundity (Hill and Hurtado, 1996; Kuzawa, 1998; Panter-Brick, 2001; Sugiyama, 2004; McDade, 2003). Human cooperation and food sharing among kin and nonkin palliate and buffer against such health insults (Gurven et al., 2000b, 2001; Hill, 2002; Gurven, 2004; Gintis et al., 2003). Empirical studies with hunter-gatherers support the proposition that human cooperative behavior buffers the impact of life-threatening situations. For example, Ache who frequently help others, regardless of their actual production, receive more aid than average when they become sick or injured (Gurven et al., 2000a).

Physiological differences between humans and chimpanzees may also contribute to lower mortality in humans. An elevated immune function may partially account for some differences in mortality rates during the subadult period (see Discussion). Humans also deposit an exceptional amount of fat in utero demonstrating nearly four times as much fat mass at birth as expected for a mammal of our size (Kuzawa, 1998). Large stores of fat may be necessary as an energetic buffer to protect the large human brain during shortfalls and as storage of long chain polyunsaturated fatty acids needed for proper brain development (Cunnane and Crawford, 2003). Though we proceed to analyze rates of change in overall body size, it is important to remember that the average composition of a human body is considerably fatter than that of a chimpanzee.
METHODS

Study populations

We chose two very different human populations to analyze differences in body mass growth trajectories: the Ache (low energy availability and high disease load) and the U.S. (high energy availability and low disease load). Particular attention is given to characteristics of growth trajectories that are mainly driven by environmental factors and others that are robust species-level patterns. Comparisons with chimpanzees using relative growth statistics may highlight key developmental periods where investments in neural and immune function are favored over growth in humans.

Ache. The Ache of Eastern Paraguay were full-time hunter-gatherers into the latter half of the 20th century (Hill and Hurtado, 1996). The data in this study were collected at the communities of Arroyo Bandera, Chupa Pou and Kuetuvy, all near the Mbaracayu Natural Reserve into which the Ache continue to make foraging treks. Activity levels are high in the forest where family groups often move each day with men spending about 7 hours per day hunting in the forest (Hill and Hawkes, 1983). Women spend over 6 hours per day moving camp, harvesting and processing food, and conducting other miscellaneous work activities (Hurtado et al, 1985). On the reservation, time is often spent working in agricultural fields or on household tasks or playing games of soccer and volleyball.

While Ache boys and girls on average are around the lowest fifth percentile of U.S. body mass and height, this difference may be largely environmental. Several Ache
children have been adopted by Americans and have consequently grown to be much taller and heavier than their age mates. At age 22 one boy who was adopted at age 4 was 10 cm taller than the average of other Ache boys his age (Hill and Hurtado, 1996).

A genealogical approach with interview-generated age ranks was used to age all individuals in the population born before fieldwork commenced in the late 1970s (see Hill and Hurtado, 1996). Ages for individuals born during the fieldwork period are exact to the day. Weights are available for 262 males and 222 females for a total of 1,020 body weights taken on males and 1,015 on females that are age 25 or younger.

Weights were taken during the years of 1980-'85, ‘87, ‘89, ‘92-94, ‘96-2001, encompassing all months of the year. The sample population at any one data collection session consisted of all the individuals that happened to be present in the village. However, absentee individuals were often (but not systematically) sampled later upon their return. All individuals were weighed using various brands of step-on bathroom scales accurate to the nearest tenth of a kilogram. Participants were weighed without shoes or heavy clothing such as jackets. While different researchers and scales introduce some methodological error, we feel that this is likely small in comparison to day-to-day variation in body mass. We found that people's weight could fluctuate as much as 1-2 kg in a single day depending on satiation and hydration levels.

**United States.** U.S. growth data are taken from NHANES which is a survey conducted by the National Center for Health Statistics designed to compile health and dietary information of people in the U.S. The survey consists of home interviews and health tests conducted in a mobile examination center. The NHANES 1999-2000 database
(http://www.cdc.gov/nchs/about/major/nhanes/NHANES99_00.htm) includes body mass and age to the nearest month for a mixed-ethnic sample of 2,664 males and 2,627 females under the age of 25.

**Chimpanzee.** Growth rates for captive chimpanzees are taken from Leigh and Shea (1996) who cross-sectionally modeled body growth in 22 female and 23 male *Pan troglodytes* in zoological parks and primate centers with known ages to the day. Additional information on the sample population can be found in Leigh (1992ab; 1994ab).

**Growth modeling**

**JPPS growth model.** This paper uses the JPPS parametric regression model developed by Jolicoeur, Pontier, Pernin and Semp (1988b) to describe the growth in body mass for the Ache, U.S. and chimpanzees. The JPPS model was originally developed to model human statural growth and has been found to fit growth rates better than Preece-Baines (Jolicoeur et al., 1991) and other parametric growth models (Hauspie and Molinari, 2004). Moreover, unlike some other models, the JPPS is designed to fit the entire growth period and asymptotes at adult size giving it a parameter that makes biological sense (Hauspie, 1989). The JPPS model used here has the following form:

\[
\text{body weight}_{ij} = B \cdot \left( 1 - \frac{1}{1 + \frac{\text{total age}_{ij}}{D_1} C_1 + \frac{\text{total age}_{ij}}{D_2} C_2 + \frac{\text{total age}_{ij}}{D_3} C_3} \right) + \mu_i + v_j + \lambda_i
\]
where total age, or biological age, refers to age since conception (postnatal age plus gestation length, 0.64 years for chimpanzees, 0.75 years for humans) for individual \( i \) at measurement date \( j \); parameter \( B \) is adult body mass; \( C_1, C_2, C_3, D_1, D_2 \), and \( D_3 \) are six function parameters that describe the flexible shape of the growth curve; and the 3 Greek symbols are random effects estimated for each individual \( i \), each month, and each year in the sample. There are some seasonal and year-to-year effects on Ache body mass but no secular trend in body weight changes. Total age is used so that the model passes through the origin when age is zero because the size of a single cell at fertilization is negligible. However, postnatal age is used in all graphs for easier comparability and because no prenatal mass data are used.

The ideal methodology would be to fit the JPPS model separately to each individual’s longitudinal data and then average each of the parameter estimates across all individuals. This method would most closely match the true growth trajectory of an average individual in the population. However, since Ache body weight data were not taken systematically for each individual at regular intervals, this is not possible. Also, Leigh and Shea (1996) fit the model to cross-sectional chimpanzee growth (see Leigh, 1994a for justification), and so it is preferable to compare the samples using a similar methodology.

**Statistical analysis.** The JPPS model is used to analyze the growth of Ache and U.S. populations for males and females under the age of 25. The model is fit to the Ache data using the non-linear mixed modeling macro in SAS (PROC NLINMIX). This macro allows both fixed and random effects. Total age is the only fixed effect in the model.
because interest is solely on the data values included in this independent variable. The following random effects are used in the model—1) year of data collection, 2) month of data collection, and 3) an individual identifier. The latter constructs a parameter for each individual and accounts for individual variation that may exist independently of age (i.e., unmeasured heterogeneity). For example, if an individual is sampled several times and is consistently underweight for his/her age, their parameter will be negative. This method is preferable to using each individual as a single data point, especially since most individuals enter the sample at various ages. The individual random effect controls for the lack of independence inherent in using multiple measurements on any one individual without making the assumption of a homogenous population (Verbeke, 1997).

Maximum likelihood estimates and associated standard errors are given for each parameter in the model. Unfortunately there are no accepted global goodness of fit tests for NLINMIX models. However, for both the Ache and U.S., the fitted model can be shown to approximate non-parametric curve fitting (e.g., LOWESS) in plots of weight by age and weight velocity by age. Leigh and Shea (1996) also find that JPPS models and LOWESS smooth curves give comparable approximations of growth trajectories for chimpanzees. As demonstrated below, JPPS models also mimic longitudinal growth trajectories.

**Growth law.** Growth rates are size-dependent across mammals and are often described according to the growth law equation:

\[
\frac{\hat{m}}{\hat{t}} = A \cdot m^{0.75}
\]
where $m$ is mass and $A$ is a constant. The 0.75 power is the empirically-derived slope of the mammalian line in Figure 1 and probably results from fractal branching of capillary networks and allocation of metabolic energy at the cellular level and (see West et al., 1997; 2001).

Smaller primates up to several kilograms in size appear to fit the typical mammalian scaling relationship while larger primate species grow at rates closer to the reptilian pattern (Figure 1). With respect to growth rate alone, humans are more like a boa constrictor than a typical mammal! This downward shift in growth rate seen in Figure 1 is reflected in the growth constant $A$, or the "height" of the mammalian growth law equation:

$$A = \frac{\dot{m}}{\dot{t}/m^{0.75}}$$

Most mammals demonstrate "$A$" values around 1, whereas the mean primate value is 0.42 (Charnov, 1993). This measure has also been used as an estimate of growth and yearly production of offspring (Charnov and Berrigan, 1993).

Values for "$A$" are generally taken as a constant for a clade, but it can be allowed to vary among species or even with age. An age-varying "$A$" value represents the relative allometric growth ratio at a certain size. Direct comparison of age-specific growth rates must control for differences in body size. The "$A$" value has an advantage in this respect because it is a relative statistic determined by dividing growth rate by a direct proportionality of metabolic rate (West et al., 1997; 2001 but see White and Seymour, 2003 who argue for a $^{2/3}$ scaling relationship). Below we demonstrate that Ache children
exhibit extremely low age-specific "A" values that approach 0.2 from age 4 to 10, approximately ½ the value for chimpanzees at that age. Low "A" values may suggest an organism is diverting growth/production resources from its metabolic budget to combat mortality (Charnov, 2004).

RESULTS

Chimpanzee vs. human growth patterns

**Body size.** Table 1 gives JPPS parameter estimates and standard errors for *Pan troglodytes*, Ache and U.S. growth. The B parameter represents asymptotic young adult body size. Chimpanzees males weigh around 57.3 kg, in between Ache men (59.5 kg) and Ache women (55.6 kg), whereas chimpanzees females are considerably smaller at 46.0 kg. Sexual dimorphism statistics (male/female mass) vary from the Ache at 1.07 to the U.S. at 1.16. Interestingly, sexual dimorphism in adult height for both these populations is approximately 1.08. Sexual dimorphism in body size for the captive chimpanzee population is 1.25, which matches reported values for wild chimpanzees at Gombe (Pusey, 1990; Pusey et al., n.d.).

[Insert Table 1 about here]

Figure 2a gives growth trajectories for males and females in each population (Ache, U.S. and chimpanzee). While human infants in both populations grow faster than chimpanzee infants, male and female chimpanzees are larger than humans from around ages 6 to 10 and male chimpanzees are larger until age 12. One notable difference between the Ache and U.S. is that Ache females are on average larger than males for a longer period, throughout childhood and until around 16 years of age.
Superimposing the longitudinal growth for two of the individual females in Figure 2a demonstrates that both girls display average growth until before their adolescent growth spurts (Figure 2b). However, individual A has a larger overall spurt and ends up larger than individual B. Individual A has a positive random effect; individual B's is negative. Like most other human children under normal to favorable conditions (Tanner, 1962; Bogin, 1999), both these individuals show a distinct growth spurt. Whether or not chimpanzees have true growth spurts is still an important debate (see below).

Growth velocities. Figure 3 compares growth velocity (rate of change in the JPPS growth model) of the Ache, U.S. and chimpanzees separated by sex. The age at peak weight velocity is much later for both human groups: 13.2 years for Ache girls and 11.5 for U.S. girls, compared to 7.5 years for captive chimpanzee females. The same trend is present in the male samples. The age at peak weight velocity for Ache boys is 14.9 years and 13.6 for U.S. boys in contrast to 9.5 for captive chimpanzee males. An especially unique feature of the two human samples is the characteristic dip in weight velocity starting at age 2 and continuing until age 9 to 12, whereas captive chimpanzee growth continues to accelerate from birth until the age at peak weight velocity.

In a hypothetical exercise, one can quickly calculate how the chimpanzee growth velocity curve would have to be manipulated in order to match the age at peak weight velocity with the human curve. For the age at peak weight velocity of male chimpanzees to match the human samples, the typical chimpanzee growth that actually takes place in 1
year must be stretched over a 1.6-year span to match the Ache and 1.4-year span to match the U.S. sample. In females, the chimpanzee growth velocity curve must be stretched by 1.8 in order for the age at peak weight velocity to match up with the Ache and 1.5 to match up with the U.S.

Analysis of longitudinal growth curves for individual Ache demonstrate a quality of the growth curve that is obscured by cross-sectional modeling. Because boys and girls have growth spurts at a variety of ages, using cross-sectional data creates overlapping spurt periods that obscure the true height of the peak in weight velocity (Hauspie et al., 2004). True weight velocity peaks range higher than 6-8 kilograms per year as shown in Figure 3. However, as long as individual growth spurts are approximately normally distributed around the mean peak weight velocity, then the average age at peak weight velocity is preserved in the analysis. Moreover, it is the earlier period from weaning to the age at takeoff velocity that is of primary theoretical interest in this paper, though better measures of peak weight velocities will be necessary to assess the real costs of the growth spurt.

"A" values. From the growth law, "A" represents the age-specific relative allometric growth. Because of population, sex, and species differences in size across development, "A" values are more directly comparable than weight velocities. Human children and juveniles demonstrate smaller "A" values than chimpanzees starting at birth and continuing to age 8 or 9 in girls and 10 or 11 in boys (Figure 4). The "A" values for Ache drop down to 0.2, about ½ the juvenile chimpanzee values. The implication is that
humans are investing significantly less of their metabolic capacity into growth than chimpanzees are investing into growth during this developmental period.

[Insert Figure 4 about here]

Human growth is distinguished by the exceptionally long interval from weaning to the age at takeoff velocity (beginning of the growth spurt). Age at takeoff velocity in both Ache and U.S. boys is approximately equal, around 10.5 years of age. The age at takeoff velocity for Ache girls is about 1.5 years later than for U.S. girls, 9 versus 7.5 years, respectively. In cross-sectional data, values for the age at takeoff velocity and peak "A" values are similar for U.S. and Ache boys and girls. The major difference between the human sexes is that the age at takeoff velocity is earlier for girls; other characteristics of the "A" curve show little sexual dimorphism (Figure 4). Comparing Figures 3 and 4 highlights the importance of using "A" values to compare growth trajectories instead of weight velocities. Weight velocities can confound comparisons since populations are likely different sizes at any age. Using "A" values makes the "growth spurt" in female chimpanzees disappear, and the spurt in male chimpanzees is largely attenuated. "A" values better demonstrate the contrast between chimpanzee and human growth patterns and the similarity in Ache and U.S. growth. Both infant growth and the peak weight velocity in the Ache and U.S. become roughly equal. Therefore, differences seen in infant growth and peak weight velocity in the velocity curves (Figure 3) might not represent significantly different metabolic allocation decisions. Importantly, there is no slow growth period for chimpanzees akin to the diapause in human growth between weaning and the age at takeoff velocity.
DISCUSSION

Environmental growth constraints such as food availability, disease load and activity levels are much lower among industrial populations compared to the Ache. In spite of the reduced environmental constraints, industrial populations still demonstrate considerably slower growth rates than chimpanzees, though slightly faster than the Ache. The slow growth period in the U.S. sample lasts nearly as long for girls, and equally as long for boys, as in the Ache sample. Using longitudinal data on a modern industrial population, Iuliano-Burns et al. (2001) found the average age at peak weight velocity to be 12.3 for girls and 13.8 for boys. These ages at peak weight velocity are only about 1-2 years earlier than those demonstrated for Ache teenagers. Assuming few genetic differences, these results suggest that while energetic limitations do affect the growth velocity curve, the effect is small in comparison to the large interspecific programmed growth pattern differences between chimpanzees and humans.

Evolution of human growth

We suggest that selection has produced the lengthy period of slow growth and the late age of takeoff velocity in humans. The question is what exactly humans are waiting for during this period. The energetics of the human growth spurt could be paid earlier with food provisions from others in both the Ache and U.S. social systems. Why not simply mature earlier when an individual's food production rate is only slightly lower? Instead, human children have adult-size brains but underdeveloped bodies for many years before the adolescent growth spurt. The potential benefits of larger body size during the period of slow growth are apparently outweighed by the maintenance costs that family
members would have to pay to support a large and still highly dependent offspring. The solution then is to stay relatively small for many years by delaying puberty and the growth spurt. A more developed brain replete with a repertoire of social and foraging skills is likely essential to be a competent adult. This learning/sculpting process of the brain probably requires considerable time (15+ years) in order to develop competence in complex social and/or foraging niches. Importantly, conserved energy not invested into growth may fuel the energy expensive brain, lower mortality through immune function, and/or allow for parents to divert resources to other offspring.

**Future direction**

**Wild chimpanzee growth.** Wild chimpanzee growth data are available from Gombe (Pusey, 1990). Because of increased morbidity and energetic constraints, wild chimpanzees appear to grow longer and slower with a smaller adult body size (adult female median = 31 kg; male = 39 kg; Pusey et al., n.d.) than their captive counterparts. Better comparisons with wild chimpanzees await a modeling procedure, such as that used in this paper, of weight velocity and "A" values by age. It is possible that true growth spurts are seen only in humans. Hamada and Udo no (2002) show that captive chimpanzees living in favorable conditions do not show a growth spurt in summed length, suggesting that catch-up growth, as opposed to a robust growth spurt as seen in humans (Bogin, 1999), characterizes adolescent growth rates in chimpanzees. More longitudinal chimpanzee data, preferably from free-ranging populations, are needed to resolve this question.
Immune function. Less well appreciated than constraints of brain size on body growth are the metabolic costs of immune function. Elevated immune function may partially account for the low human mortality rates during the juvenile period. One general indicator of immune function/response is the thymus, which is involved in the production and selection of lymphocytes (Roitt et al., 1998). The human thymus is adult size in the first year of life, continues to expand to over 150% of adult weight at about 8-9 years old, and returns to adult size by age 20 (Elia, 1998). Given the developmental pattern of the thymus and uncertainty concerning its body size allometry, one comparative measure may be infant thymus size. A human infant thymus weighs around 11.2 g versus 4.3 g for a chimpanzee infant with corresponding body masses of 3.5 kg and 2.9 kg, respectively (Kennard and Willner, 1941; Spector, 1956). These thymus sizes tentatively support a heightened immune function investment in human infants, yet lymphocyte cell counts in human adults are similar to monogamous primates and therefore much lower than polygamous chimpanzees (Nunn et al., 2000; Nunn, 2002). Thus the human pattern of a large infant thymus but with unremarkable adult white blood cell counts may suggest heightened immune function early in life only.

Importantly, preliminary evidence suggests that immune function costs compete with body growth, most notable in regimes of low energy availability (Panter-Brick, 2001; McDade, 2003). However, direct energetic costs of immune function for humans and chimpanzees are lacking. Undoubtedly, immunocompetence is a complex phenomenon (Long and Nanthakumar, 2004) and its quantification requires multiple monitoring and challenge techniques such as those used in bird studies (Norris and Evans, 2000; Blount et al., 2003). Nonetheless, developmental periods characterized by
low "A" values suggest that the most important age range of analysis is from weaning to around age 10 in humans.

**Brain evolution.** The ultimate explanation for the benefits of the large primate brain remain unresolved. Evaluating the marginal benefits of social versus foraging returns to investment in brain size (Dunbar, 2003) appears to be an especially informative avenue for future investigations. Analyzing what humans and other primates are doing during the long juvenile period should prove valuable. Many cognitive and motor skills are gained quickly during childhood, but not mastered as easily if the learning process begins later (e.g. language; Kim et al., 1997). Documenting sensitive periods of time when the brain is capable of learning important foraging and social tasks more efficiently and thoroughly (Bock, 1995; 2002) can elucidate selective pressures for certain cognitive tasks. Moreover, substantial neurophysiological changes that continue into young adulthood (e.g., Cabana et al., 1993; Pujol et al., 1993; Benes et al., 1994; Pfefferbaum, 1994; Durston et al., 2001) may offer clues to the importance of long human learning periods.

**CONCLUSION**

Humans demonstrate long periods of slow growth from weaning to age at takeoff velocity for the growth spurt. Longer lifespans and larger brain size appear to be tightly coevolved with this slow growth pattern, although this association is not obligatory in other long-lived organisms. Humans may be an extreme case where low growth rates and long juvenile periods result from a complex foraging and/or social niche where it pays to
save on body size maintenance costs by delaying the growth spurt and investing in immune function until cognitive abilities reach closer-to-adult levels of proficiency.

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FIGURE CAPTIONS

Figure 1. Log-log plot of growth rates (upper quartile averages) by adult body mass for 45 reptiles and 152 mammals including 16 primates (data from Case, 1978).

Figure 2. "Distance" curves (body size estimates by age) for a) U.S., Ache and chimpanzee females and males using the JPPS growth model. b) Longitudinal "distance" curves for two Ache females compared with the JPPS growth model.

Figure 3. Growth velocity (rate of change in the "distance" curve) of Ache, U.S. and captive chimpanzees for a) females and b) males.

Figure 4. “A” values for Ache, U.S. and captive chimpanzees for a) females and b) males calculated by dividing age-specific weight velocity at each age by mass to the 0.75 power at that same age.

TABLE CAPTIONS

Table 1. JPPS parameter estimates and standard errors. Values for captive Pan troglodytes are from Leigh and Shea (1996) and modeled for the U.S. sample (NHANES 1999-2000). JPPS maximum likelihood estimates and standard errors for the Ache control for month and year of data collection and unmeasured individual heterogeneity through the use of random effects.
LITERATURE CITED


