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 three populations: the Ache of eastern Paraguay, the US (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 US (high energy availability and low disease load). The human growth pattern contrasts with that of the chimpanzee, where absolute growth rates and relative "A" values are faster and less prolonged. We suggest that selection has acted to decrease human growth rates to allow more time for increased cognitive development with lower body-maintenance costs. Am J Phys Anthropol 129:577–583, 2006.   © 2005 Wiley-Liss, Inc.

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) vs. 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 markedly delay their adolescent growth spurt and age of first reproduction?

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 six 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).[1] Differences in growth trajectories between these two closely related species can help our understanding of the evolution of the long period of arrested growth during human childhood and juvenility, and its relationship to the evolution of other human life-history traits.

RELATIVE METABOLIC GROWTH

Comparisons of growth patterns across ages and between different populations should avoid relying solely on growth velocities, as they are directly dependent on body size. For instance, a fetus growing at 5 kg/year is obviously a very different growth decision than a 50-kg teenager growing at the same rate. To make better comparisons between individuals of different sizes, some type of relative growth velocity measure is useful.

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

$$\frac{dm}{dt} = A \cdot m^{0.75}$$

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 the fractal branching of capillary networks and allocation of metabolic energy at the cellular level (West et al., 1997, 1999, 2001; but see White and Seymour, 2001, who argued for a two-thirds scaling for metabolic rate).

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

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[1]Unfortunately, wild chimpanzee growth data, to our knowledge, are only available from Gombe (Pusey et al., 2005), and have not been made publicly available.
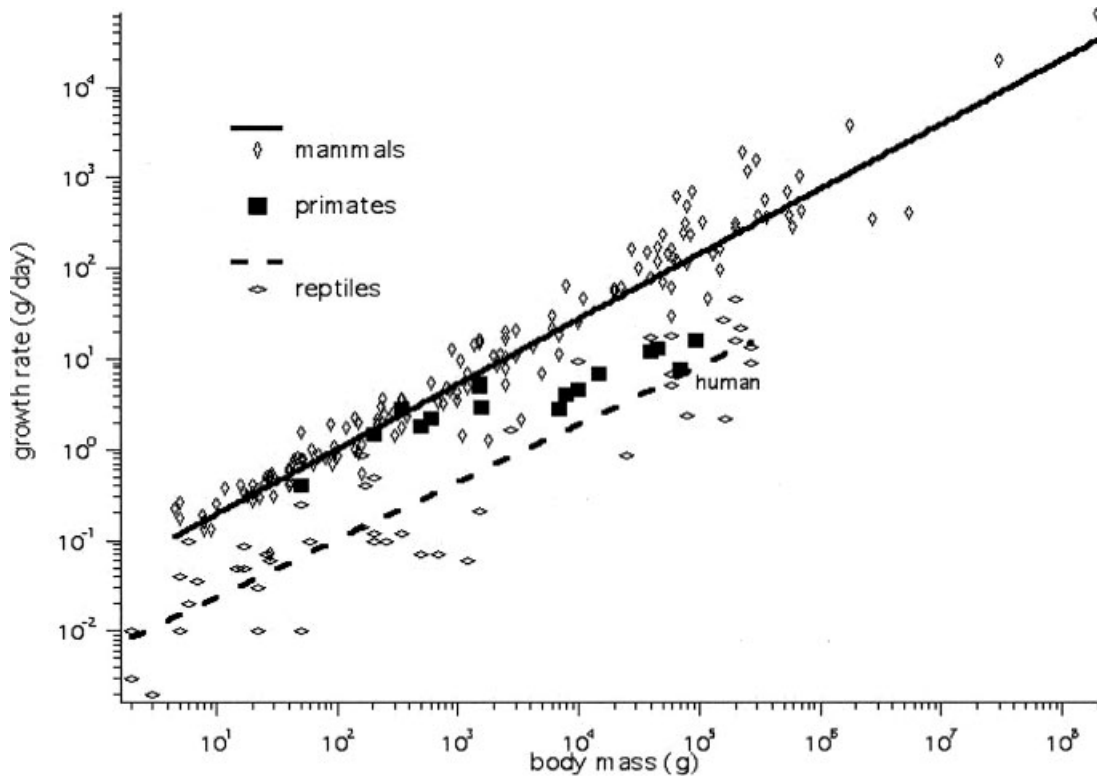
Fig. 1. Log-log plot of growth rates (upper quartile averages) by adult body mass for 45 reptiles and 152 mammals, including 16 primates (data from Case, 1978).

reptilian pattern. With respect to growth rate alone, humans are more like a reticulated python than a typical mammal! This downward shift in growth rate seen in Figure 1 is reflected in the growth constant "*A*," or "height" of the mammalian growth law equation:

$$A = \frac{\mathrm{d}m/\mathrm{d}t}{m^{0.75}}.$$

Most mammals demonstrate "*A*" values around 1, whereas the mean primate value is 0.42 (Charnov, 1993). "*A*" values were used as estimates of growth and yearly production of offspring (Charnov and Berrigan, 1993). Values for "*A*" are generally taken as a constant for a clade, but 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. In this way, direct comparison of age-specific growth rates can be made that control for differences in body size. The "*A*" value has an advantage in this respect, because it is a relative statistic determined by dividing the growth rate by a direct proportionality of metabolic rate, and therefore contains important scalar information from the growth law.

We hypothesize that "*A*" values for humans are much less than those for chimpanzees, regardless of socioeconomic circumstances. The purpose of this paper is to explore the use of "*A*" values in identifying pronounced differences in ontogenetic stages of human and chimpanzee growth patterns. If we find that human investments in growth are markedly reduced compared to chimpan-

zees during key developmental stages, we can speculate on the evolutionary importance of investments to other competing energetic requirements. Some likely candidates include the energy needed to support a large brain capital, maintain a low mortality regime, or avoid body-maintenance costs during a long subadult period.

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 US (high energy availability and low disease load). In this manner, particular attention can be given to characteristics of growth trajectories that are mainly driven by environmental factors, and others that are robust species-level patterns.

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 in 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 hr per day hunting in the forest (Hill and Hawkes, 1983). Women spend over 6 hr 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 agricul-

tural fields or on household tasks, or playing games of soccer and volleyball.

While Ache boys and girls, on average, are at around the fifth percentile of US body mass and height, this difference may be largely environmental. Several Ache children were adopted by families in the US, and consequently grew to be much taller and heavier than their age mates. For example, at age 22 years, one Ache male who was adopted at age 4 years was 10 cm taller than the average of other young adult Ache his age (Hill and Hurtado, 1996). Thus growth differences between Ache and US populations are strongly influenced by environmental conditions.

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 (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 who are age 25 or younger. Weights were taken during the years 1980–1985, 1987, 1989, 1992–1994, and 1996–2001, encompassing all months of the year. The sample population at any one data collection session consisted of all individuals who 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. US 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 US. The survey consists of home interviews and health tests conducted in a mobile examination center. The NHANES 1999–2000 database (online at 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 ages known to the day. Additional information on the sample population can be found in Leigh (1992a,b, 1994a,b).

Growth modeling

JPPS growth model. This paper uses the JPPS parametric regression model developed by Jolicoeur et al. (1988) to describe growth in body mass for the Ache, US, and chimpanzee sample populations. The JPPS model was originally developed to model human statural growth, and was found to fit growth rates better than the Preece-Baines (Jolicoeur et al., 1991) and other parametric growth models (Hauspie and Molinari, 2004). Unlike some other models, JPPS is designed to fit the entire growth period. Moreover, JPPS asymptotes at

adult size with an adult-size parameter that makes biological sense (Hauspie, 1989). The JPPS model used here has the following form:

$$
\begin{aligned}
\text{body weight}_{ij} \\
= B \cdot \left\{ 1 - \frac{1}{1 + \left[\frac{\text{total age}_{ij}}{D_1}\right]^{C_1} + \left[\frac{\text{total age}_{ij}}{D_2}\right]^{C_2} + \left[\frac{\text{total age}_{ij}}{D_3}\right]^{C_3}} \right\} \\
+ \mu_i + \nu_j + \lambda_j
\end{aligned}
$$

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 three 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 (for justification, see Leigh, 1994a), 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 US populations for males and females under age 25. The model is fit to the Ache data using the nonlinear 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 in 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 independent 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 and Molenberghs, 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

TABLE 1. JPPS parameter estimates and standard errors[1]

	B	C1	C2	C3	D1	D2	D3
Ache female	55.63	15.60	3.58	0.67	13.60	14.24	26.95
SE	0.53	1.72	0.93	0.09	0.18	0.71	8.69
Ache male	59.47	15.70	3.00	0.56	15.24	15.74	50.18
SE	0.66	1.84	0.76	0.12	0.19	0.73	32.82
US female	65.76	12.37	3.22	0.60	12.07	12.31	34.13
SE	0.69	3.15	2.16	0.25	0.54	1.63	43.62
US male	76.48	16.96	3.52	0.58	13.98	12.67	46.99
SE	0.85	3.73	1.04	0.16	0.33	0.59	0.58
Chimp female	46.04	1.92	6.95	0.79	8.48	8.93	44.81
SE	0.69	3.32	2.51	2.76	8.30	1.05	747.54
Chimp male	57.33	4.79	19.03	1.22	9.19	9.97	12.12
SE	0.51	2.50	6.24	0.29	0.53	0.40	5.39

[1] Values for captive *Pan troglodytes* are from Leigh and Shea (1996) and modeled for the US sample (NHANES, 1999–2000). JPPS maximum likelihood estimates and standard errors for Ache control for month and year of data collection and unmeasured individual heterogeneity through use of random effects.

Ache and US, the fitted model can be shown to approximate nonparametric curve-fitting (e.g., LOWESS) in plots of weight by age and weight velocity by age. Leigh and Shea (1996) also found 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.

RESULTS

Chimpanzee vs. human growth patterns

Body size. Table 1 gives JPPS parameter estimates and standard errors for *Pan troglodytes*, Ache, and US growth. The *B* parameter represents asymptotic young adult body size. Chimpanzee males weigh around 57.3 kg, 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 US at 1.16. 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., 2005).

Figure 2a gives growth trajectories for males and females in each population (Ache, US, and chimpanzee). While human infants in both populations grow faster than chimpanzee infants, male and female chimpanzees are larger than humans from around ages 6–10, and male chimpanzees are larger until age 12. One notable difference between the Ache and US is that Ache females are, on average, larger than males for a longer period, throughout childhood and until around age 16 years.

Superimposing the longitudinal growth for two of the individual females in Figure 2a demonstrates that both girls displayed average growth before their adolescent growth spurts (Fig. 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. Both these individuals show a distinct growth spurt, as expected of human children under normal to favorable conditions (Tanner, 1962; Bogin, 1999).

Growth velocities. Figure 3 compares growth velocity (rate of change in the JPPS growth model) for the Ache, US, 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 US girls, compared to 7.5 years for captive chimpanzee females. The same
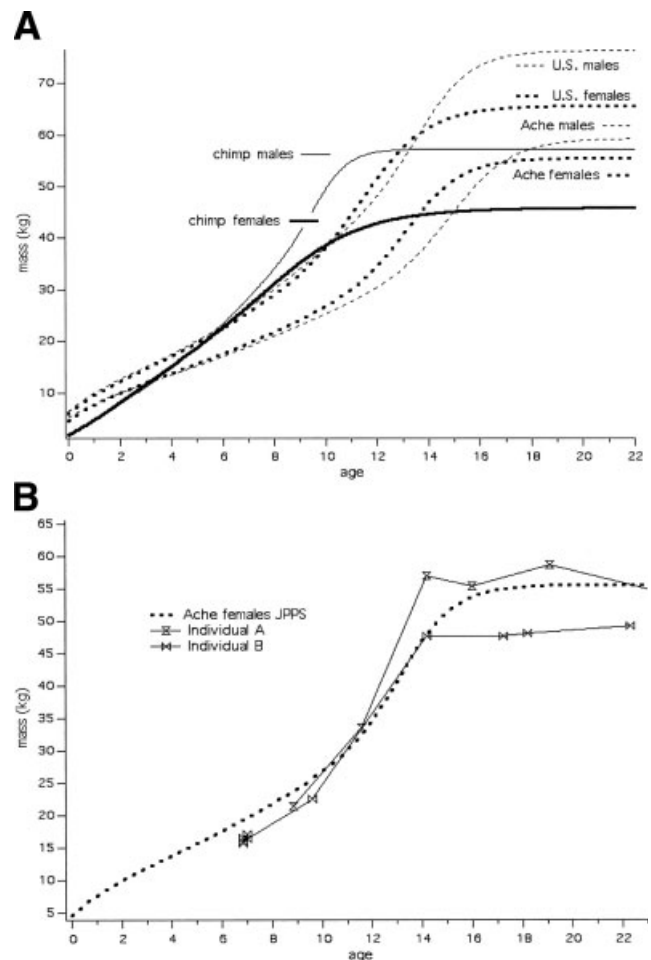
Fig. 2. A: "Distance" curves (body size estimates by age) for US, Ache, and chimpanzee females and males, using JPPS growth model. **B:** Longitudinal "distance" curves for two Ache females compared with JPPS growth model.

trend is present in the male samples. The age at peak weight velocity for Ache boys is 14.9 years and 13.6 years for US boys, in contrast to 9.5 years for captive chimpanzee males.
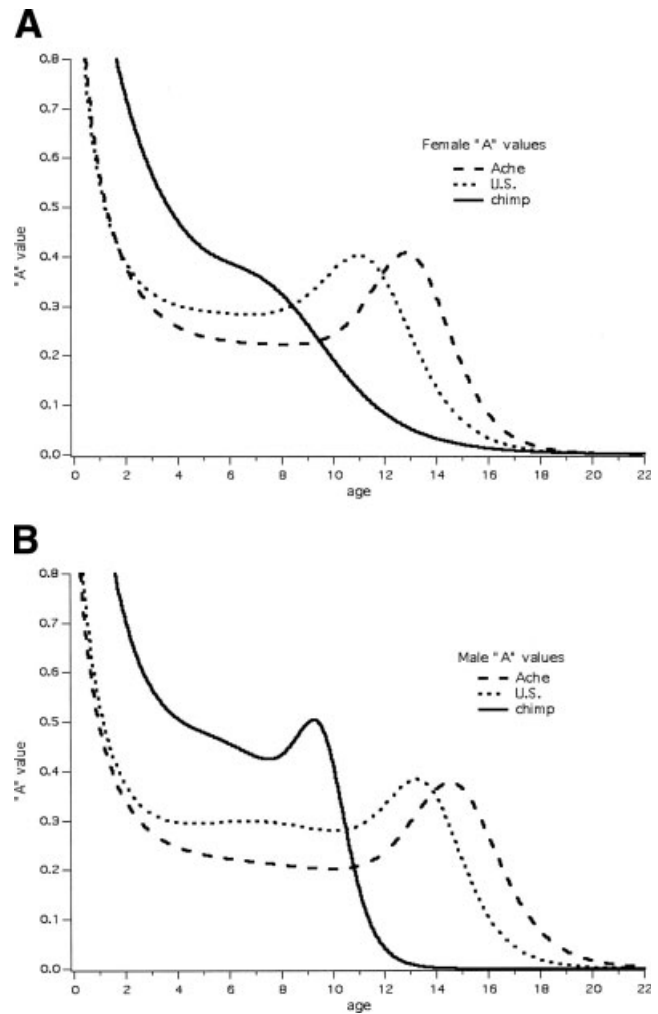
Analyses of longitudinal growth curves for individual Ache demonstrate a quality of the growth curve that is
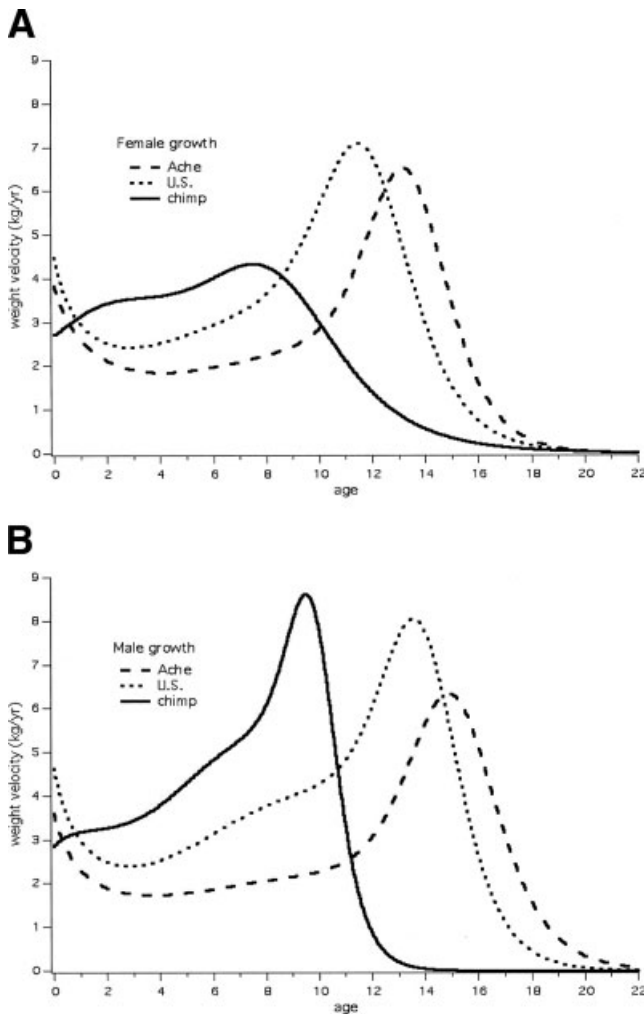
Fig. 3. Growth velocity (rate of change in "distance" curve) of Ache, US, and captive chimpanzees for (**A**) females and (**B**) males.

Fig. 4. "A" values for Ache, US, and captive chimpanzees for (**A**) females and (**B**) males, calculated by dividing age-specific weight velocity at each age by mass to 0.75 power at that same age.

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 (Boas, 1892; Hauspie et al., 2004). True weight velocity peaks range higher than 6–8 kg 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 (beginning of the adolescent growth spurt) 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.

Whether or not chimpanzees have true growth spurts is still an important debate. It is possible that true growth spurts are seen only in humans. Hamada and Udono (2002) showed 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. Because of increased morbidity and energetic constraints,

wild chimpanzees from Gombe (Pusey, 1990) appear to grow longer and slower, with a smaller adult body size (adult female median, 31 kg; male, 39 kg; Pusey et al., 2005) than their captive counterparts. More longitudinal chimpanzee data, preferably from free-ranging populations, and a modeling procedure, such as that used in this paper of weight velocity and "A" values by age, are needed to resolve the issue of whether or not chimpanzees have adolescent growth spurts.

"A" values. From the growth law, "A" represents age-specific relative allometric growth. As mentioned above, because of population 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 years in girls and 10 or 11 years in boys (Fig. 4). "A" values for the Ache drop down to 0.2, about half 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.

Human growth is distinguished by the exceptionally long interval from weaning to age at takeoff velocity (beginning of the growth spurt). Age at takeoff velocity in "A" values in both Ache and US boys are approximately equal, at around 10.5 years of age. The age at takeoff velocity for Ache girls is about 1.5 years later than for US girls (9 vs. 7.5 years, respectively). In cross-sectional data, values for age at takeoff velocity and peak "A" values are similar for US 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 (Fig. 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 US growth. Both infant growth and the peak weight velocity in the Ache and US become roughly equal. Therefore, differences seen in infant growth and peak weight velocity in the velocity curves (Fig. 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 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, the US sample still demonstrates considerably slower growth rates than chimpanzees, though slightly faster than the Ache. The slow growth period in the US 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. These results suggest that while energetic limitations do affect the growth velocity curve, the effect is small in comparison to the interspecific programmed growth pattern differences between chimpanzees and humans.

Humans exhibit extremely low age-specific "A" values that approach 0.2 from weaning to around 10 years of age, approximately half the value for chimpanzees at that age. These low "A" values suggest that humans are diverting growth/production resources from their metabolic budget for other purposes, such as to combat mortality through immune function. Importantly, preliminary evidence suggests that immune function costs compete with body growth, most notably in regimes of low energy availability (Panter-Brick et al., 2001; McDade, 2003). In conjunction or as an alternative hypothesis, humans may also be saving on body-maintenance costs during the long subadult period. A well-developed brain replete with a repertoire of social and foraging skills is likely essential to be a competent adult. This process likely requires considerable time (15+ years) in order to develop competence in complex social and/or foraging niches. The optimal solution, therefore, may be to stay small for many years by delaying puberty and the growth spurt.

CONCLUSIONS

The relative growth statistic "A" proves useful in comparisons of growth across ages, sexes, populations, and species that are not biased by differentials in size. We demonstrate how human growth is characterized by long periods of slow growth from weaning to the beginning of the adolescent growth spurt. Also, this human pattern of slow growth appears robust to variable environmental conditions. In summary, conserved energy not invested in growth and consequent body-maintenance costs may help fuel the energy-expensive brain, lower mortality through immune function, and/or allow for parents to divert resources to other offspring.

ACKNOWLEDGMENTS

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