COMPARATIVE ANALYSIS OF ANIMAL GROWTH: A PRIMATE CONTINUUM REVEALED BY A NEW DIMENSIONLESS GROWTH RATE COEFFICIENT

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The comparative analysis of animal growth still awaits full integration into life-history studies, partially due to the difficulty of defining a comparable measure of growth rate across species. Using growth data from 50 primate species, we introduce a modified "general growth model" and a dimensionless growth rate coefficient β that controls for size scaling and phylogenetic effects in the distribution of growth rates. Our results contradict the prevailing idea that slow growth characterizes primates as a group: the observed range of β values shows that not all primates grow slowly, with galago species exhibiting growth rates similar or above the mammalian average, while other strepsirrhines and most New World monkeys show limited reduction in growth rates. Low growth rate characterizes apes and some papionines. Phylogenetic regressions reveal associations between β and life-history variables, providing tests for theories of primate growth evolution. We also show that primate slow growth is an exclusively postnatal phenomenon. Our study exemplifies how the dimensionless approach promotes the integration of growth rate data into comparative life-history analysis, and demonstrates its potential applicability to other cases of adaptive diversification of animal growth patterns.

KEY WORDS: Growth rates, life history, phylogenetic regression, primates.

The study of animal growth has been only partially integrated into the broad framework of life-history theory. A first hurdle to full integration has been the changing status of body size in life history studies. Whereas adult body size was considered a key determinant of life histories by those following the tradition of allometric analysis (Huxley 1932), more recently it has been interpreted as a confounder masking correlations among life-history variables (Harvey and Purvis 1999), or sometimes as a by-product of selection for age at first reproduction (Charnov 1993). A second reason is a relative lack of interest in the problem of interspecific variation in animal growth rates. This topic has received limited attention in comprehensive accounts of life history, and when

growth rates are addressed, interspecific variation is rarely analyzed from a comparative and phylogenetic perspective (Stearns 1992).

There is however concrete evidence of diversification in growth rates across animal groups (Grand 1992). Lizards and snakes are known to grow more slowly than other vertebrates (Shine and Charnov 1992), but primates are perhaps the best-documented example of differentiation in growth patterns. Primates live "life in the slow lane", with long juvenile periods, low fertility and mortality rates, late age at first reproduction, and longer life spans in comparison to other similarly sized mammals (Harvey and Clutton-Brock 1985; Godfrey et al. 2003; Jones

2011). Primates also show prolonged growth and reduced growth rates (Leigh 1996; Mumby and Vinicius 2008). In addition to bringing attention to the challenge of explaining slow growth in primates, Charnov (Charnov 1993; Charnov and Berrigan 1993) is one of the few authors to promote the use of growth rates in broad group comparisons. Charnov modeled postweaning growth in mammals through the "growth law":

$$\frac{dW}{dt} = AW^{0.75}, \qquad (1)$$

where W is body weight, t is age, dW/dt is body growth rate at age t, and A is a growth constant. The power of 0.75 reflects the general scaling of basal metabolic rate to body size across mammals (Farrell-Gray and Gotelli 2005), whereas the coefficient A may vary across species or higher taxa. Charnov estimated A in a large sample of mammals by regressing adult body weight on age at first reproduction across species, obtaining $A = 0.42$ for primates against $A = 1$ in other mammals.

Although the analysis indicated that primates as a group grow at lower rates than other mammals, a problem with Charnov's approach is that the growth coefficient A was calculated as the slope of interspecific regressions, rather than the coefficient of growth curves. Therefore it represents a general tendency of the sample but hides potential interspecific variation; in other words, it suggests that all primates grow slowly, and equally slowly. To investigate variation in growth rates at the species level, Mumby and Vinicius (2008) calculated A values directly from primate growth curves. We predicted and observed significant interspecific variation, with strepsirrhines derived from an early primate radiation (Perelman et al. 2011) at one extreme with growth rates closer to the mammalian average of $A = 1$, and humans ($A = 0.21$) and other apes at the opposite extreme of slow growth.

However, A is not a good estimator of growth rates because the "growth law" is not a satisfactory growth model. The fit between growth data and the growth curve

$$W(t)^{0.25} = 0.25At + W_0^{0.25}, \qquad (2)$$

which is obtained by integration of the growth law, is poor because animal growth curves (W against t, or $W^{0.25}$ against t) are typically sigmoidal rather than linear as predicted by equation (2). Fitting a regression line to nonlinear curves is especially sensitive to the choice of end point (i.e., the age at growth termination or adult body size), which was especially difficult to determine in the case of some strepsirrhines due to the small sample sizes (see Ravosa et al. 1993). Although the study revealed variation around the primate average of $A = 0.42$, correlation and regression analyses of A and life-history variables across species and the effect of phylogeny were difficult to interpret due to the problems with the parameter A (Mumby and Vinicius 2008), indicating that a better measure of growth rates is still needed.

A different type of "general" growth law was proposed by West et al. (West et al. 2001; Moses et al. 2008) who derived a "parameterless universal curve" for animal growth from first principles of metabolic energy allocation. According to the model, the energy budget of a growing organism is partitioned into cell metabolism (body maintenance) and a surplus available for cell division (body growth). Although the energy budget grows in proportion to $m^{3/4}$ (where m is body weight), maintenance scales with cell number and m; for this reason, the surplus channeled to growth, and therefore growth rate, must decrease with size. Animal growth can be modeled by the "universal growth curve"

$$r = 1 - e^{-\tau}, \qquad (3)$$

where $r = (m/M)^{0.25}$ is "dimensionless size," $\tau = (at/4M^{0.25}) - \ln(1 - (m_0/M)^{0.25})$ is "dimensionless age," m is weight at age t, m_0 is birth weight, M is adult weight, and a is a taxon-specific metabolic parameter. Equation (3) converts sigmoidal growth into an asymptotic curve of dimensionless mass r versus dimensionless time τ above, and fits growth data from shrimp to cattle. The model implies that, when we eliminate the effects of size and age scaling, growth seems to unfold at a similar pace in animal species across the tree of life.

If the general model is "universal" as claimed by West et al. (2001), the answer to why growth rates seem to vary across species in general, and why primates grow slowly in particular, may be simple. If primate growth data expressed as dimensionless size r and age τ can be fitted by the general model, then primate growth is not unique and its slowness is just another example of underlying metabolic scaling. However, Vinicius (2005) showed that primate growth curves deviate at various degrees from the general pattern, with New World monkeys almost matching the general pattern at one extreme, and humans at the opposite end of extreme growth retardation (strepsirrhines were not included in the analysis). Levels of deviation have not been quantified at the species level because as currently formulated the general growth model does not provide a measure of growth rate.

Here we present a modification of the general model by introducing the coefficient β, or dimensionless growth rate, and the new curve:

$$r = 1 - e^{-\beta\tau}. \qquad (4)$$

By fitting the modified curve to growth data, we can estimate the dimensionless growth rate β for any species and obtain a measure of relative deviation from the universal pattern (i.e., from β = 1). Growth acceleration is represented by β > 1, and growth retardation by β < 1. In the following we calculate β for a sample of 50 primate species as a case study. To exemplify the use of our scale-free measure of growth rate in comparative studies, we investigate associations between β and life-history variables

through phylogenetically controlled linear regression analysis. We show how β estimates can be used to test theories for the evolution of slow growth in primates based on learning (Ross 2004), energetic constraints (Foley Lee 1991; Navarrete et al. 2011), or reduction of ecological risk (Janson and van Schaik 1993). We also analyzed ontogenetic variation in growth rates from conception to adulthood in humans and rhesus monkeys.

Based on the results of the primate case study, we argue that our modified general growth model has broader implications for comparative growth studies in mammals in general. We show that the dimensionless growth parameter β can identify variation in growth rates at the species level, while controlling for size scaling and capturing phylogenetic signal. As such, it may represent an important step toward the integration of growth studies into mammalian life-history analysis.

Materials and Methods
GROWTH DATA
Data on body weight and age from birth for 50 primate species are from published growth curves (Eisenberg 1976; Froehlich et al. 1981; Hamada et al. 1986; Gavan 1991; Deputte 1992; Leigh 1992; Wickings and Dixson 1992; Ravosa et al. 1993; Leigh 1994a; Roberts 1994; Leigh and Shea 1995; Garber and Leigh 1997; Turner et al. 1997; Fragaszy and Adam-Curtis 1998; Leigh and Terranova 1998; Altmann and Alberts 2005; Schillaci and Stallmann 2005; Bernstein et al. 2007; Schillaci et al. 2007; Schaefer 2011). The human growth curve is from a Westernized USA population (Eveleth and Tanner 1992), but growth data from a traditional population (the Aeta hunter gatherers from the Philippines) produced virtually similar results (data not shown). Growth data from conception to birth in humans and rhesus monkey are from Brenner et al. (1976) and Kerr et al. (1969). Only data from females were used, with the exception of mixed-sex curves from some New World monkeys from Leigh and Terranova (1998) characterized by very low body size dimorphism. We used as few data sources as possible; 33 of 49 nonhuman primate curves were published by Steve Leigh's group, and 42 of 49 are from captive populations. Data from captive animals are important because growth studies in wild populations are rare, and Leigh (1994b) estimated that the correlation between captive and wild animal body size was very high. Data for cattle are from Lyne (1960) and West et al. (2001).

LIFE-HISTORY DATA
Adult body weight (kg) for the 50 species was estimated from their growth curves, that is, both growth rate and adult size were taken from the same population. Brain size (g), age at first reproduction, and maximum longevity data are from Kappeler and

Pereira (2003) supplemented by Lindefors (2002), Isler et al. (2008), Rasmussen (1988), MacLean et al (2009), de Magalhaes and Costa (2009), and Weigl (2005). Duration of juvenile phase was calculated as the interval between age at weaning and age at first reproduction. EQ (encephalization quotient) was calculated as $EQ = brain weight/0.12(adult weight)^{0.67}$ (Jerison 1973). The dataset used in our analyses is provided in Appendix S1.

ANALYSIS
The coefficient β was estimated by nonlinear least square fitting of the modified general model to growth data from the 50 species. Body weight and age were converted into dimensionless body mass ratio r and dimensionless age τ using the formulae above. We performed 12 phylogenetic regressions (PGLS) of β on body size, brain size, EQ, duration of juvenile phase, age at first reproduction, and longevity, using both raw and double-logged values (Nunn 2011). We divided the conventional significance level of $P = 0.05$ by the number of phylogenetic regressions (Bonferroni correction) to set the significance threshold at $P = 0.05/12 = 0.0042$. The primate phylogeny used in regressions is from Arnold et al. (2010).

To analyze changes in growth rate during ontogeny, we calculated age-specific β values from conception in humans and the rhesus monkey *Macaca mulatta*. Age-specific β values are the solutions to $r = 1 - e^{-\beta\tau}$ for each point in the curve, that is, for each dimensionless age $\tau = (at/4M^{0.25}) - \ln(1 - (m_0/M)^{0.25})$ and dimensionless size $r = (m/M)^{0.25}$, and therefore a plot of age-specific β by age represents variation in growth rate from conception to adulthood in the two species. All analyses were carried out in R (R Development Core Team, 2011). Phylogenetic regressions were performed using the R packages *ape* (Paradis et al. 2004) and *caper* (Orme et al. 2012).

Results
DISTRIBUTION OF β VALUES
Estimates of β in the 50 species vary from $\beta = 1.08$ in the greater galago *Otolemur garnettii* to $\beta = 0.21$ in *Homo sapiens* (Table 1). We observe β significantly larger than one only in the case of *O. garnettii* (95% confidence interval: 1.02–1.14); in two cases, the Senegal bushbaby *Galago senegalensis* (1.00–1.12) and the ruffed lemur *Varecia variegata* (0.80–1.09) the 95% confidence intervals include $\beta = 1$. In the remaining species, the dimensionless growth rate β is significantly below the mammalian average of $\beta = 1$ predicted by the general growth model. With the exception of the pigtailed macaque *M. nemestrina* ($R^2 = 0.31$), estimates of goodness of fit were high ($R^2 > 0.5$) showing that the modified general growth model describes growth trajectories satisfactorily (four examples are shown in Fig. S1, Appendix S2). Pagel's λ is

Table 1. Dimensionless growth rate β in 50 primate species (95% confidence interval in brackets).

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Strepsirrhines	
H. griseus	0.75 (0.64–0.87)
E. mongoz	0.65 (0.57–0.73)
E. macaco	0.87 (0.81–0.93)
E. rubriventer	0.86 (0.80–0.92)
V. variegate	0.94 (0.81–1.07)
E. fulvus	0.84 (0.75–0.92)
P. diadema	0.69 (0.59–0.79)
P. verreauxi	0.71 (0.56–0.86)
O. garnettii	1.08 (1.02–1.14)
G. senegalensis	1.06 (1.00–1.12)
Haplorhines	
T. bancanus	0.84 (0.80–0.87)
New World monkeys	
C. moloch	0.74 (0.65–0.83)
S. sciureus	0.73 (0.57–0.90)
C. pygmaea	0.63 (0.54–0.73)
C. jacchus	0.77 (0.68–0.85)
S. imperator	0.78 (0.64–0.91)
C. apella	0.59 (0.42–0.77)
C. goeldii	0.80 (0.74–0.86)
A. paliatta	0.63 (0.47–0.79)
A. caraya	0.63 (0.5–0.76)
A. geoffroyi	0.53 (0.38–0.68)
A. fusciceps	0.52 (0.40–0.64)
A. trivirgatus	0.79 (0.61–0.97)
Old World monkeys	
C. aethiops	0.57 (0.21–0.93)
E. patas	0.60 (0.38–0.81)
C. mitis	0.46 (0.27–0.66)
C. atys	0.52 (0.42–0.62)
M. mulatta	0.50 (0.40–0.60)
M. nemestrina	0.42 (0.33–0.51)
M. fuscata	0.36 (0.26–0.46)
M. silenus	0.65 (0.42–0.87)
M. arctoides	0.36 (0.24–0.48)
M. fascicularis	0.54 (0.43–0.65)
M. ochreata	0.43 (0.27–0.58)
P. cynocephalus	0.37 (0.22–0.52)
P. papio	0.39 (0.27–0.50)
P. anubis	0.58 (0.49–0.71)
L. albigena	0.37 (0.31–0.44)
M. sphinx	0.35 (0.28–0.43)
T. obscurus	0.48 (0.28–0.68)
T. cristatus	0.75 (0.55–0.95)
S. entellus	0.46 (0.36–0.56)
C. guereza	0.60 (0.41–0.79)
Apes	
S. syndactylus	0.37 (0.25–0.50)
H. lar	0.41 (0.35–0.48)
P. pygmaeus	0.23 (0.19–0.28)
G. gorilla	0.26 (0.20–0.31)
P. paniscus	0.27 (0.21–0.33)
P. troglodytes	0.28 (0.21–0.34)
H. sapiens	0.21 (0.15–0.27)
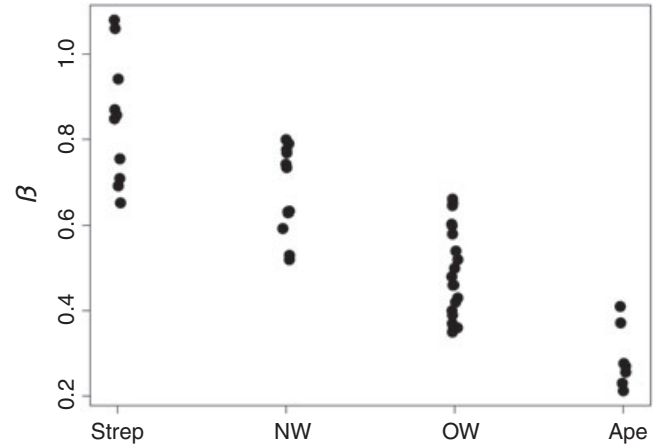
Figure 1. Values of growth rate β in 50 primate species. Circles represent distribution of β in 10 strepsirrhines (Strep), 12 New World monkeys (NW), 20 Old World monkeys (OW), and 7 apes (Ape). The tarsier *T. bancanus* (β = 0.83) is not shown.

high ($\lambda = 0.86$) suggesting that phylogenetic relatedness plays an important role in the distribution of β in our sample (Pagel 1999). The fastest growing primates are strepsirrhines, the western tarsier *Tarsius bancanus*, and small-bodied New World monkeys (Fig. 1). On average, β decreases across primates groups from the sampled strepsirrhines ($\beta = 0.85$, $N = 10$) and the single tarsier species ($\beta = 0.83$), to New World monkeys ($\beta = 0.68$, $N = 12$), Old World monkeys ($\beta = 0.49$, $N = 20$), and finally apes ($\beta = 0.29$, $N = 7$). The seven apes in our sample, including the large gorillas, exhibit the lower dimensionless growth rates ranging from $\beta = 0.21$ in humans $\beta = 0.41$ in the lar gibbon (*Hylobates lar*). Within each major group some patterns appear too: as a rule, galagos grow faster than lemurids, callitrichids faster than atelids, colobines faster than papionines, and gibbons faster than great apes. Due to the small number of age points from which curves were drawn, 95% confidence intervals of β estimates are broad and tend to overlap; for example, we cannot statistically distinguish between the ranges of β in apes and most Old World monkeys.

Phylogenetic regressions

Using double-logged values, regressions of β on weight, brain weight, encephalization (EQ), duration of juvenile period, age at first reproduction, and maximum longevity were all significant (using raw values, only regressions on brain size, juvenile phase, and age at first reproduction were significant, but R^2 values are lower). The six double-logged regressions have negative slopes: low growth rate is associated with large body size ($t = -7.06$, $P < 0.0001$, $R^2 = 0.65$), large brain size ($t = -7.68$, $P = 0.0001$, $R^2 = 0.69$), high encephalization ($t = -4.95$, $P < 0.0001$, $R^2 = 0.48$), long juvenile phase ($t = -6.53$, $P < 0.0001$, $R^2 = 0.63$), late age at first reproduction ($t = -6.69$, $P < 0.0001$, $R^2 = 0.64$), and long
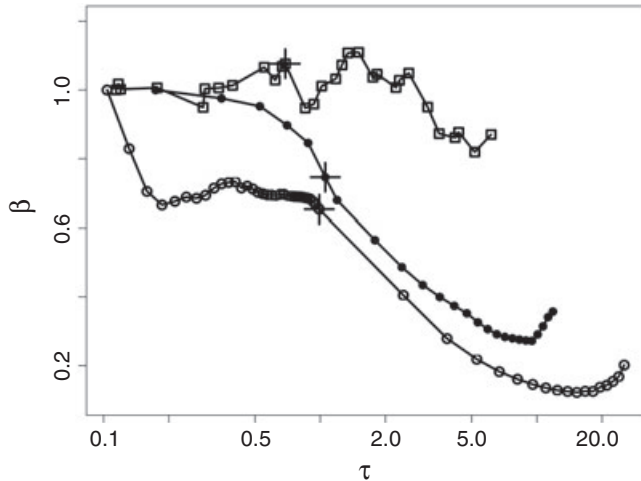
Figure 2. Variation in growth rate β by dimensionless age τ from conception in humans (open circles), rhesus monkeys (black circles), and cattle (squares). Crosses (+) indicate age at birth in the three species.

life span ($t = -3.10$, $P = 0.004$, $R^2 = 0.26$). Plots and regression parameters are shown in Figure S2 (Appendix S2).

Ontogenetic trajectories of β from conception

We also considered the possibility that growth rate measured by β may change during ontogeny (as suggested by Fig. S1, Appendix S2). In *H. sapiens* and *M. mulatta*, size at birth is higher than predicted by their average β value, whereas postnatal growth is slower (and flatter) than predicted. Figure 2 shows changes in β values from conception to adulthood and reveals that primate growth is only slow after birth. In the rhesus monkey, during early pregnancy β is as high as in cattle (a species included in the original study by West et al. 2001). However, whereas growth rates oscillate around $\beta = 1$ in cattle from conception to adulthood, in *M. mulatta* the growth coefficient decreases to values below $\beta = 0.7$ at birth and then to values lower than $\beta = 0.3$ during the juvenile period, that is, below the estimated average of $\beta = 0.5$. In humans, growth rates oscillate between $\beta = 0.6$ and $\beta = 0.8$ during pregnancy, and then decline to values below $\beta = 0.2$ after birth. In both species, a late acceleration in growth is observed and seems to correspond to the juvenile body growth spurt, a widespread feature in primates (Leigh 1996).

Discussion

We introduced the dimensionless growth rate β as a new tool with potentially broad applicability to comparative studies of animal growth and life history. The example of primates shows that the parameter β is able to reveal and quantify interspecific variation in growth dates, to estimate relative growth rates controlling for size scaling, to detect phylogenetic effects on the distribution of growth rates, and to identify associations between growth rates and other life-history variables.

The results have important implications for primate growth. The view that slow growth is a characteristic of primates as a group is rejected by our analyses. By estimating growth rates at the species level, we showed that some primates grow more slowly than others, and that some primates are not slow growing relative to the general growth pattern in West et al. (2001). Rather than a sharp contrast to other mammals as suggested by Charnov (1993), the parameter β describes a growth rate continuum that connects strepsirrhines and tarsiers to the general animal pattern at its fast extreme, and points to apes as the truly slow-growing primates. Furthermore, if one follows the suggestions that the fast–slow life-history continuum may involve more than one axis of variation (Stearns 1983; Gaillard et al. 1989; Bielby et al. 2007), a comparative measure of growth rate such as β may also help establishing the role of growth in the classification of mammalian life-history strategies.

The primate growth rate spectrum and its strong phylogenetic patterning provide insights into the origin of growth rate differentiation. Previous studies had shown that lemurids grow very rapidly and over a short period compared to other primates (Leigh and Terranova 1998). Lemurs differ from monkeys and apes in traits such as female dominance, female–female aggression, reduced sexual dimorphism, higher infant mortality, and seasonal breeding (Kappeler 1996; Wright 1999) and fail to show the association between brain size and social group size found in anthropoids (MacLean et al. 2009), a cornerstone of the social brain hypothesis (Dunbar and Schultz 2007). The comparatively high β values found in lemurs, and even higher in the two sampled galagos, provide further evidence for a grade difference between strepsirrhines and anthropoids. The western tarsier, despite being a haplorhine, shows a β value closer to the strepsirrhine average and higher than in any sampled ape, New World or Old World monkey. Some New World monkeys also show high β values that overlap with those from lemurs, and hence slow growth may have only evolved in the common ancestor of Old World monkeys and apes (Perelman et al. 2011). However, given the negative correlation between β and body size, another explanation for the higher average growth rates in strepsirrhines and New World monkeys may be the small body size of the sampled species (average of 2.3 kg in strepsirrhines and 2.8 kg in New World monkeys) compared to Old World monkeys (8.6 kg) and apes (43.6 kg). Further evidence for this explanation is that among strepsirrhines the small galagos show higher β than the larger lemurids, among New World monkeys the small callitrichids have higher β than the larger atelids, and among apes gibbons grow faster than the larger-bodied great apes. Among Old World monkeys the association between body size and β is less clear (the three *Papio* species are a good example).

Phylogenetically controlled regressions also identified associations between dimensionless growth rates and other life-history variables. In primates, species with low β (slow growth) exhibit not only large body but also large brain size, high encephalization, long juvenile phase, late age at first reproduction, and long life span. The association between growth rate and longevity confirmed previous studies (de Magalhaes et al. 2007) despite the fact that our measure of total life span was maximum observed longevity, a variable mostly obtained from zoo or captive animals and very sensitive to differences in sample size across species (Speakman 2005).

Primate growth data also exemplify how the use of β as a variable in phylogenetic regressions can provide tests of adaptive hypotheses for growth evolution. The negative correlation between growth rate and brain size is predicted by theories such as the "brain growth constraint" hypothesis (Martin 1981), which proposes that the cost of evolving and supporting large brains require either reducing energy expenditure on other energetically expensive organs (Aiello and Wheeler 1995; Isler and van Schaik 2009) or diverting energy from body growth, leading to low growth rates and growth prolongation in primates (Foley and Lee 1991; Navarrete et al. 2011). The brain constraint model predicts the negative association we found between β and encephalization. It also predicts the lowest growth rates to occur in large-brained humans, although the confidence interval of human β overlaps with values in other apes (with the exception of *H. lar*). A second hypothesis by Ross (2004) states that larger brains and higher cognition require longer learning periods and therefore prolonged growth phases and lower growth rates, and also predicts growth to be slowest in humans. The extension of juvenile phases and its association with low growth rates are predicted by the "ecological risk aversion" model (Janson and van Schaik 1993); this hypothesis explains primate slow growth as a social adaptation that evolved to reduce competition between juveniles and adults through a reduction in growth rates, body size, and metabolic costs in juveniles. The ecological risk aversion model hypothesis therefore predicts the observed negative correlations between slow growth and both long juvenile period and late age at first reproduction.

Other testable hypotheses are suggested by correlations between β and life-history variables. For example, if the ecological risk aversion hypothesis is correct, levels of competition between juvenile and adults should negatively correlate with β across species, and therefore increase from strepsirrhines to apes. In addition, the association between low β and large body and brain size, extended juvenile phase and late start of reproduction suggests that sociocognitive factors may be more important than ecology or diet to explain growth rate variation. Regressions of β against social (social group size, neocortex ratio, deception rates) and ecological variables (percentage of fruit in diet, home range) may provide a direct test for this prediction.

Finally, the analyses of β trajectories from conception in *H. sapiens* and *M. mulatta* indicate that slow body growth is an exclusively postnatal phenomenon in primates. During pregnancy and shortly after birth, β is higher than during the postnatal phase. Although the general growth model strictly applies to postnatal data (because energetic transfers from mothers affect the metabolic balance of the fetus), it remains to be explained why β is approximately the same before and after birth in cattle, but sharply decreases postnatally in the two primates. Our conclusions are limited by the fact that comparisons are based on only two primates and a single nonprimate, but a larger sample of growth curves from conception may help elucidating whether the postnatal decrease in growth rate is a primate phenomenon, and whether such reduction differs across primates. A related question is the pattern of age-related variation in β during late postnatal growth, or in the case of many primates, the preadult growth spurt (Leigh 1996). Our curves are based on female data, but growth curves from males, which undergo more intense growth spurts, may reveal pronounced changes late in ontogeny. For this reason the parameter β may be useful in future studies of primate growth dimorphism.

Conclusions

The modified general growth model has important implications for the origin and evolution of slow growth rates in primates, but we believe that this case study is only a first example of the broad applicability of the dimensionless approach to animal growth. The parameter β measures relative growth rates on a dimensionless scale that eliminates effects of body size scaling. The presence of phylogenetic signal in the distribution of β can be investigated via test statistics such as Pagel's λ, whereas phylogenetic regressions can test for association between growth rates and other life-history variables. The existence of such correlations indicate that variation in growth rates cannot be always explained as effects of metabolic scaling as proposed by West et al. (2001), but may rather reflect adaptive variation in life-history strategies. The dimensionless approach therefore integrates growth studies into the framework of life-history analysis, circumventing both the question over the status of body size and problems with existing estimators of growth rate such as the coefficient *A*.

The parameter β may be a useful tool for identifying other exceptions to the general growth model. If the links between growth rate and life history are found in other animal groups in addition to primates, it is expected that fast life histories should be associated with β > 1, whereas β < 1 may be found in large species such as the Asian and the African elephant. Given the relatively large number of cetacean species, characterized by large brains and body size, it might also be possible to identify a growth rate spectrum in dolphins, porpoises, and whales, but tests depend on the availability of data.

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Supporting Information

Additional Supporting Information may be found in the online version of this article at the publisher's website:

Appendix S1. Life-history dataset (upon acceptance, Appendix S1 is to be stored on Datadryad).

Appendix S2. Supplementary figures.

Figure S1. Growth data from four primate species fitted by the modified general growth curve.

Figure S2. Phylogenetic regressions (PGLS) of β on six life-history variables in 50 primate species.