RESEARCH ARTICLE

Primate Growth in the Slow Lane: A Study of Inter-Species Variation in the Growth Constant A

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Abstract Primates grow and develop slowly for mammalian standards. Charnov showed that primates grow at only about 40% of the rates observed in other mammals of similar size. However, previous estimates of growth rates in primates were derived from regressions of adult body weight on age at first reproduction in different species, and therefore represent only an average trend for primates. Based on Charnov's 'growth law', we estimated the growth constant A directly from published growth curves for 36 primate species from strepsirrhines to apes. We show that although primate growth is slow in all sampled species in comparison with the mammalian average, there is significant variation around the primate mean. Lemurids are particularly interesting due to their wide range of A values, and further study is required to determine whether environmental unpredictability could lead to the evolution of both very fast and very slow grow in different species. Results also indicate significant negative correlations between the growth constant A and both age at first reproduction and duration of the juvenile period, lending support to the juvenile risk hypothesis.

Keywords Primates · Growth · Life history · Lemurids · Juvenile risk hypothesis

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Introduction

Primates have been described as living 'life in the slow lane' (Stearns 1983; Harvey and Clutton-Brock 1985; Charnov and Berrigan 1993). In comparison with other mammals, primates exhibit slow growth rates, long juvenile periods, low fertility and mortality rates, large neonates and longer lifespans than predicted for their body size (Ross 1998; Walker et al. 2006). Particular attention has been dedicated to the long and slow growth process in primates. Life history theory postulates that age at maturation (when investment of metabolic energy is switched from growth to reproduction) in a given species is determined by a trade-off between growth and reproduction (Charnov 1993), i.e. as the compromise between the advantages of early sexual maturation and reproduction (that minimises the risk of death without reproduction; Johnson 2003, Kaplan et al. 2000) and the advantages of late growth termination (such as larger body size, higher social rank, decreased risk of predation among others; Stearns 1992). According to life history theory, primates would show late maturation relative to other mammals because of they are exposed to lower mortality rates and lower risk of death before reproduction, which favours prolonged investment in growth (Charnov and Berrigan 1993).

Although life history theory satisfactorily accounts for the long duration of growth in primates, an explanation for their low growth rates remains elusive. Charnov (1993) proposed that body growth from weaning to adulthood in mammalian species can be modelled by the following 'growth law':

$$\frac{dW}{dt} = A \times W^{0.75}$$

where W is body weight, t is age, dW/dt is the 'production rate' or body growth rate at time t, and A is a species-specific 'height of the production rate', or growth constant. The scaling coefficient of 0.75 is a rough measure of how much energy an organism can invest in growth as a function of its body size, and is similar to the scaling of basal metabolic rate to body size across mammals (West et al. 2001). Additionally, by regressing age at first reproduction on adult body weight across a large samples of species, average A was estimated in 0.42 in primates, in comparison to $A = 1$ in other mammals (Charnov 1993). The low value of A in primates is intriguing: given the advantages of large size mentioned above, why are primates not using their long period of growth to attain larger adult sizes? According to Charnov and Berrigan (1993), this is the fundamental question to be addressed by primate life history studies: in their words, 'why is A so low for primates? Answer that (says the theory) and many of features of primate life history will also be explained'.

Despite the interest it raises, very few studies have calculated the growth constant A in primate species directly from growth data. The average value of 0.42 was obtained from a regression of age at first reproduction on adult weight in different primate species, and therefore only represents a general or average trend potentially hiding variation among species and primate grades. Walker et al. (2006) indicated that there is a robust interspecific difference in the growth constant A between humans and the relatively faster-growing chimpanzees, but the methodology used in their study did not allow for a direct estimate of A in the two species. In humans, the constant A was calculated from growth data on the Ache from Paraguay by Hill and Hurtado (1996), who obtained a value of $A = 0.29$ for females and $A = 0.23$ for males. The results from the Ache suggest two main conclusions: first, studies based on growth data may reveal significant specific variation in the growth constant A relative to the primate average of 0.42; and second, humans may be outliers, with low growth rates even in comparison to the already depressed primate average.

For the reasons above, it may be highly informative to obtain A values directly from growth curves and from a broader range of primate species. Knowing A values for various primate species might potentially help answering questions such as whether all primates share similar levels of slowness in life history relative to other mammals, and whether the distribution of growth rates exhibited across primate groups can be explained by phylogenetic or adaptive factors. For example, one might expect strepsirrhines to show A values closer to the mammalian average of 1, since they represent an earlier primate radiation linking anthropoid primates to other mammals

(Purvis 1995). One could also predict differences in the growth constant A as a function of diet: Leigh (1994a, b) for example found that folivorous anthropoids tend to exhibit lower growth rates than frugivorous ones, but he did not present a comparison between effects of diet and effects of life history factors such as duration of juvenile period and age at maturity.

Strong correlations between A and brain size would also support explanations based upon learning or brain growth and maintenance. The 'needing to learn' models discussed by Ross (2004) for example postulate that larger brains, slow growth rates and a prolonged juvenile learning period evolved together in species that need to process complex social and environmental information. On the other hand, 'brain growth constraint' models such as proposed by Foley and Lee (1991) also imply a negative correlation between brain size and body growth rates, this time due to energetic constraints: large brains are expensive organs to grow and maintain, which necessarily reduces rates of other metabolic processes such as body growth. If this is the case, highly encephalised modern humans should be outliers among primates as the ultimate slow developers. Finally, variation in the growth constant A may be correlated with life history variables: for example, correlations between the growth constant A and age at first reproduction, maximum longevity or duration of the juvenile period would support explanations of slowness based upon juvenile risk avoidance. According to the 'juvenile risk' model (Janson and van Schaik 1993), primates evolved low growth rates as an adaptive response that reduces energetic needs during the growth period. As a consequence, they can channel energy for mortality-reducing, longevity-extending activities such as predator avoidance. In the following, we discuss those hypotheses in the light of estimates of the growth constant A from 36 primate species.

Methods and Data

Growth Data

Weight (W) and age (T) growth data were obtained from published body weight or velocity curves in 36 primate species representing strepsirrhines ($N = 9$), New World monkeys ($N = 8$), Old World monkeys ($N = 12$) and apes ($N = 7$). In order to reduce experimental noise due to differences in samples, methodology and measurement techniques, our data were restricted to the fewest possible sources necessary to maintain a broad sample of species (Leigh 1994a, 1996; Leigh and Shea 1996; Leigh and Terranova 1998; Ravosa et al. 1993). Velocity curves,

when used, were converted into growth curves by cumulatively adding yearly body weight increments to neonatal weight. Human growth data are from Eveleth and Tanner (1992).

Our data sources represent measurements taken from captive animals with the exception of Ravosa et al. (1993), whose sample also included trapped wild animals and was the only available source of growth curves for sifakas. Although data from captive animals are in principle not ideal as they reflect growth taking place under artificial conditions, they are extremely useful in evolutionary studies (Leigh and Shea 1996): data from wild animals are extremely rare, and Leigh (1994b) has shown that correlations between body weight in captive and wild animals are very high. When published growth curves were separated by sex, only the female curve was used. Our Supplementary File presents all growth curve data used in our analyses.

Size and Life History Data

Data on brain size, duration of juvenile period and age at first reproduction in females are from Kappeler and Pereira (2003) and refer to captive animals. Maximum longevity data are from the *AnAge* (the 'Animal Ageing and Longevity Database') website (http://genomics.senescence.info/species/) and were almost exclusively obtained from captive animals. Adult female body size was estimated as the point of growth termination in our curves.

Calculation of A

Integration of the growth law (Charnov 1993) leads to the equation

$$W(T)^{0.25} = 0.25AT + W_0^{0.25}$$

where W_0 is weaning size, T is age, and age zero is taken not at birth but at weaning (the point of independence from the parent). We only used growth data between weaning and adulthood, as in Charnov (1993). Since integration linearises the growth law, regression analysis can be applied to our obtained growth data points. Since the weaning size W_0 is known for each species, we forced the regression through the intercept $W_0^{0.25}$ and calculated the regression slope $0.25A$ to obtain the growth constant A in all 36 sampled primate species.

Results

Distribution of A Values

All estimates of the growth constant A were statistically significant. The growth constant A varies from 0.19 in the cercopithecoid *C. mitis* to 0.61 in the lemurid *E. flavifrons* (Table 1). Therefore, all species exhibit A values lower than 1, and are therefore below the average for non-primate mammals. The results also mean that humans ($A = 0.26$) are not the slowest growing primate in the sample. Figure 1 shows the distribution of A values by primate group (strepsirrhines, New World monkeys, Old World monkeys, and apes). We obtained a mean value of $A = 0.35$, close to Charnov's estimate of 0.42. When data are separated by major grade subdivision, we obtain an average of $A = 0.39$ in strepsirrhines, $A = 0.42$ in New World monkeys, $A = 0.32$ in Old World monkeys, and $A = 0.29$ in apes. Thus, all four major primate subdivisions have mean A values well below the mammalian average. Strepsirrhines are closer to the remaining primates than to the mammalian average of $A = 1$, but this must be seen with caution since the only species in our sample were lemurids. *Propithecus*

Table 1 Growth constant A calculated for 36 primate species

| Strepsirhines		New world monkeys		Old world monkeys		Apes	
Species	A	Species	A	Species	A	Species	A
Hapalemur griseus	0.39	*Callicebus moloch*	0.44	*Cercopithecus mitis*	0.19	*Hylobates lar*	0.21
Eulemur mongoz	0.31	*Saimiri sciureus*	0.34	*Cercopithecus neglectus*	0.26	*Hylobates syndactylus*	0.27
Eulemur macaco	0.51	*Cebuella pygmaea*	0.45	*Cercopithecus aethiops*	0.40	*Pongo pygmaeus*	0.27
Eulemur flavifrons	0.61	*Callithrix jacchus*	0.43	*Erythrocebus patas*	0.40	*Gorilla gorilla*	0.39
Eulemur rubriventer	0.47	*Saguinus imperator*	0.46	*Macaca mulatta*	0.35	*Pan paniscus*	0.33
Varecia variegata	0.43	*Saguinus fuscicollis*	0.37	*Macaca nemestrina*	0.33	*Pan troglodytes*	0.28
Eulemur sanfordi	0.34	*Cebus apella*	0.27	*Macaca fuscata*	0.34	*Homo sapiens*	0.26
Propithecus diadema	0.23	*Callimico goeldii*	0.57	*Macaca silenus*	0.30		
Propithecus verreauxi	0.23			*Macaca fascicularis*	0.36		
				Papio papio	0.26		
				Presbytis entellus	0.37		
				Colobus guereza	0.27		
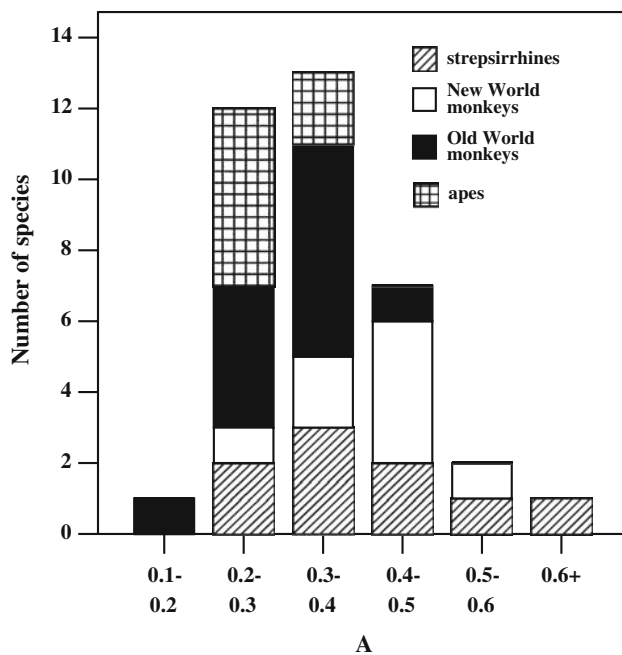
Fig. 1 Distribution of values of the growth constant A by primate grade

diadema ($A = 0.23$) and *Propithecus verreauxi* ($A = 0.23$) for example exhibit very slow growth and have a large effect on the average A of strepsirrhines (when the two species are excluded, mean value is 0.44). Among apes, gorillas grow faster than average ($A = 0.39$), and surprisingly the slowest growing species is the gibbon *Hylobates lar* ($A = 0.21$) instead of *Homo sapiens* ($A = 0.26$).

Relation of a to Body Size, Brain Size and Life History Variables

Linear regression analysis was also used to estimate bivariate (Pearson) correlations between the growth constant A and a set of size and life history variables. The relationship between A and adult body weight across all primate species is not statistically significant ($P = 0.167$, $N = 36$). To minimise phylogenetic or sampling effects (Purvis et al. 2003), all analyses were also repeated with species separated into strepsirrhines, New World monkeys, Old World monkeys and apes, but none of the four regressions of A on body size was significant (at the $P < 0.05$ level). Linear regression is however unable to capture the existence of non-linear relationships between variables, such as allometric relationships frequently observed between quantitative variables (Martin et al. 2005). In order to test for allometric relationships, we linearised our data by calculating logarithmic values and then performed linear regression (Lande 1985). We did obtain a significant negative correlation between log(A)

and log(adult body weight) across all sampled primates ($r = -0.47$, $P = 0.004$), but no relation was observed separately for strepsirrhines, New World monkeys, Old World monkeys and apes.

We also tested for possible correlations between A and brain weight. Some authors have linked large brains to slow growth (Foley and Lee 1991; Ross 2004), and predicted a negative correlation between brain weight and A. Our regressions show that, for the total primate sample ($N = 32$; brain data were missing for four species), the correlation between A and brain weight is not significant ($P = 0.14$). Separate regressions for strepsirrhines, New World monkeys, Old World monkeys or apes are not significant either (at the $P < 0.05$ level). Using log-transformed data, we found a significant relationship between log(A) and log(brain weight) across the primate sample ($r = -0.483$, $P = 0.005$), but once again no relation was observed separately for strepsirrhines, New World monkeys, Old World monkeys and apes. We also tested the relationship between A and encephalisation or EQ, calculated as EQ = brain weight/0.17(body weight)$^{0.72}$ (Marino 1998), and found no significant correlation across primates either using our original data ($P = 0.078$, $N = 32$) or log-transformed data ($P = 0.11$).

Since primates are characterised not only by low growth rates but also by extended juvenile period and late maturation (Charnov 1993; Janson and van Schaik 1993; Leigh 2001), we tested whether the growth constant A negatively correlates with duration of the juvenile period, age at first reproduction and longevity. First, regression of A on duration of juvenile period across primates is significant both when raw data ($r = -0.58$, $P < 0.01$) and log-transformed values ($r = -0.70$, $P < 0.001$, $N = 21$) are used (Figs. 2, 3). Regressions for separate groups are not significant at the $P < 0.05$ level (raw or log-transformed data), possibly due to the exceptionally small number of species for which data of duration of juvenile period is available ($N = 21$; 4 strepsirrhines, 4 New World monkeys, 8 Old World monkeys, 5 apes).

Second, the regression of A on age at first reproduction (Fig. 4) including all species is highly significant ($r = -0.56$, $P < 0.001$, $N = 32$). Importantly, the separate regressions for strepsirrhines ($r = -0.87$, $P < 0.01$), New World monkeys ($r = -0.78$, $P < 0.05$) and Old World monkeys ($r = -0.66$, $P < 0.05$) were all negative and statistically significant. Only the regression for apes is not significant ($P = 0.75$). Log-transformed data produce stronger negative correlations for the total primate sample ($r = -0.68$, $P < 0.001$; see Fig. 5), strepsirrhines ($r = -0.92$, $P = 0.003$), New Wold monkeys ($r = -0.86$, $P = 0.014$) and Old World monkeys ($r = -0.65$, $P = 0.02$) but not for apes ($P = 0.85$).
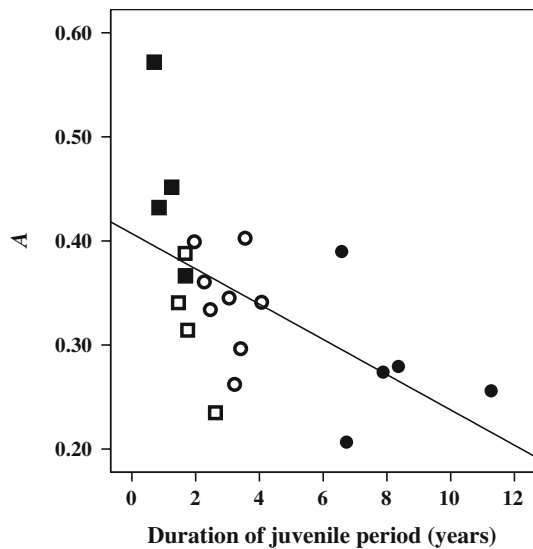
Fig. 2 Regression of *A* values against duration of juvenile period in primate species. Symbols: strepsirrhines (□), New World monkeys (■), Old World monkeys (○) and apes (●)
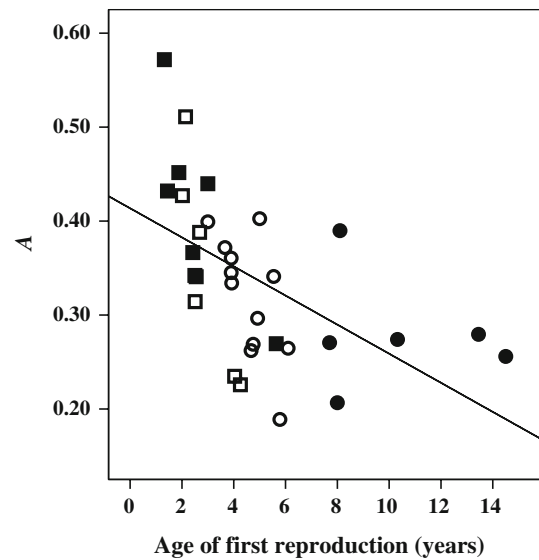

Fig. 4 Regression of *A* values against age at first reproduction in primate species (symbols as in Fig. 2)
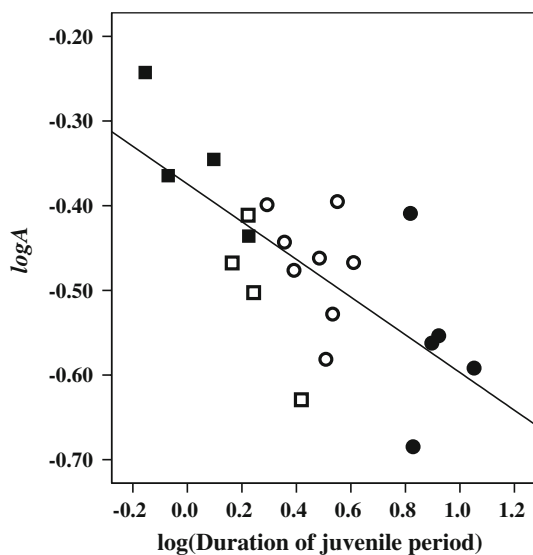

Fig. 3 Regression of *A* values against duration of juvenile period in primate species, logarithmic scale (symbols as in Fig. 2)
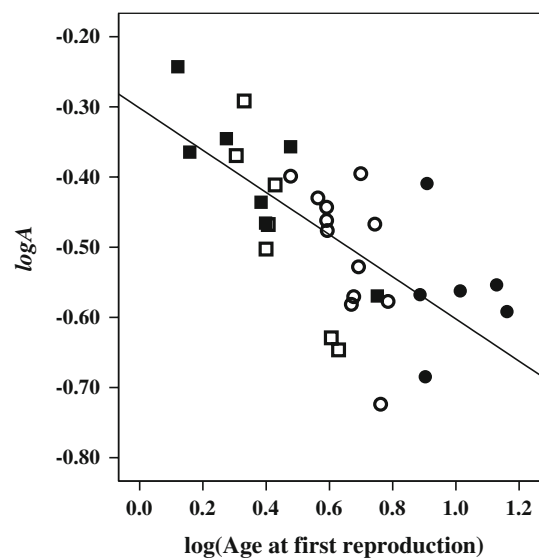

Fig. 5 Regression of *A* values against age at first reproduction in primate species, logarithmic values (symbols as in Fig. 2)

Finally, the regression of *A* on maximum longevity including all species is statistically significant ($r = -0.45$, $P < 0.01$, $N = 32$), but only the separate regression for apes is ($r = -0.79$, $P = 0.03$). Log-transformed data produce the same pattern, with a significant regression for the total primate sample ($r = -0.55$, $P = 0.001$) and for apes ($r = -0.88$, $P < 0.01$).

Partial Correlation Analysis

The analyses above revealed correlations between the growth constant *A* and a number of life history variables

(duration of juvenile period and age at first reproduction in special, and also maximum longevity). However, correlations were also found between *A* and both body size and brain size, suggesting that variation in *A* might be at least partially a size scaling effect; besides, some of the variables we used may be correlated among themselves. In order to assess the independent contribution of our variables to the distribution of *A* values we calculated partial correlations, which control for the confounding effects of underlying variables.

We first tested whether *A* correlates with life history landmarks (duration of juvenile period, age at first

reproduction, maximum longevity) after controlling for body and brain size. The partial correlation between A and duration of juvenile period is still significant and negative after controlling for body weight (partial $r = -0.685$, $P = 0.001$); the same is true for A and age at first reproduction (partial $r = -0.67$, $P < 0.001$), and A and maximum longevity (partial $r = -0.45$, $P = 0.001$). Similar results were obtained using log-transformed data for duration of juvenile period (partial $r = -0.79$, $P < 0.001$), age at first reproduction (partial $r = -0.79$, $P < 0.001$) and maximum longevity (partial $r = -0.62$, $P < 0.001$). Thus, life history variables and A correlate independently from any underlying effects of body size.

Controlling for brain weight, partial correlations were also significant and negative between A and duration of juvenile period (partial $r = -0.61$, $P = 0.004$), age at first reproduction (partial $r = -0.63$, $P < 0.001$) and maximum longevity (partial $r = -0.53$, $P = 0.004$). Partial correlations using logarithmic values were also significant between A and duration of juvenile period (partial $r = -0.69$, $P = 0.001$), age at first reproduction (partial $r = -0.73$, $P < 0.001$) and maximum longevity (partial $r = -0.57$, $P < 0.001$). This shows that life history variables and A correlate independently from any effects of brain size. Controlling simultaneously for brain and body size still also results in significant partial correlations between A and duration of the juvenile period ($r = -0.69$, $P < 0.001$), age at first reproduction ($r = -0.68$, $P < 0.001$) and maximum longevity ($r = -0.53$, $P < 0.005$). The same is true for log-transformed data (not shown).

We inversely tested whether the Pearson correlations of A with body size and brain size (which were weaker, limited to log-transformed data and only observed for the total primate sample) identified above would persist after controlling for life history variables. The partial correlation between A and body size is significant after simultaneously controlling for the three life history variables (partial $r = 0.58$, $P < 0.02$), or individually for duration of juvenile growth period ($r = 0.50$, $P < 0.02$) and age at first reproduction ($r = 0.48$, $P = 0.006$), but not maximum longevity; log-transformed data did not render any significant partial correlations (data not shown). Finally, partial correlations between A and brain weight are significant after simultaneously controlling for the three life history variables ($r = 0.58$, $P = 0.02$), and individually for age at first reproduction ($r = 0.427$, $P = 0.019$) and maximum longevity ($r = 0.428$, $P = 0.023$). Log-transformed data did not produce any significant partial correlations (data not shown). Thus, partial correlations between A and brain and body size are sometimes observed after controlling for life history, but their sign is positive (in contrast to the originally negative bivariate correlations).

Discussion

The strongest pattern we observed between the growth constant A and primate life history variables is that species with low values of A exhibit late age at first reproduction, although tests also revealed negative correlations between A and both duration of juvenile period and maximum longevity. We also identified (weaker) negative correlations between A and both body size and brain size using log-transformed data; however, when the effect of life history variables is controlled for, positive partial correlations were observed. In the following we discuss our results in the light of current theories of primate growth and life history.

Body Size: Effects of Ecology and Life History

Our results showed a weak negative bivariate correlation between A and body size (only when log-transformed data are used), but a positive partial correlation after controlling for the effects of life history. According to Charnov's (1993) mammalian life history model, mortality rates faced by species determine how fast development towards adulthood is: high mortality favours early sexual maturation and first reproduction (so as to minimise the risk of death before reproduction), leading to earlier termination of growth, faster growth rates (as a response to shorter time available for growth), and smaller body size (the 'mouse end' of the mammalian spectrum). Low mortality rates on the other hand would favour late maturation, longer growth, slower growth rates, and larger body size (the 'elephant end' of the mammalian spectrum). That logic would account for the negative correlations between A and body size we identified. But as seen, when the dominating effect of life history variables is controlled for, the correlation between A and body size is inverted and becomes positive. This might be explained by the operation of other ecological factors on primate growth in addition to life history. For example, Leigh (1994a) argued that folivorous anthropoid primates tend to exhibit higher growth rates than frugivorous primates, and in some cases (namely in species weighing less than one kilogram, and also gorillas and siamangs) such higher growth rates are associated with larger adult size. However, the relationship between size and diet observed by Leigh (1994a) was not universal: although larger folivorous anthropoids grow faster, they also tend to terminate growth earlier.

Brain Growth Models

A similar pattern was observed between A and brain size, namely a negative bivariate correlation but a positive

partial correlation after controlling for life history variables. Various studies had predicted links between brain size and growth rates in primates. The 'brain growth constraint' hypothesis (Foley and Lee 1991; Ross 2004) for example is based on the energetic costliness of large primate brains and the burden that it would impose on other metabolic functions such as body growth. Primates would be able to support their large brains by decreasing energy expenditure on other energetically expensive organs such as the gut (Aiello and Wheeler 1995), or by diverting energy from body growth, leading to both low growth rates and growth prolongation due to the limited energy budget available. Thus, the 'brain growth constraint model' seems to imply the links between large brains, slow body growth, longer juvenility and late age at first reproduction revealed by our analyses.

On the other hand, the positive partial correlation between brain size and the growth coefficient A after controlling for duration of juvenility and late maturation, although weak, cannot be easily explained by ecological factors such as diet. For example, folivorous primates not only growth relatively faster as pointed out by Leigh (1994a) but also show smaller brain sizes than frugivorous primates (Clutton-Brock and Harvey 1980); this would however still imply a negative correlation between growth rate and brain size. The explanation for the positive partial correlations between A and brain weight might depend on other aspects of dietary variation, or by factors other than diet.

Finally, the brain growth constraint model predicts that humans would display very low A values because of their very large brains, but surprisingly, humans do not show the lowest A value in our study sample. We found species with relatively lower brain size and encephalisation compared to humans such as the strepsirrhine *Propithecus diadema* but showing even lower A values.

Juvenile Risk Model

The juvenile risk model (Janson and Van Schaik 1993) postulates that slow primate growth is adaptive and the product of primate sociality. A basic assumption of the model is that living in large groups, as commonly observed in primates, is favoured when predation risks are high. However, life in large groups poses particular problems for juveniles, which must spend more time foraging than adults due to their smaller size and less developed foraging skills. The smaller body size of juveniles also makes them more vulnerable to predation than adults, and forces them to forage in the centre of the group, where feeding competition is the most severe. Juveniles are also more susceptible to food shortages and more likely to starve than adults (Ross 1998).

According to the juvenile risk hypothesis, low growth rates in juvenile primates evolved as the solution to the problems above. The reason is that lower growth rates would imply lower metabolic cost, as juveniles would maintain small body size for longer. The juvenile risk model still allows growth to a large adult body size, due to late maturation and the evolution of a growth spurt. In this way, the benefits of large body size would be maintained, but the growth schedule in a given species would be determined by the prevailing levels of juvenile risk. In summary, the juvenile risk model predicts that low A values are linked to late age at sexual maturation and first reproduction, and prolonged juvenile period. The correlation we found between low A and late age at first reproduction provide strong support to the model, but further evidence and larger sample sizes are required to determine whether the extended juvenile period found in primates itself is associated with low A.

The Puzzle of Lemurs

We predicted that lemurs would exhibit the highest A values, since previous studies had shown that as a rule lemurids grow very rapidly and over a short period relative to anthropoids (Leigh and Terranova 1998). However, our results showed that they did not form a group with A values intermediate between anthropoid primates (average $A = 0.42$) and other mammals (average $A = 1$), but instead fall within the range of the anthropoid primates. The reason seems to be that lemurids show great variation in A values, ranging from slow growing species such as *Propithecus diadema* ($A = 0.23$) and *Propithecus verreauxi* ($A = 0.23$) to fast growing species such as *Eulemur macaco* ($A = 0.51$) and *Eulemur flavifrons* ($A = 0.61$). It is known that lemurs (and strepsirrhines in general) contrast with anthropoids in several behavioural features, including female dominance, targeted female–female aggression, lack of sexual dimorphism regardless of mating system, higher infant mortality, and strict seasonal breeding (Kappeler 1996; Wright 1999), and it is possible that the large variation in A is linked with these characteristics.

For example, seasonal variation in resource availability, seasonal reproduction and high infant mortality have been used to explain both rapid and slow growth in different lemurs. On the one hand, Leigh and Terranova (1998) pointed to the high rates of infant mortality in lemurid species such as *E. macaco*, showing that around 52% of infants die in the first year in comparison to around 25% in anthropoid primates. For this reason, they suggest that infants from this species must grow rapidly in order to reach adult size and foraging capacity. In addition, because reproduction is seasonal in lemurs, it could be a great loss to overall reproductive output if a breeding season is

missed, which also selects against prolonged growth. On the other hand, studies focusing on slow growing lemurs such as *Propithecus diadema* (diademed sifaka) revealed late age at first reproduction; in fact, sifakas were shown to be the only studied folivorous primates to begin reproduction later than expected for their body size (Pochron et al. 2004). Richard et al. (2002) also showed that in *Propithecus verreauxi* fewer than half of females gave birth for the first time by the age of six, and the median age of first reproduction was 16 years. Fertility rates in females remained stable after the age of 7 rather than quickly decaying, with females aged 20 and 21 years reproducing at the same rate as those aged seven. Fertility rates only fell after 21 years, and one female did give birth at 28 years indicating a very long reproductive span.

Richard et al. (2002) termed sifakas 'snails among tortoises' because of their slow life histories even for primate standards. They argued that the habitat in which sifakas live is highly variable, with a high chance of experiencing drought per decade, and explained the sifaka strategy in terms of 'bet hedging', or risk spreading over seasons under the circumstance of high environmental unpredictability. Stearns (1992) argued that if there is seasonal variation in the probability that offspring will survive to breed, it is advantageous to spread the reproductive effort into multiple seasons, sampling a larger number of ecological conditions and therefore increasing the number of offspring born in at least one 'good' season. The lower adult mortality rates, stable in females until the age of 21, would allow them to adopt a 'bet hedging' strategy. To sum up, lemurid species representing the most primitive primate grade and showing lower encephalisation than anthropoids occupy both extremes of fast and slow development among primates, possibly as a direct adaptive response to environmental conditions.

Conclusions

The inspiration for this study was Charnov's (1993) statement that primates have a low height of production rate or growth constant A, defining a 'keystone difference' between primates and other mammals. The average A values calculated here directly from growth curves for 36 primate species are close to the value estimated by Charnov (1993) from data on age at sexual maturity and adult body weight. A values appear to follow a decreasing pattern from strepsirrhines and New World monkeys, through Old World monkeys and finally apes, although there are several notable outliers in each of the four major groups. The fact that lemurs are not intermediate between anthropoids and other mammals suggests that slow growth is a primitive

characteristic of primates, rather than a consequence of the relatively larger brains of the more derived anthropoids. Our results also showed that the highly encephalised *Homo sapiens* is not the only species growing exceptionally slowly even for primate standards.

Overall, results indicate some broad trends in A across primates, the strongest being the trend of decreasing A with later age at first reproduction, and by extension, later age of sexual maturity. The observed correlations lend support the juvenile risk model, in which juvenile growth is slow because of high risk of predation and high feeding competition in large groups for patchily distributed resources. However, the results show that some primates in the sample exhibit low A values although not living in large groups, such as the gibbon *H. lar*. Data on further species would be desirable and would help to confirm the existence of links between low A, extended juvenility, late age at first reproduction and high maximum longevity.

Finally, it must be emphasised that although our analyses are based on data representing the four major primate grades (which partially minimises phylogenetic biases) we did not employ any formal method of correction for phylogenetic effects (Purvis et al. 2003). Future studies based on the same dataset (or an ideally a larger one) should test whether procedures such as the analyses of independent contrasts would lead to different results.

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