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MPIDR WORKING PAPER WP 2010-031
NOVEMBER 2010

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This working paper has been approved for release by: Alexander Scheuerlein (scheuerlein@demogr.mpg.de), Deputy Head of the Laboratory of Evolutionary Biodemography.

Human longevity and post-fertile survival are not predicted by primate allometric patterns

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ABSTRACT:

The tendency of women to outlive their own fertility has been explained allometrically, with age at reproductive cessation attributed to ovarian follicle depletion in allometrically appropriate ovaries, and longevity related to brain and body scaling. However, because women's age at reproductive cessation is extraordinarily early compared to their longevity, we question whether both of these aspects of our demography can be predicted from primate allometric patterns. We employ a measure of longevity more useful for interspecies comparisons than the traditionally used maximum longevity to examine these allometric patterns. Using information-criterion based model selection, we find that brain size alone, rather than body size or their combined effects, produces preferred predictive models of longevity and of age at reproductive cessation. These models predict human longevity of 54-60 years, well below observed values, but accurately predict women's age at reproductive cessation. Rejecting previous conclusions, we find that human longevity, and; therefore, human post-fertile survival, are not predicted by primate patterns. We suggest that women's allometrically inappropriate longevity, and post-fertile survival, cannot be sufficiently explained in terms of proximate and phylogenetic constraints, and must be explained in terms of the unusual selective costs and benefits experienced by older women.

Key Words: Life-history scaling, Post-reproductive lifespan, demographic evolution

Women are unique among primates in regularly experiencing significant post-fertile survival under ecologically relevant conditions, and many hypotheses have been proposed to explain the evolution of this trait. The Grandmother Hypothesis ((Hawkes 2004; Hawkes et al. 1989)), proposes that women's post-fertile survival evolved through kin selection effects associated with grand-maternal care. Women above a certain age are thought to gain more selective advantage by investing in the care and provisioning of grand-offspring than by continuing the risk and expense of having more babies of their own. The Mother Hypothesis ((Peccei 2001; Williams 1957)) parallels the Grandmother Hypothesis, but relies upon the value of maternal rather than grand-maternal care. The extended dependency of human young requires mothers to live well past the age at which they have their last child, lest that child, and their investment in it, fail. A recent hypothesis proposed by Cant and Johnstone ((2008)) focuses on the selective benefits to temporal separation of breeding generations in terms of reduced competition within groups. All three of these hypotheses involve transfers of resources from older women to their kin, a phenomenon that life-history optimization studies suggest selects for post-fertile survival (Chu and Lee 2006; Lee 2003; Lee 2008) Alternatively, the Patriarch Hypothesis ((Marlowe 2000; Tuljapurkar et al. 2007)) attributes human longevity to the reproductive success of older males and the correlation between male and female longevity.

On a proximate level, several authors have studied the allometric scaling of brain, body and longevity in primates and mammals (e.g Austad and Fischer 1992; Barton 1999; Sacher 1959), and some ((Allman et al. 1993; Hakeem et al. 1996)) have suggested that given the size of our brains and bodies, human longevity is predictable. Body mass has long been known to correlate with longevity ((Lindstedt and Calder 1981)). Brain mass is potentially more closely related to longevity ((Allman et al. 1993; Sacher 1959)), because it both scales allometrically with

body mass and because brain tissue is more energetically expensive to build and maintain than similar masses of most other tissues, requiring extended investment and necessitating extended payoff ((Kaplan and Robson 2002)). Larger brains may also require a slower demography because of the time needed to acquire skills and knowledge ((Kaplan et al. 2007)). Primates are unusually longevous for our size, which has been attributed to our large brains ((Austad and Fischer 1992)).

Comparative studies of longevity have consistently measured a species' longevity as the longest recorded lifespan of any member of that species (maximum longevity). This measure makes comparisons to humans difficult. Maximum observed longevity, like any maximum drawn from a tailed distribution, increases with sample size. Maximum longevity of a species is likely also increased by investment in species-specific life-extending infrastructure and knowledge. The size of our sample of human lifespans exceeds availability of similar data on all other primates by many orders of magnitude (although exact sample sizes are often unknown), and economic investment in life-extending technology in humans is incomparably greater than our investment in extending life in any other species. The maximum verified human longevity (122 years) is therefore not comparable to, or predicted by ((Judge and Carey 2000)), the maximum longevities of other primates. For this reason, maximum longevities in captive primates have been compared not to the maximum longevity of humans, but to non-maximum advanced ages (e.g., the age to which 25% of hunter-gatherer women survive). Judge and Carey (2000) used maximum primate longevities to generate predictions of human longevity ranging from 51 to 87 years. This finding, in combination with the view that age at reproductive cessation is also allometrically determined, was the basis for their claim that human's extended post-fertile survival may be "predicted by primate patterns."

Women's age at reproductive cessation is thought to be explicable allometrically because larger primates have larger stores of oocytes, which require longer to deplete. Women as a population reach menopause at approximately the age predicted based on their oocyte stores, which is in turn roughly predictable based on their mean body mass ((Gosden and Telfer 1987a)). However, this allometric argument is potentially troubling; if both age at reproductive cessation and lifespan scale with the size of the organism, they should clearly also scale with each other (Figure 1). Across most primates they do, but human females with long post-reproductive lifespans do not conform to this pattern. Economic development tends to extend human longevity, while decreasing mean age at last reproduction, suggesting the possibility that pre-agricultural humans may have lacked meaningful post-fertile survival. As mean age at reproductive cessation in human natural fertility populations (those whose members do not make fertility decisions based on parity) is fairly constant (at about 41, (Bongaarts 1982)) populations of women without extended post-fertile survival would have necessarily lived much shorter lives than women in any current population, a notion given credence by some paleodemographic data ((Lovejoy et al. 1977)). Several authors have examined and rejected this possibility (e.g., (Bocquet-Appel and Masset 1982; Hawkes and Jones 2005)), implying that the divergence of human longevity from human age at reproductive cessation is a biologically important exception to allometric scaling and not a purely cultural artifact.

Allometric and selective explanations for women's post-fertile survival need not be considered as either alternative or disconnected. Embodied capital theory ((Kaplan et al. 2007; Kaplan et al. 2000a)), a synthesis of economic methods with life-history theory, explains allometric patterns in terms of cost-benefit relationships in which the optimal investments in different structures and goals change predictably with scale, while the form of the underlying trade-offs is maintained. The size of the organism and its brain are expected to scale with

longevity according to predictable patterns, so long as the system of costs and benefits relating reproduction and longevity to size is maintained. In this context the failure of human reproductive lifespan to scale with human longevity along primate patterns could be explained if the selective forces affecting one or both were structurally different than in other primates. Potentially critical difference in this regard include humans' selectively important grand-maternal care (Lahdenpera et al. 2004; Sear 2000; Voland 2002), which has not been found in other primates (Packer et al. 1998; Pavelka et al. 2002), and the extraordinary length of the human period of juvenile dependence relative to reproductive lifespan (Kaplan et al. 2000b). In combination with these unique features of human demography, effects described by the selective hypotheses mentioned above may alter those cost-benefit structures which cause the scaling of reproductive lifespan to longevity in primates, thereby explaining human deviations from allometric norms.

Embodied capital theory also offers an explanation for why longevity should be mechanistically linked to brain size. Brain tissue not only requires greater caloric investment to build and maintain than other tissues, it also requires significant inputs of time to make good use of. Indeed, (Aiello 2007) suggests that larger brained organisms are generally slower to achieve maturity because of the complexity of the nervous system they must learn to use effectively. Brain size then may be correlated with longevity for three reasons. First, larger brains are associated with larger organisms, and larger species generally live longer. Second, building and maintaining a large mass of nervous tissue is calorically expensive. Third, the behavioral plasticity, capacity for learning, social complexity and skill acquisition which big brains allow for require an extended development period. This investment may require many years to recoup, requiring longevity, but also decrease mortality, feeding back to allow for greater longevity.

In this paper we examine the allometry of both age at reproductive cessation and of longevity, in relation to brain and body mass in primates. Is reproductive cessation allometrically

appropriate, not only with our ovaries, but also with our brains? Is humans' extraordinary longevity explicable based on our large bodies of our outsized brains? Can the human post-fertile period be attributed to these factors? Rather than testing the hypotheses that allometric scaling relationships exist (and are robust to phylogenetic analysis), which we know to be the case (Judge and Carey 2000) (Hakeem et al. 1996), we examine whether human demography follows the allometric patterns observed in other primates.

MATERIALS AND METHODS:

Primate body mass data (Thoren et al. 2006) and brain mass data (Kappeler and Pereira 2003) were gathered from published compilations. We evaluated three potential sources of longevity data. We took the longest lifespan provided for each species by Carey and Judge (2000), MaxCJ) as the maximum lifespan for that species. We repeated this procedure using a more recent compilation by de Magalhaes et al. (2009), MaxDM). Finally, we included a life-table based measure of lifespan, labeled as age Z. Age Z represents the near-end of lifespan of a population. Operationally, we define Z as the first age by which at least 95% of total time lived is past. Where l_x represents the probability of an individual surviving from age 0 to age x, Z is defined by:

$\sum_{x=0}^{z} l_x \geq 0.95 \sum_{x=0}^{\infty} l_x$, for Z an integer.

Z represents the age by which 95% of all individual-years lived by an average cohort are past. As such it is potentially more resistant to outliers, false records and correlation with sample size than are maximum longevity estimates. It is also potentially more susceptible to inconsistency in

quality of care received by captive populations, in that short-lived as well as long-lived individuals contribute to the calculation of Z. We included Z values only for commonly kept species thought to do well in captivity; most maximum longevity records are also from captive individuals. While captive animals in the institutions from which we are drawing data are generally given the best available care, individuals may suffer from lack of exercise, overeating, and lack of species-specific medical research and technology. Nevertheless, captive primates of most commonly kept species outlive their wild relatives and therefore are thought to represent our best estimates of a species' capacity for longevity in the absence of extrinsic threats such as predators and famines (Austad and Fischer 1992).

Demographic data (other than maximum longevities) for non-humans were calculated based on data from the International Species Information System (ISIS), which compiles data on zoo animals (Earnhardt et al. 1995). We measured age at reproductive cessation as age M. Operationally, we define M as the first time period of age at which 95% of life time fecundity has been realized, on average, abstracting from mortality. That is:

$$\sum_{x=0}^{M} m_x \geq 0.95 \sum_{x=0}^{\infty} m_x \text{ for M an integer.}$$

This parameter estimates mean age at last birth, which generally precedes physiological menopause by several years in humans not using birth control. Similarly, the beginning of fecund lifespan is measured as age B. We define B as the first time period of age at which 5% of life time fertility has been realized, on average, abstracting from mortality.

$$\sum_{x=0}^{B} m_x \geq 0.05 \sum_{x=0}^{\infty} m_x \text{ for B an integer.}$$

We included 63 species for which data were available for Z and at least one measure of maximum longevity.

We used the primate section of the preferred species level phylogeny by Bininda-Emonds et al. (2007) and the Analysis of Traits module of the program Phylocom (Webb et al. 2008) for phylogenetic analyses. The phylogenetic correlation of each measure of longevity was assessed under the expectation that the measure with the least random noise would have the highest correlation with phylogeny. Before inputting data to Phylocom we corrected for differences in mean magnitude between traits by multiplying each MaxCJ value by Mean(Z)/Mean(MaxCJ) and each MaxDM value by Mean(Z)/Mean(DM). Phylocom measures phylogenetic correlation using the method of Blomber et al. ((2003)) which calculates the variance in the magnitude of contrast in a trait between the branches off each node. The more similar related species are in a trait, the lower this variance will tend to be (Webb et al. 2008). Trait values were randomized on the tree 999 times, and the number of these trees where phylogenetic correlation was higher than in the original data were tallied. The higher this number, the more confident we were that the similarity between closely related species was because of that relationship, and did not arise randomly. A noisy measure of longevity was expected to more closely resemble a random distribution across the tree than one in which value differences represented biological differences between species. Each of these tests was performed using the same 47 species.

Estimates of species-specific age of reproductive onset and brain and body mass (log transformed) were used individually in regressions to predict Z, MaxCJ and MaxDM. We examined which measure of longevity was most closely correlated with each predictor. Based on these analyses we concluded that Z includes the least random error, and is the most predictable allometrically of our measures of longevity (see results), and continue our analyses using Z.

We then ask whether Z and M in humans are "predicted by primate patterns" (Judge and Carey 2000) of allometry. Due to the difficulty in singling out a human population experiencing an environment exactly comparable to that provided to well-managed zoo animals, we employ two demographically extreme human populations as upper and lower bounds for demographic parameters. Dobe !Kung hunter-gatherers (Howell 2000) have among the shortest lifespans of any well-studied human population, and modern Japanese women (Human Mortality Database (2008)) have among the longest lives of any human population. We assume that the demographic influences experienced by primates in the ISIS database would not lead to human longevity shorter than the !Kung (Z=67) or longer than the Japanese (Z=86). This assumption rests on the idea that animals in well-run zoos are no worse protected and cared for than hunter-gatherers, and no better protected or cared for than the longest-lived modern humans. While generally not stated as explicitly, this assumption is common to most studies comparing the longevity of humans to captive primates (Allman et al. 1993; Hakeem et al. 1996; Judge and Carey 2000; Kaplan et al. 2007).

We generated prediction formulae for natural log transformed Z and M by excluding humans and fitting linear models using Log(Brain Mass) and Log(Body Mass) and their interaction as model effects. Using the small-sample variant of Akaike's Information Criterion based model selection (Johnson and Omland 2004), we examined which of these predictive terms were useful to include in the final model. We calculated Akaike weights, which give the likelihood that a model is correct, given the range of available models. In each case the preferred model has brain as the only model effect. We then inserted !Kung brain and body masses into the prediction formulae based on the preferred model, and compared the predicted values to the observed demographic values based on !Kung morphology. As taxonomic groups more closely related to humans may produce more meaningful allometric baselines (Allman et al. 1993), we

repeated this analysis using data on only anthropoids (monkeys and apes) and with only catarrhines (Old World monkeys and apes). Because using only one representative per genus may avoid undue influence of species-rich genera, we generated further predictions by repeating each analysis using one randomly selected representative of each genus, using only one representative of each anthropoid genus, and one representative of each catarrhine genus. We predicted that if human demography is allometrically consistent with other primates, the range of estimates of Z and M produced by these models would fall somewhere between observed values for !Kung and Japanese women.

Results:

Parameters for each species or human population included in analysis are given in the appendix. All three measures of longevity (Z, MaxCJ and MaxDM) are significantly correlated ($p < 0.001$) with B (age of reproductive onset), brain mass and body mass (Table 1). Z is the only measure of longevity to be strongly correlated ($r^2 > .6$) with all three predictive variables, and is the best fitting measure for both brain and body mass. Interestingly, B is slightly more closely correlated with Max CJ and Max DM than with Z, despite the fact that Z and B are calculated from the same data.

Z is also the measure of longevity in which closely related species are most likely to have very similar longevity values. The variance of contrasts (between sister taxa) for Z (2.449) was much lower than the same statistic for MaxDM (7.092) or MaxCJ (8.135). Phylocom's randomization test produced 0 out of 999 scenarios in which Z could be more closely correlated with phylogeny, but 18 scenarios in which MaxCJ was more closely correlated with phylogeny, and 21 in which MaxDM was more closely correlated with phylogeny. Z being most closely

correlated with both phylogeny and brain and body mass, we continued our analysis with Z as our sole measure of longevity.

Based on Akaike's Information Criterion (Table 2), the preferred model in each case included brain mass, but excluded body mass and their interaction. Prediction formulae for demographic parameters Z (longevity) and M (age at reproductive cessation) based on regression against brain mass (with humans excluded during model construction) are presented in Table 3.

Table 4 provides predicted values from each model of Z, M and Z-M and observed values for !Kung, and for the Japanese population in 2001. Taxon sampling scheme had relatively little effect on predicted values of Z and M. Analyses limited to more closely related taxa tended to produce predictions closer to observed human Z values, and models using multiple species per genus produced more accurate values of Z than those relying on one species to represent the genus. Nevertheless, observed values for human Z (67-86 years) exceed all five predictions. In contrast, estimates of M exceed observed values only slightly, and there is no tendency for more closely related taxa to produce better predictions of human M, and using single or multiple species per genus had no meaningful effect. Indeed the highest, and; therefore, least accurate estimates for M (49.1-49.2 years) were derived using only catarrhines, the most closely related to humans of the groups examined. Catarrhine based models also yielded the lowest r^2 values (0.45-0.59), suggesting that the relationship between longevity and brain size may be more variable within the catarrhines than in other primate taxa. Z-M, a measure of post-reproductive lifespan, is significantly underestimated in every model, despite zoo primates' greatly expanded post-fertile survival compared to their wild relatives.

DISCUSSION:

Our analyses contradict previous conclusions regarding the allometric and phylogenetic underpinnings of women's post-reproductive lifespan. This difference in conclusion arises largely because of the measure of longevity we employ, Z. Previous authors have recognized that human maximum longevity is not directly comparable to non-human maximum longevity data, and compared maximum longevities in other species to non-maximum, but old ages in humans. Eschewing maximum longevity, we are able to show that the range of values of Z observed in humans and the range of values of Z predicted from an assortment of taxon sampling schemes using non-humans do not overlap.

A methodological improvement with relatively less effect on our conclusion is our use of model selection (models using only body mass or brain and body mass produce similar results, with lower r^2 values, to those shown). Where other authors have concluded that human longevity is successfully predicted based on brain and body allometry, we find that body size adds little predictive power to the allometric model. Judge and Carey (2000) assumed that body size was important before adding brain mass as an additional effect. Allman et al. (1993) evaluated the usefulness of the mass of several organs in predicting primate longevity, and concluded that brain mass is most predictive. Body mass is not a meaningful addition to our models primarily because brain mass encodes much of the information on body mass, and adds information on encephalization (brain mass relative to body mass), which is independently correlated with longevity (Allman et al. 1993). Body mass makes the model more complex, while adding little additional information.

For these reasons, we conclude that human longevity is not predicted by allometric scaling with brain and body mass. Rather our examination of ages at reproductive cessation suggests that human's reproductive longevity is allometrically appropriate, but that allometry cannot explain the post-fertile survival enjoyed by women.

It has been argued that the allometric predictability of women's reproductive cessation reveals physiological constraints on the effects of natural selection, such that adaptive explanations are unnecessary (Cohen 2004), a view we do not share. Women in natural fertility populations have their last child some years (mean=~6 years) before menopause (Bongaarts 1982). At a proximate level, menopause is caused by depletion of oocytes in the ovaries. Gosden and colleagues (Faddy and Gosden 1996; Gosden and Telfer 1987a); (Gosden and Telfer 1987b) have shown that humans' mid-life menopause is determined not by disproportionately small ovaries, but by proportionate ovaries and unexceptional rates of follicle wastage coupled with exceptional longevity. Mean natural age at menopause in humans plus or minus two standard deviations gives a range of 45-57. This variation is highly heritable (0.85 to 0.87 for singleton sisters, (De Bruin et al. 2001)) suggesting that lack of heritable variation is not what prevents the evolved lengthening of the human reproductive lifespan. Similarly, arguments suggesting that human longevity has increased too recently for reproductive lifespan to keep pace, (e.g., (Fedigan et al. 2007)) are challenged by the infrequent but not unheard of natural pregnancies of women in their mid 50s. Given women's lifespan, it is notable that they have not evolved mechanisms allowing later menopause. Possible mechanisms could include larger ovaries, slower follicle wastage, or investment in adult replacement of oocytes, a capacity we now know some mammals possess (Eggan et al. 2006). The lack of any such adaptation in the presence of considerable heritable variation, and its nearly normal distribution around the mean, suggests that directional selection on age at menopause is weak, absent or counteracted by opposing selective pressures.

In light of this, and human's allometrically inappropriate longevity, the allometrically appropriate timing of human menopause must be explained in terms of its fitness effects, in addition to the proximate terms of allometry, endocrinology and the ontogeny of follicle depletion. Fitness benefits could include the ability to have more offspring, or space them further.

But having more children late in life poses mortality risk to the mother and creates competition for care and resources from pre-existing children and grandchildren. Add to this that building and maintaining larger ovaries, or the capacity to maintain follicles more efficiently, would require the redirection of physiological capital from other purposes. These physiological costs may explain why women's reproductive cessation is allometrically predictable, but only in concert with the fact that other selective pressures do not justify bearing these increased costs.

Humans are the only primate species in which post-fertile individuals have been shown to contribute significantly to the fitness of younger kin (Pavard et al. 2008; Pavard et al. 2007). Post-fertile women cannot engage in the reproductive activities most fundamental to other female primates. In addition to not producing babies, they generally do not lactate and most do not have their own infants to care for. In contributing meaningfully to the care and provisioning of grandchildren and older offspring, post-fertile women's reproductive efforts, and the risks and costs associated with them, are different than any other organism, fertile or post-fertile. If allometric patterns are caused by differential scaling of costs and benefits within a structurally consistent system of trade-offs, then novel life-stages, novel forms of reproduction, and novel avenues to costs and benefits should be expected to cause exceptions to and modifications in those patterns (Kaplan and Robson (2002)). We suggest that the failure of allometry to predict women's longevity derives from the indirect and novel manner in which older women reproduce.

ACKNOWLEDGEMENTS:

This work was vastly improved by insightful comments from Leslea Hlusko, Ronald Lee, Eileen Lacey, Craig Moritz and Walter Koenig. Nik Hajny and Jay Stampfl provided valuable assistance with data analysis. We thank ISIS for providing the majority of the data. Financial support to D. Levitis was generously provided by the National Institute on Aging, via the Ruth L.

Kirschstein National Research Service Award (NIA grant T32-AG000246), by Ronald Lee's subproject in P01 AG022500, by Ronald Lee's chair fund, by the Museum of Vertebrate Zoology and by the Max Planck Institute for Demographic Research.

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Table 1. Correlations of three measures of longevity with three variables with which longevity is known to scale allometrically.

r^2 values	Z	MaxDM	MaxCJ
B	0.78	0.86	0.8
brain mass	0.72	0.58	0.5
body mass	0.72	0.72	0.56

# species included	Z	MaxDM	MaxCJ
B	63	62	47
brain mass	50	49	43
body mass	57	57	43

Three sources of longevity measurements, Z (Chapter 1), MaxDM (de Magalhaes et al. 2009) and MaxCJ (Carey and Judge 2000) were evaluated for use in allometric models partly based on which was most highly correlated with three variables known to scale allometrically with longevity, B (age at reproductive initiation), brain mass and body mass. Z, the measure most consistently highly correlated with these variables, was used in predictive models. Numbers of species of captive primates included in each analysis are given. Humans were excluded from analysis during model selection. For all tests $p<0.001$.

Table 2. Model Selection for predictive models using Akaike's Information Criterion

Dependent variable	Model Effects	AICc	Relative likelihood	Akaike Weight
log(Z)	brain	-23.021	1.000	0.555
log(Z)	brain and	-20.854	0.338	0.188

	interaction			
log(Z)	brain and body	-20.688	0.311	0.173
log(M)	brain	-31.617	1.000	0.562
log(M)	brain and body	-29.393	0.329	0.185
log(M)	brain and interaction	-29.308	0.315	0.177

Small-sample Akaike's Information Criterion AICc values were calculated for model selection. The three most likely models for each dependent variable are shown. Model effects are log(brain mass), "brain;" log(body mass), "body;" and log(brain mass)*log(body mass), "interaction."

Table 3. Prediction formulae for human M and Z values based on 6 taxon sampling schemes

Taxon sampling	Dependent	Intercept	Slope	r2	df	F
All Species	Log(M)	2.211	0.221	0.76	48	150.38
All genera	Log(M)	2.218	0.222	0.78	32	114.69
Anthropoid species	Log(M)	2.229	0.216	0.69	38	85
Anthropoid genera	Log(M)	2.232	0.217	0.69	24	53.89
Catarrhine species	Log(M)	2.232	0.277	0.45	23	23.6
Catarrhine genera	Log(M)	1.922	0.274	0.56	13	17.42
All Species	Log(Z)	2.278	0.240	0.74	48	138.09
All genera	Log(Z)	2.335	0.226	0.72	32	84.4
Anthropoid species	Log(Z)	2.189	0.255	0.71	24	58.78
Anthropoid genera	Log(Z)	2.126	0.269	0.76	38	119.96
Catarrhine species	Log(Z)	1.867	0.315	0.59	23	34.22
Catarrhine genera	Log(Z)	1.834	0.317	0.59	14	19.85

Six predictive models using data from zoo primates on brain mass, longevity (Z) and age at reproductive cessation (M) were generated using different sets of species (all

primates in sample, anthropoids only or catarrhines only, each with all species included or one species acting as representative of each genus). Regression was log-log with brain mass as the dependent variable.

Table 4 Estimated and observed demographic parameters

Data used	Z	M	Z-M	# species
All species	53.8	44	9.8	49
All genera	50.6	43.9	6.7	33
Anthropoid species	57.2	43.9	13.3	40
Anthropoid genera	53.5	43.1	10.4	22
Catarrhine species	58.1	49.1	9	26
Catarrhine genera	56.2	49.2	7	20
Observed value, !Kung	67	42	25	-
Observed value, Japan	86	37	59	-

Regression models (table 3), were used to predict parameters M and Z for humans based on brain mass of 1250g. Compared to actual human populations, Z was underestimated and M was slightly overestimated. Z-M, a measure of post-reproductive lifespan, was greatly underestimated by all models, primarily due to error in Z (8.9 to 16.4 years below !Kung), rather than error in M (1.1 to 7.2 years above !Kung). Models based on catarrhine data generated higher estimates of both Z and M and slightly lower estimates of Z-M than other models.

Appendix 1: Demographic and morphological parameters used

Population	M (years)	B (years)	Z (years)	brain mass (gm)	body mass (kg)
Alouatta caraya	21	4	23	56.7	4.33
Aotus lemurinus	17	3	17		0.859
Aotus trivirgatus	22	3	19	18.2	0.77
Ateles fusciceps	32	6	39	114.7	9.16
Ateles geoffroyi	30	5	38	110.9	7.29
Callimico goeldii	17	3	13	10.8	0.47
Callithrix argentata	11	2	11		0.38
Callithrix geoffroyi	15	2	12		0.345
Callithrix jacchus	13	2	14	7.9	0.34
Callithrix penicillata	10	2	9		0.307
Callithrix pygmaea	12	2	12	4.2	0.122
Cebus apella	29	5	35	71	2.52
Cercocebus torquatus	23	5	25	109.6	5.85
Cercopithecus ascanius	22	3	25	66.5	4.10
Cercopithecus diana	23	5	27	77.3	3.9
Cercopithecus neglectus	22	5	24	70.8	4.13
Cheirogaleus medius	12	1	18	2.9	0.139
Chlorocebus aethiops	19	3	26	59.8	3.34
Colobus guereza	26	5	22	73.5	8.55
Erythrocebus patas	21	3	25	106.6	6.5
Eulemur fulvus	22	3	27	29.2	2.08
Eulemur macaco	21	2	28	25.6	2.14
Galago moholi	8	1	10		0.194
Galago senegalensis	10	2	11	4.8	0.22
Gorilla gorilla	35	8	43	505.9	80
Hylobates lar	34	7	41	107.7	5.34
Hylobates syndactylus	32	7	37	121.7	10.7
Lagotricha lagotricha	23	5	27	96.4	7.09
Lemur catta	23	2	26	25.6	2.21
Leontopithecus chrysomelas	13	2	18		0.57
Leontopithecus rosalia	13	2	17	12.9	0.6
Macaca fascicularis	21	3	27	69.2	3.59

Macaca fuscata	21	4	28	109.1	8.03
Macaca mulatta	24	4	30	95	7.09
Macaca nemestrina	18	3	28	106	5.7
Macaca nigra	32	5	25	94.4	6.14
Macaca silenus	24	4	33	85	6.1
Macaca sylvanus	19	4	22	93.2	11
Mandrillus leucophaeus	20	4	28		
Mandrillus sphinx	22	5	28	159.4	12.8
Microcebus murinus	12	1	13	1.8	0.07
Nycticebus coucang	15	2	16	10	0.82
Nycticebus pygmaeus	11	2	15		0.376
Otolemur crassicaudatus	13	2	18	11.8	1.11
Pan troglodytes	42	9	46	410.3	40.37
Papio hamadryas	32	4	34	165.3	10.65
Pithecia pithecia	22	4	25	31.7	1.58
Pongo abelii	36	9	47	413.3	45
Pongo pygmaeus	36	9	45	413.3	35.7
Saguinus fuscicollis	17	3	19	9.3	0.36
Saguinus geoffroyi	15	3	12	10.5	0.503
Saguinus imperator	13	3	14		0.48
Saguinus labiatus	17	3	14		0.53
Saguinus midas	12	2	15	10.4	0.58
Saguinus oedipus	18	3	19	9	0.45
Saimiri boliviensis	19	4	22		0.75
Saimiri sciureus	19	3	25	24.4	0.7
Semnopithecus entellus	20	4	20	135.2	13.52
Therapithecus gelada	19	4	23		11.7
Trachypithecus auratus	20	4	20		
Trachypithecus obscurus	24	4	22	67.6	6.22
Varecia variegata	22	3	29	34.2	3.52
!Kung	42	17	67	1250	40.8
Ache	44		68		
Hadza	42		69		
Least Developed Countries	42		73		
Less Developed Countries	39		78		
More Developed Countries	38		81		
Japan	37		86		

Figure 1: Scaling of age at reproductive cessation (M) with longevity (Z) in humans and non-human primates.

M, a population measure of age at reproductive cessation (see methods) is plotted against Z, a measure of population longevity (ibid), both in years. All data represent females only. Dots represent zoo populations of non-human primate species, and the solid line is the linear regression excluding humans ($M= 3.27+0.73*Z$, $r^2=0.85$, df=61, f=355.4, $p<0.0001$). Numbers are human populations: 1. !Kung, 2. Ache, 3. Hadza, 4. United Nations Least Developed Countries, 5. United Nations Less Developed Countries, 6. United Nations More Developed Countries, 7. Japan in 2001.

The dashed line giving regression through these human populations ($M=65.52-0.33*Z$, $r^2=0.90$, df=6, F=42.75, $p<0.0013$) portrays that with increasing development, human Z increases while M decreases. Natural fertility human populations (1-4) experience M=~42. Human populations range between !Kung (Z=67) and Japan (Z=86) in longevity, and a human demography comparable to zoo primates in environmental influences is assumed to fall between these values. No known human population falls within the 95% density limits around the allometric line generated from other primates. Given the unlikelihood of a real human population falling at the intersection of these two lines (Z=58.2, M=45.9), we see that humans do not follow this allometric rule.

Figure 2: Allometric relationship of Z (longevity) and M (reproductive cessation) to brain size.

Longevity (a) and age at reproductive cessation (b) both scale with brain mass (grams) across the primates. Regressions are log/log, years are displayed linearly for ease of interpretation. Six regression models representing different taxon sampling schemes are shown. Dotted lines are catarrhines only, dashed lines are anthropoids and solid lines are all primates. Thick lines are based on data with more than one species per genus, while thin lines have one representative of each genus in our dataset.

(a) Small dots represent captive non-human primates, large dots are two human populations, !Kung hunter-gatherers (Z=67) and modern Japanese (Z=86), assumed to represent environments respectively less and more conducive to longevity than that experienced by zoo primates. If humans' longevity fit the allometric scaling of other primates, the regression line should pass close to or between the two human populations. Based on these regressions we reject the hypothesis that human longevity is explicable based on brain mass.

(b) Only one human population, the !Kung (M=42, large dot, upper right) is represented, as M varies little between natural fertility populations. The six regression lines overlap considerably, indicating that taxon sampling has relatively little influence on the model outcome. The largest disparity is between models based only on catarrhines, and those based on wider taxa. Catarrhine models overpredict M more so than other models. The

predicted values (44-49.2) overlap with the range of natural human variation, indicating that human's age at reproductive cessation is successfully predicted based on brain mass.

Figure 1.

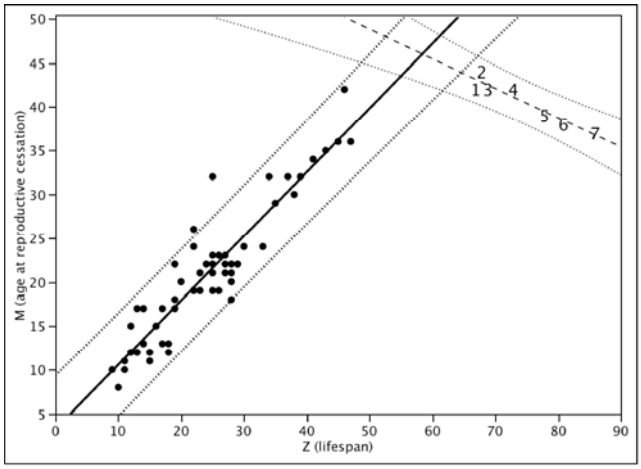


Figure 2a

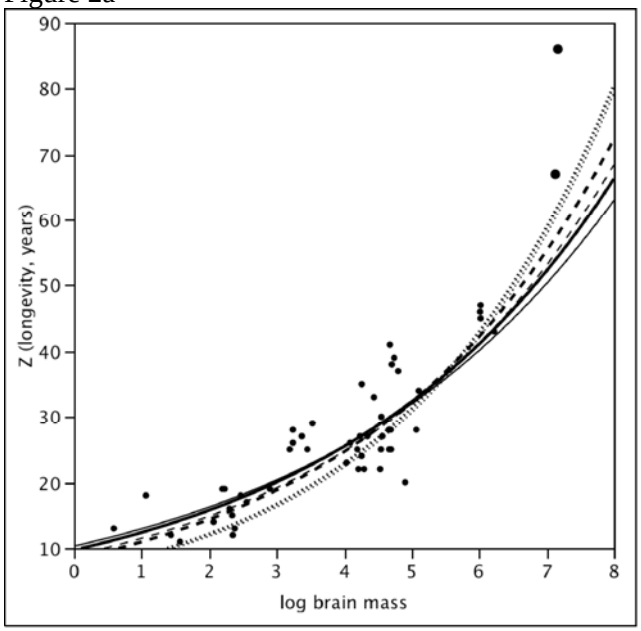
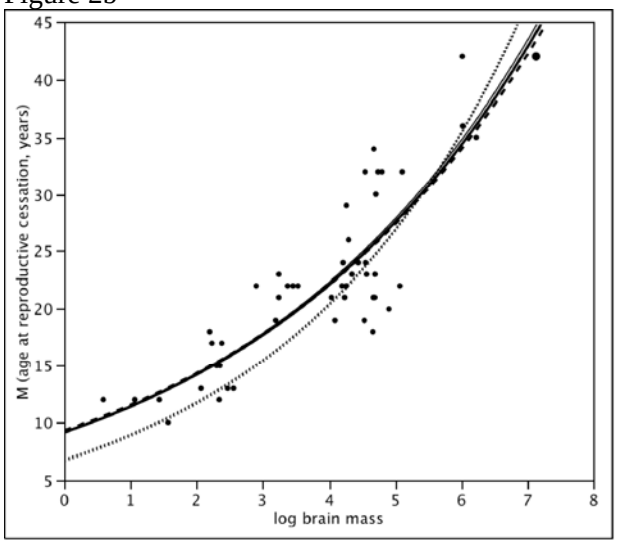


Figure 2b


M (age at reproductive cessation, years)
log brain mass
45
40
35
30
25
20
15
10
5
0
1
2
3
4
5
6
7
8