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Brain weight and life-span in primate species

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ABSTRACT **In haplorhine primates (tarsiers, monkeys, apes, and humans), there is a significant correlation between brain weight and maximum life-span when the effect of body size is removed. There is also a significant correlation in haplorhine primates between brain weight and female age at first reproduction. For strepsirhine primates (lorises and lemurs), there are no significant correlations between brain weight and either life-span or female reproductive age when the effect of body size is removed. This lack of correlation in strepsirhine primates may be related to the fact that these primates are nocturnal and/or natives of the island of Madagascar, both of which conditions may reduce competition for resources and predation pressure. These findings suggest that in haplorhine primates the genetic systems controlling brain growth are linked to the systems governing the life cycle so that species with longer cycles have larger brains. When the effect of body weight is removed, leaf-eating haplorhines have significantly smaller brains and shorter lives than haplorhines with other diets. Harem-living haplorhines also have significantly smaller brains and shorter life-spans than troop-living haplorhines when the effect of body weight is removed. We also sought to test the rate-of-living hypothesis by determining whether primates with basal metabolic rates that are higher than would be expected for their body size have shorter maximum life-spans than would be expected for their body size. Metabolic rate is not correlated with life-span or female age at first reproduction when the effect of body size is removed.**

It has long been postulated that species with larger brains tend to live longer (1–3). In a sample of 63 mammalian species, Sacher found a somewhat stronger correlation between brain weight and maximum recorded life-span (r = 0.88) than between body weight and life-span (r = 0.77). Sacher's conclusion that brain weight was a better predictor of life-span than body weight was challenged by Economos (4), who (using a different data set) found that liver and adrenal weight predicted life-span about as well as brain weight. He concluded that the brain has no special relationship with life-span. Economos's criticism of the brain–life-span conjecture has been widely accepted (5–7). We decided to reexamine this issue in primate species by using a statistical technique to remove the effect of body weight because larger animals tend to live longer. We reasoned that if the brain–life-span conjecture were true, one would predict that primates with brains larger than would be expected for their body size would also have longer life-spans than would be expected for their body size. Lindstedt and Calder (8) found that, for a sample of mammals containing a wide range of sizes, many life cycle parameters including maximum life-span in captivity and age at reproductive maturity scale with body weight at very similar slopes. We reasoned that if there were a relationship between relative brain size and maximum life-span, there might be a similar relationship between relative brain size and the duration of parts of the life cycle.

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Finally, we also sought to test a popular alternative to the brain–life-span conjecture, the rate-of-living hypothesis [see Finch (7) for a review]. To test this theory, which is epitomized by the expression "live fast, die young," we sought to determine whether primates with basal metabolic rates that are higher than would be expected for their body size have shorter maximum life-spans than would be expected for their body size.

MATERIALS AND METHODS

We used maximum recorded life-span because it should measure, under ideal circumstances, the genetic potential for longevity for each species. To obtain the maximum life-span data, we queried 190 zoos and research institutions throughout the world. We received 138 responses to our queries. Our queries were guided in large part by the International Species Inventory System (ISIS), which lists primate holdings. ISIS provides age information but unfortunately only up to 20 years. Thus, we used ISIS locations of older primates to direct our queries. We also obtained information from Marvin Jones (registrar of the Zoological Society of San Diego), who has made a long-standing practice of recording longevity data from throughout the world. We sought the name, sex, date of birth or acquisition, estimated age of wild-caught primates, and date of death for long-lived representatives of each species. We obtained maximum life-span data and brain and body weights of 65 of these species, which are the basis of the data used in this paper. The quality of the life-span data is limited by two major factors. First, many of the long-lived primates were born in the wild and their age at acquisition could only be estimated; we used only conservative estimates for the age at acquisition. Second, because of improved husbandry, in 28 species the maximum life-span record is for a living animal. Thus, the maximum life-spans will be underestimated for these species. All of the life-span data are for primates in captivity. There are not sufficient data to determine the maximum life-spans for primates living under natural conditions because there are very few natural primate populations that have been under continuous observation for long enough periods to obtain maximum life-span data. There also are not sufficient data to measure sexual differences in life-span for a large number of species. Because of improved primate husbandry and record keeping, the maximum life-spans we obtained were considerably higher than those previously reported (6, 9–11). Due to space limitations, the life-span records will be published separately. The maximum recorded human life-span was obtained from the *Guinness Book of World Records* (12).

We suspected that because of small sample sizes the life-spans for rare species might be underreported. On examining this possibility, we found that there does exist a small but significant correlation between life-span residuals and the number of animals of a particular species in captivity (n = 60; r = 0.289; P = 0.025), but the major axis regression slope was so close to 0 (s = 0.000120) that the effect on our data was negligible.

We obtained data for adult and neonatal brain and body weights from a data base compiled by Bob Martin and

colleagues at the University of Zurich (personal communication). This was supplemented by brain and body weight data from Stephan *et al.* (13) and from Brauer and Schober (14). Data for brain structure volumes were obtained from Stephan *et al.* (13) and from Frahm *et al.* (15). For stages in the life cycle, we used gestation lengths obtained from Bob Martin's data base. We also used data on female average age at first reproduction from a published list compiled by Ross (16). The weights of the heart, kidneys, liver, and adrenals were obtained from Altman and Dittmer (17). Data on diet and social organization were obtained from the book *Primate Societies* (18) and from a paper by Clutton-Brock and Harvey (19). We obtained basal metabolic rates from an unpublished data base compiled by Bob Martin. This data set includes only those primate species in which basal metabolic rate measurements were conducted on a resting, postabsorptive adult primate in the thermoneutral zone of ambient temperature (20). We used SYSTAT 5.2 to assist us in the statistical analysis.

RESULTS

Fig. 1 illustrates the relationship between body and brain weight (Fig. 1*A*) and between body weight and life-span (Fig. 1*B*). The distance in the *y* dimension between the regression line and each data point was added to 1, giving a value >1 for points that fall above the line and <1 for points that fall below the line. This value is the residual value for each species. The addition to 1 was used to make all the residual values positive. We used the least-squares regression as the basis for calculating brain and life-span residuals because this procedure removes the effect of body size plotted along the *x* axis (21). For example, the human data point (cross) lies considerably above the regression for brain–body weight; this distance is the human brain residual. Similarly, there is a large residual for human life-span relative to body weight. Thus, the residuals indicate that humans have both a larger brain and a longer life-span than one would expect for a primate of the same body weight. We have sought to determine, by using a Pearson correlation, to what degree these residuals for primate species are correlated. In the following discussion, N is the sample size, r is the Pearson correlation coefficient, and P is the probability associated with the χ^2 test of the correlation's significance.

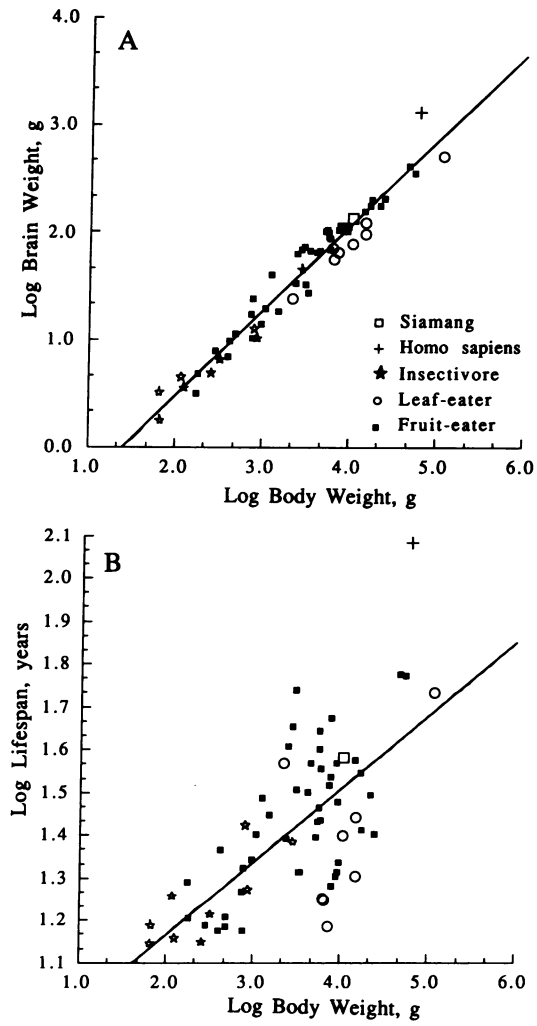

FIG. 1. (*A*) Log brain weight (g) vs. log body weight (g) for the entire set of species, both haplorhines and strepsirhines ($N = 65$; $r = 0.974$; $P < 0.001$; slope = 0.791). (*B*) Log life-span (years) vs. log body weight (g) for the entire set of species ($N = 65$; $r = 0.675$; $P < 0.001$; slope = 0.171). Effects of body weight were removed from the brain weight and life-span data by taking the residuals from these lines. We used least-squares regressions in these plots because they depend solely on *y* values and thus are not influenced by body weight.

For haplorhine primates (tarsiers, monkeys, apes, and humans), the residuals for brain and life-span are correlated (Table 1 and Fig. 2). By contrast, in strepsirhine primates (lorises and lemurs), the brain and life-span residuals are not correlated. In haplorhines, neonatal brain weight residuals are also significantly correlated with life-span residuals (see Table 1).

The distribution of brain–life-span residuals reflects dietary specializations (Fig. 2*A*). Leaf eaters (circles) are in the lower left part of the distribution with smaller brains and shorter life-spans; fruit eaters (squares) are in the middle and upper right with larger brains and longer life-spans; the small numbers of insect eaters (stars) are mixed with the fruit eaters; the omnivorous human (cross) is the extreme upper right data point. The leaf eaters have significantly smaller brains and shorter life-spans for their body sizes. Fig. 2*B* classifies primates according to social structure and reveals that the harem-living species have significantly smaller brains and shorter life-spans than do haplorhine primates with other types of social organization.

Fig. 3 illustrates the distribution for great apes (gorillas, orangutans, chimpanzees) and humans. The correlation coefficient for the great apes and humans is very high ($r = 0.989$; $P = 0.017$). The same relationship with dietary specialization is present in ape and human samples as in haplorhines as a whole. The slope of the major axis regression for great apes and humans is about half the slope for the whole haplorhine group.

We sought to determine whether the brain residuals might be correlated with other stages of the life cycle. Fig. 4 illustrates the relationship between brain residuals and the residuals for female average age at first reproduction. While the sample size is smaller and the correlation is somewhat lower, the overall distribution is similar to the life-span residuals and the slope of the major axis regression is close to the maximum life-span slope. Another parallel between the findings for life-span and female average age at first reproduction is that the correlation for strepsirhines is not significant ($N = 9$; $r = 0.476$; $P = 0.196$).

There is no significant correlation between brain residuals and gestation residuals ($N = 34$; $r = 0.147$; $P = 0.408$). This

Table 1. Correlations between brain weight and life-span residuals

	Adult			Neonatal		
	N	*r*	*P*	*N*	*r*	*P*
Haplorhine primates	49	0.657	<0.001	20	0.602	0.005
Strepsirhine primates	16	0.056	0.838	9	−0.062	0.873

N, sample size; *r*, correlation coefficient (Pearson's *r*); *P*, χ^2 probability that correlation is due to random chance.

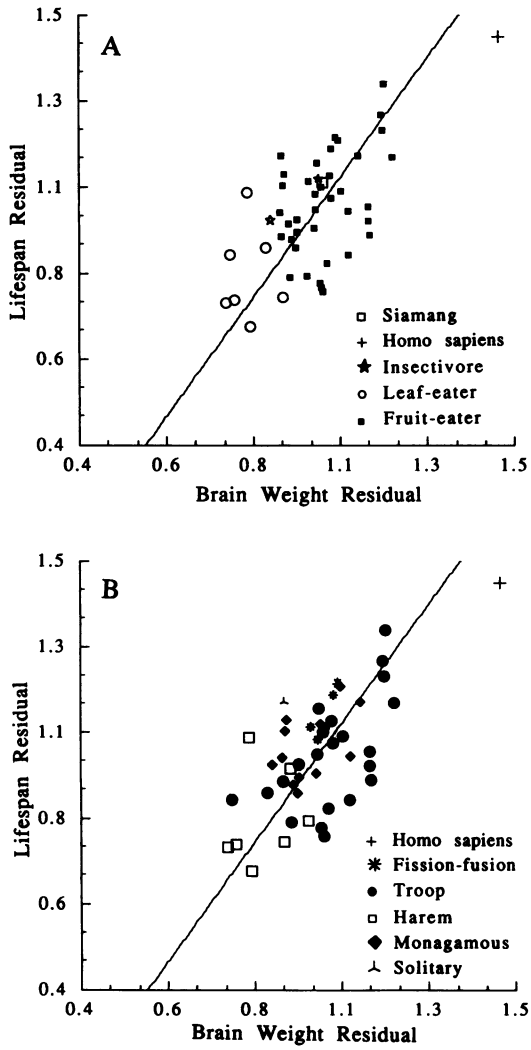

FIG. 2. Life-span residuals vs. brain weight residuals for haplorhine primates ($N = 49$; $r = 0.657$; $P < 0.001$; slope = 1.388). The lines were fit using a major axis regression because it provides a more accurate estimation of the true slope (22). (*A*) Labeled by primary dietary type. Leaf eaters have significantly smaller brains than fruit eaters (least-squares means: leaf eaters = 0.828; fruit eaters = 1.035; $P < 0.001$). They also have significantly shorter life-spans (least-squares means: leaf eaters = 0.835, fruit eaters = 1.028; $P = 0.005$). The two insect-eating species of tarsiers (stars) are not significantly different from the other two groups. The siamang (*Hylobates syndactylus*) has dietary proportions of 43.5% fruit and 43.75% leaves (18). It appears as the open square among the fruit-eating primates' solid squares and was omitted from the dietary statistical analyses because its extremely similar dietary proportions made it difficult to classify. Its location among the fruit eaters is probably due to the much larger proportion of fruit in its diet than in those of the classified leaf eaters. (*B*) Labeled by social structure. Harem-living haplorhines (open squares) have statistically smaller brains than troop-living haplorhines (solid circles) (least squares means: harem = 0.864, troop = 1.037; $P = 0.001$). Harem-living haplorhines also have significantly shorter life-spans than both monogamous ($P = 0.034$) and troop-living ($P = 0.049$) haplorhines (least-squares means: harem = 0.837, monogamous = 1.024, troop = 1.002).

lack of correlation is probably due to wide variability in the stage of development of different primate species at birth.

We also sought to determine the relationships between the weights of other organs and life-span. The relationships for heart, kidney, liver, adrenals, and brain for a set of haplorhines are shown in Table 2. From the raw correlations, which do not remove the effect of body size, one might be tempted to conclude along with Economos that brain weight

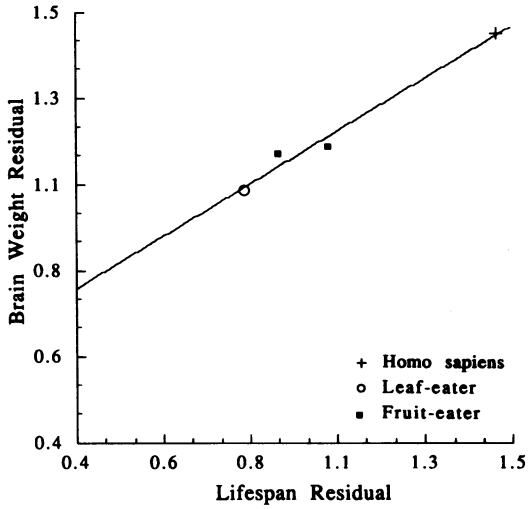

FIG. 3. Life-span residuals vs. brain weight residuals for (left to right) gorilla, orangutan, chimpanzee, and human. Correlation is very good ($N = 4$; $r = 0.989$; $P = 0.017$; slope = 0.609). A major axis regression line was used here as in Fig. 2.

is only a slightly better predictor of life-span than other organs; however, an entirely different picture emerges when the effects of body weight are removed for each organ data set by taking the residuals from the organ weight vs. body weight regression line. The correlation between the residuals for brain and life-span for this haplorhine data set is 0.920 with a probability that the correlation is due to random chance of <0.001. By contrast, the residuals for the other organs do not correlate significantly with life-span residuals.

Finally, there is no support for the rate-of-living hypothesis for either strepsirhine or haplorhine primates when the effect of body size is removed, because the basal metabolic rate residuals do not correlate with the life-span residuals in either strepsirhine or haplorhine primates (Table 3). There also is no correlation with female age at first reproduction residuals.

DISCUSSION

There is a strong relationship between brain size and life-span in haplorhine primates when the effect of body size is removed. It is particularly strong for the great apes and

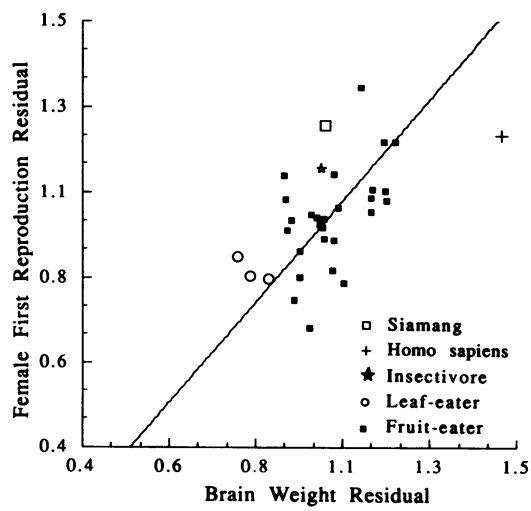

FIG. 4. Residual for female age at first reproduction plotted vs. brain weight residual ($N = 33$; $r = 0.562$; $P = 0.001$; slope = 1.187). Although least-squares mean for leaf eaters is lower than that for fruit eaters, the difference is not significant, possibly owing to the small number of leaf eaters in this set (three) (least-squares means: leaf eaters = 0.857, fruit eaters = 1.002; $P = 0.176$). A major axis regression line was used here as in Figs. 2 and 3.

Table 2. Organ weight correlations

	Log organ weight vs. log life-span			Organ weight residuals vs. life-span residuals		
	N	*r*	*P*	*N*	*r*	*P*
Heart	10	0.795	0.006	10	0.406	0.245
Kidney	9	0.733	0.025	9	0.116	0.767
Liver	9	0.799	0.010	9	0.118	0.741
Adrenals	10	0.769	0.010	10	0.357	0.230
Brain	10	0.890	<0.001	10	0.920	<0.001

N, sample size; *r*, correlation coefficient (Pearson's *r*); *P*, χ^2 probability that correlation is due to random chance.

humans, which probably reflects the close taxonomic affinity of this group, the large populations sampled, and the high quality of the record keeping for these species. The close taxonomic affinity of the great apes and humans may also account for the reduction in slope of the major axis regression for this group as compared to haplorhines as a whole. A hypothesis consistent with these findings is that one of the important functions of the brain is to store information about resources in the environment so that the organism can survive occasional catastrophes by switching to alternative resources. The longer the life-span of the animal, the more likely it is to encounter severe crises during its lifetime. Thus, it might be expected that species with longer life-spans would have larger brains in order to sustain individuals through the more severe crises likely to occur in a longer life. The capacity to survive catastrophes presumably is also related to such factors as the ability to down-regulate metabolism or to subsist on relatively ubiquitous foods during periods of environmental stress.

Strepsirhine brain and life-span residuals are not correlated. We considered the possibility that strepsirhine life-spans might be underreported and that this might account for the lack of correlation. However, strepsirhine life-span residuals relative to body weight are slightly but not significantly greater than those for haplorhines (strepsirhine: 1.019 ± 0.141, mean ± SD; haplorhine: 0.994 ± 0.103, mean ± SD; $P = 0.342$). The lack of difference in average life-span residuals between haplorhines and strepsirhines indicates that life-spans probably have not been underreported for strepsirhines. The lack of correlation in strepsirhines may be due to the fact that all strepsirhine species are nocturnal and/or are native to the island of Madagascar. Nocturnality and the island habitat probably both result in less competition for resources and less predation pressure by other animals, and this may account for the lack of correlation between brain and life-span residuals in this primate group.

The similarity in the correlations and the regression slopes for brain residuals relative to maximum life-span, female reproductive age, and adult life-span suggest that common mechanisms govern these parameters of adult life-span. This interpretation is supported by the finding that female reproductive age is strongly correlated with life expectancy in adults for a broad sample of mammals (23). These findings point to the likelihood in haplorhine primates that the genetic systems controlling brain growth are linked to the systems governing the life cycle such that species with longer cycles have larger brains. When the volumes of different structures in the brain are related to life-span, the correlation for most structures is slightly lower for female reproductive age than for life-span just as is true for the whole brain. However, there are some conspicuous differences. For example, the hippocampus is not correlated with life-span ($N = 26$; $r = 0.257$; $P = 0.205$), whereas the hippocampus is reasonably well correlated with female age at first reproduction ($N = 23$; $r = 0.580$; $P = 0.004$). By contrast neocortical grey matter is

Table 3. Correlations between basal metabolic rate residuals and residuals of life-span and female age at first reproduction

	N	*r*	*P*
Life-span residuals			
Strepsirhine primates	11	0.249	0.460
Haplorhine primates	13	0.220	0.471
Female age at first reproduction residuals			
Strepsirhine primates	6	0.441	0.384
Haplorhine primates	11	−0.138	0.686

N, sample size; *r*, correlation coefficient (Pearson's *r*); *P*, χ^2 probability that correlation is due to random chance.

reasonably well correlated with life-span ($N = 13$; $r = 0.611$; $P = 0.026$), whereas female age at first reproduction is not ($N = 12$; $r = 0.363$; $P = 0.246$). Such differences suggest that the exact mechanisms of the relationships between brain and life-span and brain and female reproductive age may be different. We intend to discuss the analyses with individual brain regions further in another paper.

Harvey *et al.* (6) reported a correlation of 0.55 between brain weight residuals and maximum recorded life-span residuals for primates calculated by subfamily; yet, they dismissed the significance of their finding because of Economos' objections to the brain–life-span conjecture and because they believed that the correlation could be attributed to the relationship between brain and female reproductive age. We have already evaluated Economos' objections. We see no reason to attribute the correlation to female reproductive age as opposed to life-span. We analyzed the brain, body, and life-span data of Harvey *et al.* for primate species and obtained a weak and barely significant correlation between brain and life-span residuals ($N = 51$; $r = 0.257$; $P = 0.050$). We found a stronger correlation for their haplorhine data set ($N = 37$; $r = 0.481$; $P = 0.003$). The correlation found for the haplorhine data of Harvey *et al.* is lower than for our data set probably because they included a mixture of life-spans for captive and wild populations.

Our findings confirm earlier observations that fruit-eating primates have significantly larger brains than leaf-eating primates (6). Fruit-eating bats also have larger brains for their body size than insect eaters (24, 27). A fruit eater's food supply is not constant because different plants bear fruit at different times and at different locations in the complex matrix of the tropical forest (25). As Allman (26) pointed out: "an animal guided by memory of the locations of fruit-bearing trees can more efficiently exploit the available fruit resources than would otherwise be possible; thus natural selection may have favored the development of capacities for visuospatial memory in frugivorous primates." This study has shown that fruit eaters also live significantly longer lives than do leaf eaters when the effect of body size is removed. Because of the ubiquitous nature of leaves, the computational and memory requirements necessary to support a leaf eater might be less than those required to support a fruit eater of the same body size.

Finch's (7) massive comparative study of senescence indicates the taxon-specific nature of the mechanisms governing life-span. Our findings are consistent with this theme in that they suggest that different factors may be involved in the regulation of life-span in haplorhine and strepsirhine primates. This observation gives rise to several intriguing questions. Do brain–life-span residuals correlate for other groups of animals? Are strepsirhine primates unusual among animals in lacking the correlation or are haplorhine primates unique in possessing a strong correlation between brain and life-span residuals?

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