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Brain Size Scaling and Body Composition in Mammals

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Key Words

Mammals · Encephalization · Fat-free weight · Brain size · Body composition · Neanderthal · Sex differences

Abstract

Brain size scales with body size across large groups of animals, but exactly why this should be the case has not been resolved. It is generally assumed that body size is a general proxy for some more important or specific underlying variable, such as metabolic resources available, surface area of the body, or total muscle mass (which is more extensively innervated than is, e.g., adipose tissue). The present study tests whether brain size in mammals scales more closely with muscle mass (and other components of lean body mass) than with total fat. Felsenstein's independent comparisons method was used to control for phylogenetic effects on body composition in organ weight data taken from a previously published comparative sample of 39 species in 8 different orders of mammals, all collected and processed by the same researchers. The analysis shows that the size of the central nervous system (CNS) is more closely associated with components of fat-free weight than it is to fat weight. These results suggest a possible explanation for why metabolic resources and brain size both share the same general relationship with body size across mammals. They also suggest that some measure of lean body mass is a more appropriate scaling parameter for comparing brain size across species than is overall body weight.

Introduction

It has long been known that brain weight scales with body weight across large groups of animals. The correlation between these variables in different studies is very high, typically greater than r = 0.95 [Martin, 1981]. Exactly why this would be the case remains a fundamental question in the study of brain evolution. Although existence of a correlation does not prove direct causation, any truly causal relationship must necessarily also be a correlational one. To the extent we accept that a given correlation is not due to chance, we also accept that some causal connection exists between the two variables, although the connection might only be through the influence of other (perhaps unmeasured) variables. A key step in understanding why two variables are correlated involves exploring the web of associations among possibly relevant variables [Blalock, 1970]. In a 1990 review article in Science, Harvey and Krebs point out that in studies of brain/body size scaling, '... for the most part, body weight is employed as a surrogate measure for some (perhaps unidentified) underlying variable.' [Harvey and Krebs, 1990]. Body

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weight might simply be associated with brain weight (in the statistical sense) without directly causing differences in brain weight. Many biological features covary with body weight, such that the exact nature of the cause-effect relationship between body weight and brain weight remains unclear.

If body weight is just a proxy measure, what is it a proxy for? Two well known hypotheses concern body surface area [Jerison, 1973] and metabolic resources [Martin, 1981; Armstrong, 1983]. Jerison's [1973] data suggested brain and body weight scaled with an allometric exponent of ~ 0.67, which is the same as that relating surface area to volume (among identically shaped objects). He therefore suggested that brain size might be keeping pace with the surface area of the body, perhaps because the amount of information about the external world that a species has access to (and needs to process) might be proportional to surface area [Jerison, 1985]. Martin [1981] used a larger dataset and a more statistically appropriate line-fitting technique (major axis) and found that the allometric exponent was actually closer to 0.76, which matches empirical estimates of the exponent relating basal metabolic rate to body size. From this he hypothesized that the mother's basal metabolic rate might be the key limiting factor for the size of the brain of any offspring. This argument has been criticized on the basis that the association between brain and metabolism disappears after controlling for body size [McNab and Eisenberg, 1989; Pagel and Harvey, 1989], although this simply shows that the variation in brain size that is unrelated to body size is also unrelated to metabolism. Because we are interested specifically in the variance that is related to body size, these criticisms do not disprove the metabolic resources hypothesis. It is likely that the metabolic resources of an organism are relevant to brain size variation in some fashion [Aiello and Wheeler, 1995]. In any case, these studies demonstrate that in order to explain the correlation between brain size and body size, we must fully investigate the web of associations among various biological features in groups of mammals and other animals.

The present study seeks to expand our understanding of these associations by focusing on sub-components of body size. Body size is, after all, the sum of a number of different parts. These parts are clearly inter-related, but they are not perfect functions of each other. Some subcomponents might be more closely related to brain size than others. Given that muscle tissue is more intimately associated with brain function than adipose tissue, it is reasonable to expect that some estimate of muscle mass would scale more closely with brain size than adipose tissue. In fact, this argument has long been used as a possible explanation for the larger average brain size (as estimated by cranial capacity) found in Neanderthal specimens compared to modern humans [Dubois, 1921; Trinkaus and Howells, 1979; Holloway, 1981, 1985]. Neanderthal post-crania suggest they were significantly more heavily muscled than modern humans [Trinkaus and Howells, 1979; Ruff et al., 1993, 1994; Abbott et al., 1996; Trinkaus, 1997]. If brain size varies specifically with muscle mass (or something closely associated), we would expect Neanderthals to have larger brains. In fact, at least one recent study [Wood and Collard, 1999] suggests that the encephalization quotient (EQ: the ratio of actual/predicted brain size) for Neanderthal is essentially the same as in modern humans. Although there are problems with this analysis (EQ's hide large absolute differences in brain size; see discussion below), it supports the contention that muscle and/or lean body mass might be highly relevant for making sense of differences in estimated cranial capacities in hominids.

In addition, the idea that brain weight might scale specifically with fat-free weight has also long been used as a possible explanation for sex differences in brain size (which average about 100 g favoring males, after correcting for differences in total body weight) [Manouvrier, 1903; Gould, 1981; Ankney, 1992]. Sexual dimorphism in fat-free weight is substantially greater than is sexual dimorphism in overall weight (males weigh more and have proportionately less fat).

However, because metabolic resources have been implicated in brain size variation and brain evolution [Aiello and Wheeler, 1995], and because adipose tissue represents an important storage of metabolic resources, it is possible that brain size would vary closely with fat levels across mammals. It is true that muscle also contains significant metabolic stores (primarily in the form of glycogen), and is also very metabolically active. However, glycogen stores in muscle are generally unavailable to other tissues [Lehninger, 1982], and in any case such stores are more limited compared to fat. The body typically has only enough glycogen to supply about a day's worth of basal metabolism, whereas fat stores generally contain enough calories for several weeks [Lehninger, 1982; Campbell, 1993]. From a metabolic perspective, fat would seem to be a particularly important gauge of a species energy resources. However, fat and muscle are obviously not in competition with each other: fat stores exist to supply a variety of energy needs in the body, including those of muscles. So it is reasonable to expect that muscle mass and fat mass would be associated with one another across

species, and therefore that fat and muscle might both scale closely with brain size if metabolic resources are critical.

It remains an open question, therefore, whether brain weight should be more closely associated with fat-free weight, fat weight, or both. This question has never been empirically investigated on any comparative dataset to date, even though the assumption of tight scaling with muscle mass has been repeatedly used as an explanation for brain size differences in various contexts. This study addresses the question directly.

Materials and Methods

There are two ways to approach the question of data selection in comparative studies. Typically, researchers cull data on as many different species as possible. The hope is that the statistical advantages conferred by a large data set will be greater than the errors obviously introduced by the fact that different studies often use different methodologies with different limitations carried out by different investigators at different times. Conversely, one could emphasize consistency of methodology, but typically at the expense of the size of the data set. It is always preferable to use a set of data that includes both brain weight and body composition measurements taken by the same investigators on the same individual specimens, but for practical reasons this effectively limits the diversity of species that one can include. Neither of these approaches to data selection is a perfect solution. If a relationship actually exists across some large range of taxonomic groups, both kinds of studies should point to the same conclusions. In the present study, the second approach, emphasizing consistent methodology, is used. Data were taken from Pitts and Bullard [1968]. In this study, body composition was determined by the same set of investigators through a consistent methodology applied to all specimens: gross dissection of major components followed by the determination of fat content by ether extraction. This appears to be the only large data set of its kind in the literature.

The data includes values for 39 species from 8 different orders of mammals. Their specimens were almost entirely wild-caught in Alaska, Virginia, and Brazil. The weights of the following body segments were taken (or calculated) from their data tables: (1) CNS, brain + spinal cord (which were not separately listed, unfortunately; see below); (2) WT, total body minus gut contents and fur; (3) FFWT, fat-free body minus CNS; (4) FAT, total fat weight; (5) MUSCLE, total muscle weight; (6) BONE, bone (unscraped, and including marrow) weight; (7) HEART, heart weight.

Ten of the 39 species could not be used because CNS data were not reported. When individuals of a species were collected in two different locations, the data from the location which yielded the largest number of individuals were used in the analyses. For one species, *Marmota monax*, two specimens (one male and one female) substantially different in body weight and collected in different regions were reported. These two were averaged together to provide a single estimate for this species.

Because CNS includes the weight of the spinal cord, this measure is not identical to brain weight variables used in other comparative studies. However, because spinal cord weight is much smaller than brain weight (typically ~ 10% of brain weight in primates), and correlates very highly with brain size across mammals [r = 0.95, MacLarnon, 1996], the addition of spinal cord weight should not materially affect the conclusions and analyses presented here.

Table 1 lists the species used, along with the location and numbers of individuals of each sex, and the raw data for these measures as calculated from tables in Pitts and Bullard [1968]. Five of the eight orders of mammals are represented by only one species each: Marsupials, Edentates, Primates, Insectivores, and Lagomorphs. There were six Carnivore species, nine Chiroptera, and 19 Rodent. The possible biasing effects of this uneven representation is discussed below.

The body sizes included in this sample cover a huge range: from just over 7 grams for *Glossophaga soricina* to 9,362 grams for *Gulo luscus* (wolverine). Although this is almost a 1,300-fold difference, it represents only a fraction of the entire range found across all mammals. The conclusions based on this data set, therefore, might not be applicable for species significantly outside this range.

Methodological Considerations

Sex Distribution. There are additional limitations to this dataset. One is that different species are represented by different proportions of males and females. Unfortunately, Pitts and Bullard [1968] do not list separate values for each sex (or for each individual), so it is not possible to use the average of male and female values for the present analysis. However, it is possible to determine the extent to which differences in proportional sex representation explain species differences in degree of fatness by calculating the partial correlation between percent of individuals that were female in the species sample and log FAT, controlling for log WT. This was not significant (r^2 = 0.041, n = 27, independent comparisons), suggesting that variability introduced because of different proportions of females and males across the samples is not likely to materially affect the conclusions.

Seasonality and Geographic Distribution. Another consideration is that species were collected in different seasons and locations, in such a way that seasonality and geographic location are conflated. Pitts and Bullard [1968] report that almost all of the Alaskan and Wisconsin specimens were collected in summer, whereas the Virginia and Brazilian specimens were collected in the winter. Although it is not possible to completely disentangle these variables, it is possible to determine whether fat levels were higher in species collected during the winter rather than the summer. This was done by regressing percent FAT against log WT for all species, and then subjecting the residuals to an ANOVA to see whether winter-collected species had significantly higher residuals than summer-collected species. Figure 1 plots percent FAT against log WT, and shows that Summer-collected species do have a slight tendency to display lower than average percent FAT for their log WT, as compared to the winter-collected species in this sample. An ANOVA of the residuals of this regression (using season collected as the grouping variable) shows that this tendency is not statistically significant (mean summer-collected residual = –1.282, mean winter-collected residual = 0.522, $F_{(1,36)}$ = 1.878, p = 0.179). However, because geographic location is conflated with season for this data, it is still possible that a seasonality effect exists but is hidden from this analysis (though notice that Brazilian specimens – which experience a milder winter – appear to average slightly higher percent fat than the Virginian specimens, which is contrary to the expectation that animals in colder regions would have more percent fat). The regression and correlation analyses were therefore repeated separately within both the winter-collected (Brazilian and Virginian) and summer-collected (Alaskan) sub-samples. If the pattern of associations between variables is *entire-*

Table 1. Species data from Pitts and Bullard [1968] used in this study. See text for definitions of the body composition variables

Order	Family	Genus	Species	Location	♂	♀	Wt*	FAT	FFWT	CNS	HEART	MUSCLE	BONE
Carnivora	*Felidae*	*Felis*	*canadensis*	Alaska		1	7,688.00	1,120.00	6,568.00	105.09	27.59	4,341.45	631.18
Carnivora	*Felidae*	*Felis*	*rufus*	Virginia	1	2	6,152.00	738.00	5,414.00	81.75	25.45	3,600.31	552.23
Carnivora	*Mustelidae*	*Gulo*	*luscus*	Alaska		1	9,362.00	562.00	8,800.00	85.36	80.96	5,271.20	879.12
Carnivora	*Mustelidae*	*Mustela*	*erminea*	Alaska	3		183.30	3.10	180.20	6.69	1.87	104.70	21.98
Carnivora	*Mustelidae*	*Mustela*	*vison*	Virginia	2		1,032.00	66.00	966.00	18.06	7.63	581.53	80.27
Carnivora	*Procyonidae*	*Procyon*	*lotor*	Virginia	3		6,040.00	1,013.00	5,027.00	58.31	36.19	2,920.69	517.78
Chiroptera	*Molossidae*	*Molossus*	*major*	Brazil	3		11.07	0.22	10.89	0.35	0.15	5.51	1.36
Chiroptera	*Phyllostomidae*	*Artibeus*	*jamaicensis*	Brazil	5	9	40.47	3.79	36.18	0.96	0.47	18.02	4.48
Chiroptera	*Phyllostomidae*	*Artibeus*	*lituratus*	Brazil	1	4	63.65	6.22	57.19	1.21	0.74	29.05	8.09
Chiroptera	*Phyllostomidae*	*Glossophaga*	*soricina*	Brazil	2	1	7.22	0.25	7.15	0.37	0.10	3.86	0.69
Chiroptera	*Phyllostomidae*	*Phyllostomus*	*discolor*	Brazil	7		34.37	2.38	32.20	1.00	0.36	16.49	3.87
Chiroptera	*Phyllostomidae*	*Phyllostomus*	*hastatus*	Brazil	2		92.26	5.41	87.05	2.10	0.89	47.01	11.75
Chiroptera	*Phyllostomidae*	*Sturnira*	*lilium*	Brazil		2	15.39	1.21	14.25	0.62	0.16	6.33	2.04
Chiroptera	*Phyllostomidae*	*Vampyrops*	*lineatus*	Brazil		3	22.03	1.59	20.24	0.76	0.24	10.59	2.23
Chiroptera	*Vespertilionidae*	*Eptesicus*	*fuscus*	Virginia	2		17.88	1.51	16.37	0.32	0.19	7.43	2.26
Edentata	*Dasypodidae*	*Euphractos*	*sexcinctus*	Brazil	2		2,459.00	252.20	2,123.00	19.32	12.95	864.06	269.20
Insectivora	*Talpidae*	*Scalopus*	*aquaticus*	Virginia	1		44.64	1.23	43.41	1.01	0.34	21.88	5.30
Lagomorpha	*Ochotonidae*	*Ochotona*	*collaris*	Alaska		1	120.90	7.00	113.90	3.06	0.73	57.18	11.32
Marsupialia	*Didelphiidae*	*Didelphis*	*marsupialis*	Virginia	1	1	1,411.00	107.00	1,304.00	7.56	7.56	681.99	203.42
Primates	*Calllitrichidae*	*Callithrix*	*jacchus*	Brazil	1	3	186.00	8.70	176.20	7.56	1.22	87.92	26.69
Rodentia	*Castoridae*	*Castor*	*canadensis*	Virginia	1	1	9,331.00	865.00	8,466.00	53.34	27.94	4,622.44	897.40
Rodentia	*Cricetidae*	*Clethrionomys*	*gapperi*	Virginia	1		18.34	0.14	18.20	0.64	0.13	9.25	2.22
Rodentia	*Cricetidae*	*Clethrionomys*	*rutilus*	Alaska	10	10	25.27	0.72	24.55	0.60	0.19	11.34	1.94
Rodentia	*Cricetidae*	*Lemmus*	*trimucronatus*	Alaska	3	2	41.62	0.75	40.87	1.03	0.28	19.94	3.82
Rodentia	*Cricetidae*	*Microtus*	*pennsylvanicus*	Virginia	2	5	31.38	1.20	30.18	0.76	0.26	14.46	2.59
Rodentia	*Cricetidae*	*Microtus*	*oeconomus*	Alaska	1	8	24.83	0.45	24.38	0.67	0.19	11.12	2.44
Rodentia	*Cricetidae*	*Microtus*	*pinetorum*	Virginia	1	6	19.41	0.45	18.96	0.57	0.15	9.46	1.86
Rodentia	*Cricetidae*	*Ondatra*	*zibethica*	Virginia	3	5	1,180.00	86.00	1,094.00	7.11	3.50	679.37	115.96
Rodentia	*Cricetidae*	*Oryzomys*	*palustris*	Virginia	1		61.62	7.88	53.74	1.11	0.34	26.92	5.33
Rodentia	*Cricetidae*	*Peromyscus*	*leucopus*	Virginia	5	4	16.99	0.59	16.40	0.61	0.17	8.02	1.49
Rodentia	*Cuniculidae*	*Cuniculus*	*paca*	Brazil	1		1,565.00	196.50	1,368.00	29.00	7.80	737.35	140.90
Rodentia	*Dasyproctidae*	*Dasyprocta*	*aguti*	Brazil	1	1	2,097.00	263.40	1,833.80	25.86	13.94	1,115.13	168.53
Rodentia	*Erethizontidae*	*Erethizon*	*dorsatum*	Virginia	1	1	5,339.00	674.00	4,725.00	37.80	24.10	2,197.13	576.45
Rodentia	*Muridae*	*Mus*	*musculus*	Virginia	2	2	15.88	0.96	14.92	0.48	0.15	7.07	1.22
Rodentia	*Sciuridae*	*Citellus*	*undulatus*	Alaska	3	1	479.00	21.00	458.00	6.00	2.56	257.85	38.01
Rodentia	*Sciuridae*	*Marmota*	*caligata*	Alaska		2	3,558.00	749.00	2,809.00	20.37	16.57	1,671.36	257.02
Rodentia	*Sciuridae*	*Marmota*	*monax*	Alaska, Virginia	1	1	2,194.00	536.50	1,657.50	12.73	8.11	817.13	149.49
Rodentia	*Sciuridae*	*Sciurus*	*carolinensis*	Virginia	1		499.00	11.00	488.00	8.88	2.83	306.46	51.73
Rodentia	*Sciuridae*	*Tamiasciurus*	*hudsonicus*	Alaska	9	2	192.80	3.80	189.00	5.50	1.68	114.16	18.16

* WT excludes gut contents and fur.

ly explained by location/seasonality, then these within-subgroup analyses will show null results. Thus, this analysis will be useful even though it cannot strictly rule out seasonality effects.

Control for Phylogenetic Effects. Because calculations using individual species as data points do not take into account the potential for statistical bias caused by the non-independence (in a phylogenetic sense) of these species, Felsenstein's [1985] independent comparisons method was also applied, as implemented by Purvis and Rambaut's CAIC software package [Purvis and Rambaut, 1995]. This method compares sets of differences between different nodes in a phylogenetic tree. Each data point therefore does not represent an individual species, but rather the difference between a given pair of species (or nodes in the phylogenetic tree). Because these paired comparisons potentially represent differences accumulating in vastly differing amounts of time, they cannot be considered to have the same expected variance, so they are weighted by a factor proportional to the amount of evolutionary time presumed to separate the two nodes [Felsenstein, 1985]. The specific phylogenetic hypothesis used in this study is shown in figure 2. It is based on immunological distance data collected over the last 30 years by the Wilson and Sarich Lab at U.C. Berkeley [Vincent Sarich, personal communication].

Felsenstein [1985] pointed out that the problem of overestimating the statistical independence of species data does not apply if the characters of interest respond very rapidly to natural selection, although he cautioned that, 'It may be doubted how often phylogenetic inertia is effectively absent' (p. 6). The facts of human brain evolution, however, demonstrate that brain size and body weight can be dramatically decoupled in a very short time (by evolutionary standards). Consistent patterns of brain/body weight associations across species therefore might be evidence of natural selection maintaining such patterns, rather than phylogenetic inertia. Nevertheless, it would seem prudent to analyze the data using independent contrasts in addition to the raw species values.

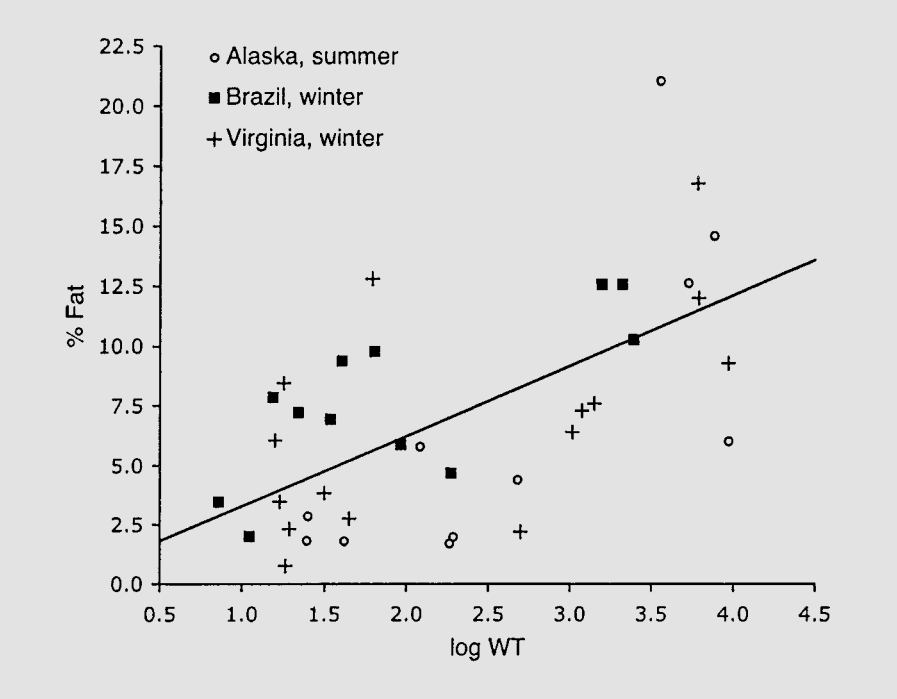

Fig. 1. Relationship between log WT and percent FAT for species collected in summer vs. winter, with geographic location of the specimens also coded. Line represents the least-squares regression (% Fat = 0.339 + 2.942(log WT); $r^2 = 0.388$, $p < 0.001$).

Correlations between Parts of a Whole. Another crucial consideration is that all the associations with CNS will likely be high, whether or not they have any direct causal connection with CNS. This is true because cross-species variability of any sub-component of WT is necessarily constrained by the variability of WT itself. These variables are thus not truly independent of one another, thus invalidating the traditional statistical tests applied to correlations. Because the species in this study differ so widely in body size, and because FFWT, MUSCLE, HEART, BONE, and CNS obviously cannot be larger than their respective species body sizes, there will likely be a correlation between any two of these components. The larger the variability in WT across the species used in an analysis, the larger the correlations will tend to be between sub-components of WT in that sample. It is possible, however, to estimate the size of this effect. To do this a set of 1000 dummy body component variables was calculated, to which (for each species individually, and for each of the 1000 dummy variables) a random proportion of that species WT was assigned. These variables are therefore constrained only by the species WT, and are random subcomponents of it. Correlations were then calculated between these dummy variables and CNS. This represents the lower-bound, limiting case for the present data set. The degree to which correlations between CNS and other (real) subcomponents of WT might simply be explained by random chance can then be directly estimated through comparison to the distribution obtained for these dummy variables.

The average size of the correlations between CNS and 1000 dummy body composition variables created by randomly assigning subcomponents of WT turns out to be remarkably high: $r = 0.901$ (ranging from $r = 0.731$ to $r = 0.968$). The highest 5% of these ranged from $r = 0.947$ to $r = 0.968$ (fig. 3A). Thus, any correlation between a body component and CNS that is *less* than 0.947 should be not be considered statistically significant at the $p < 0.05$ level. This analysis demonstrates that the interpretation of correlations among subcomponents of WT (or any single summary measure of a set of statistical cases) is not a simple one. Correlations are going to be high regardless of their true causal connections to CNS, simply because both measures are constrained by WT (which varies so much among species).

In addition, independent contrasts were also calculated for the 1000 dummy variables, and correlations [calculated via regression through the origin as is appropriate for independent contrasts; Purvis and Rambaut, 1995] were then calculated between these dummy variable contrasts and CNS contrasts. The correlations between independent contrasts of CNS and the 1000 dummy body composition variables tended to be lower than for the non-independent contrast calculations, averaging $r = 0.649$ (with a clearly skewed distribution ranging from $r = 0.201$ to $r = 0.916$; fig. 3B). The highest 5% of these ranged from $r = 0.837$ to $r = 0.916$, thus indicating that correlations among independent contrasts that are lower than this would not be significant at the $p < 0.05$ level. It is important to note that if the standard statistical assumptions were taken, any correlation equal to or higher than $r = 0.331$ would be considered statistically significant at the $p < 0.05$ level (for $N = 27$, the number of independent contrasts).

Biased Sampling. The independent contrast method does not control for the differences in the number of species sampled within different orders of mammals [Smith, 1994]. To address this problem within the constraints of the present dataset, the analyses were repeated within the 3 orders of mammals (Rodentia, Chiroptera, and Carnivora) for which several species were represented. This at least allows us to determine the extent to which any relationship among mammals appears simply because it is present in only one of the orders with large numbers of data points. It is important to note here

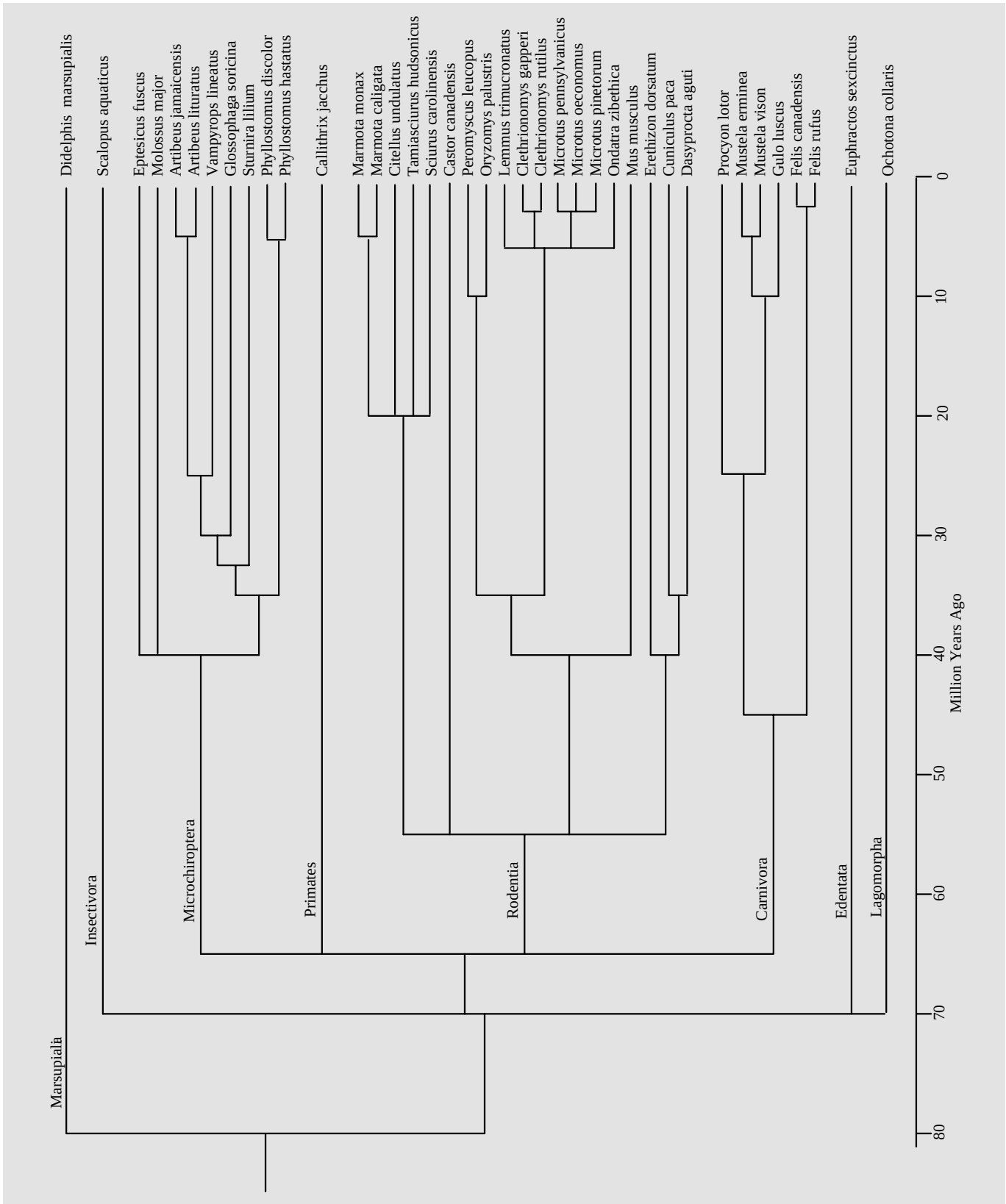

Fig. 2. Phylogeny used for the independent comparison analysis in this study.

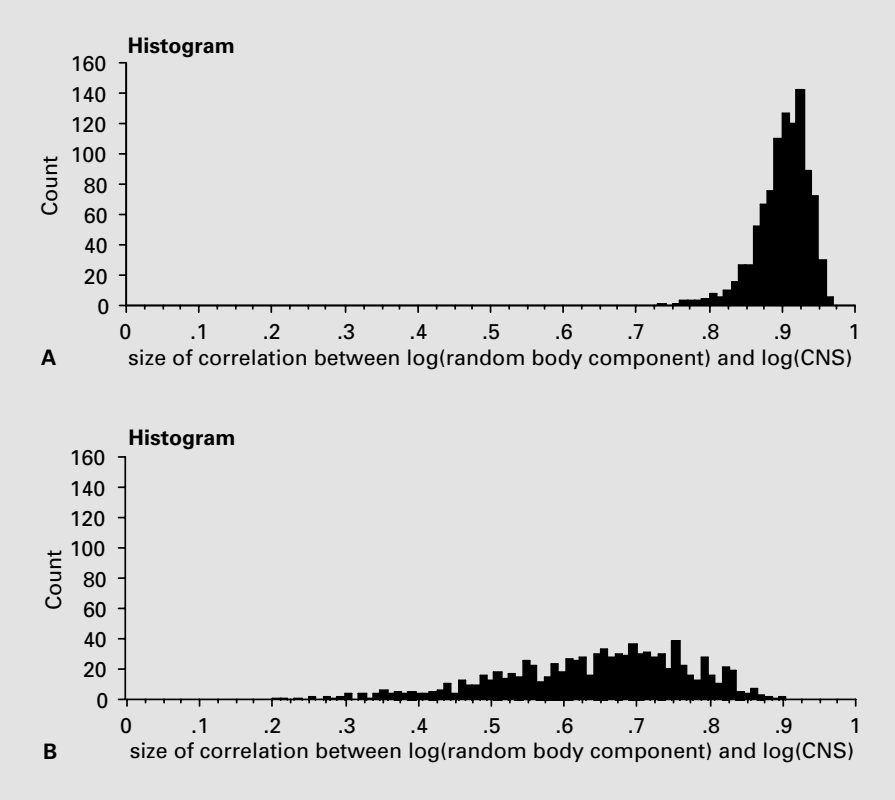

Fig. 3. Histogram of correlations between log CNS and 1000 dummy body component variables (see text for details). **A** Raw correlations. **B** Independent contrast correlations. These are the range of correlations one obtains between randomly-constructed variables and log CNS. In order for correlations between real body components and CNS to be considered statistically significant at the p = 0.05 level, they would need to be as high or higher than the top 5% of these distributions.

that these sorts of phylogenetic controls do not constitute independent confirmations of the hypothesis. They simply assess the extent to which the relationships found in this data set can be explained by phylogeny.

Data Analysis

To determine whether there are significant differences in the strength of associations with CNS of different body components, the data were analyzed in several different ways. First, correlations of CNS with WT, FFWT, FAT, MUSCLE, HEART and BONE were calculated for both log-transformations of the raw data and independent contrasts. Second, multiple regression was used to estimate the extent to which each variable was associated with CNS independent of the others. It is important to keep in mind, however, that if a variable does not show a significant multiple regression coefficient, this does not demonstrate that the variable is functionally unrelated to CNS. It could be that several of the variables correlate with CNS for similar underlying reasons, and if so their *independent* contributions to CNS could be small and insignificant. Nevertheless, it is of interest to know if any variables do have significant independent contributions to CNS. Two multiple regressions were calculated: one using all the variables that are independent components of body mass (i.e., FAT, MUSCLE, HEART, and BONE), as well as using just FAT and FFWT. These were performed for both the log-transformed raw data and the independent-constrast data. Regression through the origin was used for all independent contrast analyses.

Results

Figures 4A and B plot independent-contrast data for both log CNS vs. log FFWT (fig. 4A) as well as log CNS vs. log FAT (fig. 4B). It is apparent from these that CNS is much more tightly associated with FFWT than with FAT. Table 2 lists the correlations for all the body components in this study derived from the raw log-transformed data (without phylogenetic controls) and the independent-contrast data. FFWT is not significantly different from WT with respect to the strength of its association with CNS, either for the raw correlations or the independent contrast correlations. This suggests that these two measures would not show large differences overall in estimates of mammal EQ's (but see further discussion below). This might be due in part to the fact that FFWT makes up such a large proportion of WT, but it cannot be the whole explanation because both MUSCLE and BONE (which of course are even smaller components of WT than is FFWT) correlate just as strongly with CNS as FFWT and WT. The strength of the correlation between CNS and a given sub-component of body mass is not simply a question of how large a proportion it is of WT.

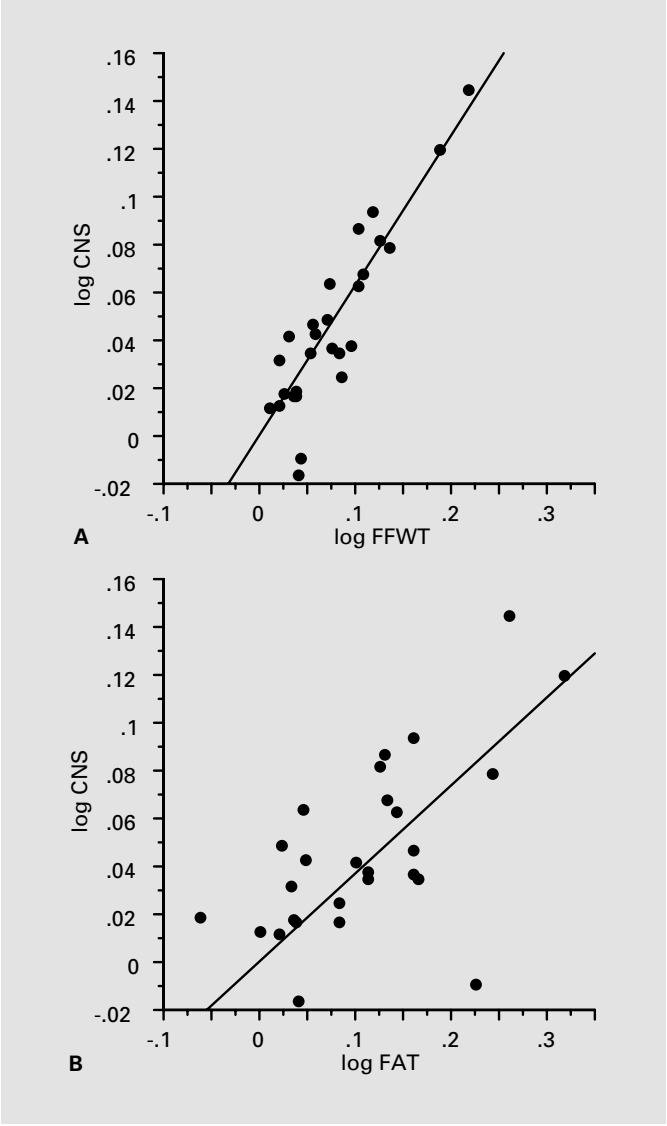

Fig. 4. Least-squares regression between independent contrasts for log CNS and both: **A** log FFWT (log CNS = 0.629(log FFWT); r^2 = 0.923), and **B** log FAT (log CNS = 0.368(log FAT); r^2 = 0.736).

FAT shows the lowest correlations of any of the variables with CNS (independent contrasts: r = 0.858, p < 0.021; raw correlations: r = 0.942, p < 0.092). Although these correlations are not small, it is important to note that FAT correlates with FFWT even more strongly than it does with CNS (independent contrasts correlations between FAT and FFWT: r = 0.912, p < 0.003; raw correlations: r = 0.970, p < 0.001). This pattern shows that it is not simply that FAT has relatively lower correlations with all body components, but rather that it selectively shows lower correlations specifically with CNS. This raises the possibility that FAT correlates with CNS only because both are correlated strongly with FFWT. This suspicion is confirmed by the multiple regression analyses performed using FFWT and FAT to predict CNS. Table 3 contains the coefficients of this analysis for both the raw values and independent contrast values. In both cases, FAT does not make a significant contribution to predicting CNS values independent of FFWT. The standardized coefficients indicate that a change in 1 standard deviation in FFWT corresponds to almost an equivalent change in CNS (1.142 standard deviations for the raw values, 0.944 standard deviations for independent contrasts) after controlling for FAT. Additionally, although not statistically significant, the coefficients for FAT are negative.

Table 4 shows multiple regression coefficients for predicting CNS from FAT and subcomponents of FFWT (i.e., MUSCLE, HEART, and BONE). For both the raw values and independent contrast values, MUSCLE shows the largest independent contribution, whereas FAT does not show a significant independent contribution in either case. The independent contribution of HEART just barely misses significance at the $p < 0.05$ level for raw values, whereas BONE shows a significant independent contribution using independent contrasts. Even given a high degree of multicolinearity among variables, the pattern of FAT being the poorest predictor and MUSCLE being the best is consistent.

When calculated separately within both the winter-collected (Brazilian and Virginian) and summer-collected (Alaskan) sub-samples, the patterns mirror those found for the whole dataset, with FFWT consistently showing higher correlations with CNS than does FAT, and multiple regression coefficients (table 5) showing large inde-

Table 2. Correlations with log CNS using both raw correlations and independent contrast correlations

Variable	Raw correlations	p of being a random component	independent comparisons correlations	p of being a random component
log WT	0.980	<0.001	0.961	<0.001
log FFWT	0.981	<0.001	0.961	<0.001
log MUSCLE	0.984	<0.001	0.959	<0.001
log HEART	0.981	<0.001	0.927	<0.001
log BONE	0.978	<0.001	0.960	<0.001
log FAT	0.942	0.092	0.858	<0.021

Probabilities are determined by comparison to the distribution of correlations obtained with 1,000 dummy variables (see text for details).

Table 3. Multiple regression coefficients predicting log CNS from log FAT and log FFWT

	Coefficient	Std. error	Std. coeff.	t value	p value
Raw values					
log FFWT	0.910	0.104	1.142	8.768	<0.0001
log FAT	−0.106	0.083	−0.166	−1.276	0.2101
Intercept	−1.379	0.153	−1.379	−9.012	<0.0001
Independent contrast values					
log FFWT	0.694	0.088	0.944	7.913	<0.0001
log FAT	−0.047	0.057	−0.109	−0.814	0.4235

Table 4. Multiple regression coefficients predicting log CNS from log MUSCLE, log HEART, log BONE, and log FAT

	Coefficient	Std. error	Std. coeff.	t value	p value
Raw values					
log MUSCLE	0.693	0.249	0.892	2.782	0.0087
log HEART	0.430	0.213	0.478	2.017	0.0517
log BONE	−0.167	0.253	−0.208	−0.660	0.5138
log FAT	−0.115	0.075	−0.181	−1.534	0.1342
Independent contrast values					
log MUSCLE	0.608	0.226	0.843	2.691	0.0130
log HEART	−0.324	0.198	−0.447	−1.640	0.1147
log BONE	0.287	0.122	0.431	2.341	0.0283
log FAT	0.018	0.047	0.042	0.383	0.7049

Table 5. Multiple regression coefficients predicting log CNS from log FAT and log FFWT calculated separately for species collected in Summer vs. Winter

	Coefficient	Std. error	Std. coeff.	t value	p value
Raw values					
Summer (n = 11)					
log FFWT	1.211	0.249	1.495	4.867	0.0012
log FAT	−0.309	0.181	−0.523	−1.703	0.127
Intercept	−1.889	0.419	−1.889	−4.510	0.002
Winter (n = 27)					
log FFWT	0.797	0.137	1.003	5.814	<0.0001
log FAT	−0.015	0.113	−0.023	−0.131	0.8971
Intercept	−1.224	0.191	−1.224	−6.397	<0.0001
Independent contrast values					
Summer (n = 7)					
log FFWT	0.881	0.415	1.113	2.125	0.0869
log FAT	−0.081	0.316	−0.131	−0.256	0.8082
Winter (n = 19)					
log FFWT	0.613	0.110	0.851	5.561	<0.0001
log FAT	0.013	0.074	0.033	0.177	0.8619

Table 6. Multiple regression coefficients predicting log CNS from log FAT and log FFWT calculated within orders of mammals

	Coefficient	Std. error	Std. coeff.	t value	p value
Raw values					
Carnivora (n = 6)					
log FFWT	0.606	0.303	0.843	2.002	0.1391
log FAT	0.07	0.198	0.148	0.351	0.7491
Intercept	−0.603	0.588	−0.603	−1.026	0.3805
Chiroptera (n = 9)					
log FFWT	0.793	0.323	1.016	2.459	0.0492
log FAT	−0.054	0.217	−0.103	−0.250	0.8108
Intercept	−1.211	0.402	−1.211	−3.012	0.0236
Rodentia (n = 19)					
log FFWT	0.895	0.127	1.173	7.076	<0.0001
log FAT	−0.109	0.093	−0.194	−1.168	0.2600
Intercept	−1.394	0.200	−1.394	−6.965	<0.0001
Independent contrast values					
Carnivora (n = 5)					
log FFWT	0.709	0.163	1.171	4.351	0.0224
log FAT	−0.039	0.108	−0.084	−0.363	0.7404
Chiroptera (n = 7)					
log FFWT	0.448	0.154	0.738	2.919	0.0331
log FAT	0.129	0.097	0.348	1.332	0.2402
Rodentia (n = 11)					
log FFWT	0.722	0.146	0.672	4.958	0.0008
log FAT	−0.113	0.089	−0.287	−1.272	0.2352

pendent associations of CNS with FFWT and only small and non-significant independent associations with FAT (although in the independent contrasts for the Summer sub-sample, for which there were only 7 contrasts possible, the independent association of FFWT with CNS did not quite reach statistical significance: $p = 0.0869$).

For completeness, multiple regressions predicting CNS from FFWT and FAT were carried out separately within each of the geographic sub-samples of the winter-collected species (those collected from Brazil vs. those from Virginia). The same patterns were once again found, with FFWT, but not FAT, showing significant independent associations with CNS (in the interests of space, these values are not reported here). Thus, it would appear that these patterns are broadly found, and are not solely explained by either geographic location nor seasonality of collection.

Lastly, table 6 shows the multiple regressions predicting CNS from FFWT and FAT carried out separately within each of the orders of mammals represented by more than one species (Carnivora, Chiroptera, and Rodentia). In none of these orders does FAT show a statistically significant independent contribution to predicting CNS, and in only one case (Carnivora using raw values; N = 6) does FFWT fail to reach significance. Thus, the pattern shown across the whole dataset cannot easily be attributed to its appearance in only one order. This does not constitute proof that the pattern holds within the other mammalian orders, however.

Discussion

The fact that dummy sub-component variables representing random percentages of weight (WT) have such high correlations (for either log-transformed raw or independent contrast data) illustrates how important it is to think carefully about the statistical assumptions one makes in comparative studies when using variables that are sub-components of a third variable that differs so greatly across species. The huge discrepancy between what the present randomization study suggested was statistically significant for this data ($r \geq 0.837$ for independent contrast data with $n = 27$) versus what would be considered statistically significant under standard assumptions ($r \geq 0.331$) shows that this can be a very important consideration. Future comparative studies investigating associations between variables that are sub-components of weight need to take this fact into account [e.g., the 'ex-

pensive tissue hypothesis'; Aiello and Wheeler, 1995; Aiello and Bates, 1998].

The finding that total fat (FAT) is less strongly associated with the weight of the central nervous system (CNS) than are fat-free weight (FFWT), muscle weight (MUSCLE), heart weight (HEART), and bone weight (BONE) has never been empirically demonstrated before with any data set, although it is consistent with what we would expect given that fat is not highly innervated. FFWT is only slightly (and non-significantly) more highly correlated with CNS overall. However, given that FAT does not appear to have an independent association with CNS, whereas FFWT does, and because by definition FAT + FFWT (+ CNS) = WT, it would appear that WT does not deserve equal theoretical status with FFWT with respect to CNS scaling.

The fact that these patterns hold within seasonality/location subgroups suggests that these variables do not entirely explain the results here. However, given the conflation of seasonality and location in this sample, the possibility that seasonality within a given geographical locale might swamp the effects, or that seasonality or location might both have important effects on the associations, cannot be addressed with this data.

To what extent are the differences between FAT and FFWT (with respect to the strength of their associations with CNS) due to FAT being inherently more variable (and having larger measurement error) than FFWT? To what extent can we be confident that FAT values used in this study are truly representative of their respective species? It would appear that species really do vary with respect to relative fattiness. The possibility that FAT might be more difficult to measure accurately than other components seems unlikely given that the measurement of fat was done chemically by ether extraction [see Pitts and Bullard, 1968], rather than by blunt dissection (as was done with, e.g., muscle mass) which would appear to be an inherently less precise method. There was some indication in the present dataset that FAT might show slight seasonality (though, again, this was conflated with location), and certainly species which hibernate generally tend to put on fat for the winter months. However, whereas % FAT is significantly associated with log WT in this dataset ($r^2 = 0.405$, $p > 0.01$, $n = 18$ independent comparisons), it is not significantly associated with seasonality/location. This indicates that WT explains more variability in fattiness than the seasonality/location variable. Furthermore, the pattern of associations among FFWT, FAT, and CNS remained the same within seasonality/location sub-samples, which suggests that seasonality does not entirely explain the patterns found (even if seasonality cannot be ruled out completely given limitations in the dataset). If fat is particularly variable across individuals within a species, then sampling error might affect FAT more than other components. It is true that humans can gain and lose fat reasonably rapidly without presumably changing their brain size, but this is also true of lean body mass (e.g., lifting weights adds muscle mass fairly effectively). Pitts and Bullard report standard deviations for different body components for 11 species with large enough sample sizes, and these suggest that FAT is relatively more variable within species than FFWT (the within-species coefficient of variation for FAT averages 43.6%, compared to 17.9% for FFWT + CNS). However, if within-species individual variation in FAT lead to significant sampling error in estimating species FAT in this sample, this should be more of a problem for species represented by few individuals than for species represented by many. There should be a negative correlation between (1) the number of individuals sampled in a given species and (2) the absolute value of residuals of FAT on WT (which represents the degree to which a species FAT differs from the average value for a species of that WT). For the present dataset, this association is very small and statistically non-significant ($r = 0.064$, $p = 0.70$). For all these reasons, the lack of association between FAT and CNS (independent of FFWT) would appear to be genuine.

As was mentioned in the introduction, although correlation does not prove direct causation we are justified in asking what causal influences might explain the associations that are apparent. We cannot begin to unravel the web of influences unless we consider how the current correlations might fit into some causal picture, just as Jerison's [1973], Martin's [1981], and Armstrong's [1983], hypotheses spurred further refinement of this question.

Assuming that the present findings can be generalized beyond this data set, they are not in conflict with the hypothesis [Martin, 1981; Armstrong, 1983] that metabolic rate is the ultimate evolutionary cause of the brain ↔ body weight scaling relationship. They do suggest additional explanations, however. Within humans at least, metabolic rate is more highly correlated with FFWT than it is with WT [Ravussin et al., 1982; Halliday et al., 1983; Jensen et al., 1988; Owen, 1988; Salomon et al., 1992]. There is apparently no comparative analysis of this question, but assuming this pattern holds across species it suggests that metabolic rate might be related to brain size not in a direct causal sense (as in Martin, 1981), but only as a consequence of both of these being causally related to FFWT. In this model, greater FFWT would allow for –

but not require – greater brain mass, and would at the same time directly cause an increase in total metabolic rate. This would be consistent with the finding that neonatal brain size is not related to basal metabolic rate when differences in adult WT are taken into account [Pagel and Harvey, 1988], although variation in gestation length may also allow species to adjust brain size even if maternal energy budgets are a key constraint [Martin, 1981, 1995; Martin and MacLarnon, 1985]. It is important to recognize that body weight does not perfectly predict brain weight in mammals. The r = ~0.95 correlation typically found in fact corresponds to ~10-fold variation in brain weight for a given body weight [Martin, 1981; Schoenemann, 2001], which means that a variety of other influences are at play.

If the confidence intervals on the interspecies relationships between brain size and WT were small enough to definitely exclude either the prediction of body surface area (e.g., slope of 0.67) or metabolic rate (slope of 0.75), we might be in a better position to choose between these competing hypotheses. Earlier analyses [e.g., Martin, 1981] apparently excluded the body surface area hypothesis on this very basis, but the current best estimate is actually closer to that predicted by the body surface area hypothesis [Harvey and Krebs, 1990]. However, it is likely that a number of influences are at work here, such that we cannot use the value of the slope by itself to determine causality [Deacon, 1990].

In any case, it is important to stress that for both the metabolic rate and body surface area hypotheses, the direction of the causal arrows explaining evolutionary changes in brain size could point in either direction. Brain size might be caused by evolutionary changes in metabolic rate and/or body surface area, but there is no clear theoretical expectation that this *must* be the case. It is equally possible, a priori, that evolutionary changes in brain size cause changes in metabolic rate and/or body surface area. That is, the selection might always (or predominantly) be operating on brain size for behavioral reasons (e.g., behavioral complexity, flexibility, or whatever), but that changes in brain size require (in the case of increases) or allow (in the case of decreases) changes in metabolic rate and/or body surface area [see also Schoenemann, 2001]. A correlation would be apparent between these other physiological variables and brain size, but not because they are causing brain size variation. A larger body size might be required to support a larger brain, perhaps because of the larger total metabolic resources available to an animal with a larger body, but brain size could still be under the most direct selection. Given that brain tissue is very metabolically expensive, if an increase in brain size was selected for, it is reasonable to suppose that overall metabolic rate would be affected. Unfortunately nothing in this study, nor in other studies so far published, can demonstrate which way the causal arrows point. This is obviously an important question for future research.

Implications for Explaining Differences in Brain Size

With respect to comparative studies of brain size scaling, WT is such a good predictor of FFWT that the slope of the regression relating CNS and FFWT is only slightly steeper than the slope relating CNS and WT (0.629 vs. 0.619, respectively, for this dataset). Because, as was pointed out above, there are a priori reasons to suppose that FFWT might share a particularly close association with CNS, and because FFWT but not FAT (the other component that sums to WT) appears empirically to be associated with CNS, estimating encephalization quotients [EQ's; Jerison, 1973] should probably more properly be done using FFWT. However, because FFWT is much more difficult to estimate than WT, and because the differences in EQ's are likely to be small between the two methods, this is impractical even if theoretically more appropriate. There might nevertheless be subtle but important differences in EQ estimates, which could be significant in some cases given the vast range of FFWT and WT in mammals, and this should be kept in mind when comparing brain sizes across species.

The extent to which this finding might account for the increased Neanderthal cranial capacities, which average 157 cm^3 greater than modern humans [Wood and Collard, 1999], is an interesting question. Barring extensive primate data on body composition and brain size, any estimates will obviously have to be considered tentative, and serve only to give some idea of the potential size of the effect. Using the least-squares regression of brain weight on body weight for Stephan et al.'s [1981] primate data, one can calculate the average (expected) difference in brain weight for two primates weighing the same as Neanderthal and modern humans and use this to estimate cranial capacity differences [cranial capacity is ~14% larger than brain volume; Count, 1947; Hartwig-Scherer, 1993; Kappelman, 1996]. Depending on what values we use for Neanderthal and modern human WT [Wilmore and Behnke, 1969, 1970; Wood and Collard, 1999], between 30–80% of the absolute difference in cranial capacity can be explained by WT alone. However, if Neanderthals had lower percent fat than modern humans [as implied by arguments in Dubois, 1921; Trinkaus and Howells, 1979; Holloway, 1981, 1985], their WT differ-

ence would not be as large as their FFWT difference. It does appear that modern humans tend towards the high end of percent fat among mammals [the regression in figure 1 predicts modern humans should be ~15% fat – although the 95% confidence intervals range from 10 to 20% – whereas humans actually average more than 20%, Wilmore and Behnke, 1969, 1970]. If Neanderthal averaged only 15% fat, their FFWT would be ~4 kg larger than expected for their WT; if they averaged 10% fat, their FFWT would be ~8 kg larger than expected. Adding these additional FFWT values to the Neanderthal calculations suggest that perhaps an additional 20–25% of the actual difference in cranial capacity might be explained beyond WT differences alone, and that somewhere between 60 and 100% of the total cranial capacity difference might be explained this way. It cannot, however, be emphasized enough that these estimates are subject to large possible errors, and simply serve to give some idea of the possible magnitude of the effect. Note also that Neanderthals might simply have large brains because they are cold-adapted [populations in colder regions have larger cranial volumes – and presumably larger brains; Beals et al., 1984], or the specimens that have been found may be a biased sample.

It is not clear how much of the sex difference in brain weight [~140 g in one European-derived sample; Ho et al., 1980] might be explained by the substantially larger FFWT difference between the sexes (~15 kg) than WT differences [Wilmore and Behnke, 1969, 1970]. Ankney [1992] showed that about 16% of the brain weight difference is explained by WT differences [see also Falk et al., 1999]. Estimating from primate scaling trends as in the Neanderthal case, the FFWT difference might explain another ~16%. However, because no study reports FFWT and brain size estimates for the same subjects, this estimate is little more than a guess, even though the sex difference in FFWT would appear to be relevant.

In conclusion, FFWT is strongly correlated with CNS independent of FAT, but the converse is not true. This relationship does not appear to be affected by sample characteristics such as different numbers of males vs. females, location or season of collection, or phylogenetic effects. Subcomponents of FFWT: MUSCLE, HEART, and BONE all show higher associations with CNS than does FAT. Because FFWT + FAT = WT, it would appear that the relationship between brain size and weight is actually the result of brain size scaling with FFWT. This analysis substantiates long-standing assumptions about brain scaling that have in fact never been tested. This might explain a portion of the Neanderthal – modern human difference in brain size, as well as a portion of the sex differences in brain size.

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