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The Expensive-Tissue Hypothesis

The Brain and the Digestive System in Human and Primate Evolution[1]

by Leslie C. Aiello and Peter Wheeler

Brain tissue is metabolically expensive, but there is no significant correlation between relative basal metabolic rate and relative brain size in humans and other encephalized mammals. The expensive-tissue hypothesis suggests that the metabolic requirements of relatively large brains are offset by a corresponding reduction of the gut. The splanchnic organs (liver and gastrointestinal tract) are as metabolically expensive as brains, and the gut is the only one of the metabolically expensive organs in the human body that is markedly small in relation to body size. Gut size is highly correlated with diet, and relatively small guts are compatible only with high-quality, easy-to-digest food. The often-cited relationship between diet and relative brain size is more properly viewed as a relationship between relative brain size and relative gut size, the latter being determined by dietary quality. No matter what is selecting for relatively large brains in humans and other primates, they cannot be achieved without a shift to a high-quality diet unless there is a rise in the metabolic rate. Therefore the incorporation of increasingly greater amounts of animal products into the diet was essential in the evolution of the large human brain.

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Much of the work that has been done on encephalization in humans and other primates has been oriented toward why questions—why different primate taxa have different relative brain sizes or why the human line has undergone such a phenomenal increase in brain size during the past 2 million years. Hypotheses that have been put forward to answer these questions primarily invoke socio-ecological factors such as group size (Aiello and Dunbar 1993), social (or Machiavellian) intelligence (Byrne and Whiten 1988), or complexity of foraging strategy (Milton 1979, Parker and Gibson 1979, Clutton-Brock and Harvey 1980, Gibson 1986, MacNab and Eisenberg 1989). These questions and their answers are undoubtedly important for an understanding of encephalization, but there are other issues that must be taken into consideration. Brains are metabolically very expensive organs, and large brains have specific chemical and thermoregulatory requirements (Wheeler 1984, Falk 1990). One of the most interesting questions is how encephalized primates, and particularly humans, can afford such large brains (Martin 1983, Foley and Lee 1991).

Relatively few studies have been oriented toward this question of cost. Those that have suggest a relationship between dietary quality and relative brain size, mediated either through the brain's chemical requirements and specifically long-chain fatty acids (Crawford 1992) or through basal metabolic rate (BMR), reflecting the energy needed for brain growth and maintenance (Martin 1981, 1983; Armstrong 1982, 1983, 1985*a*, *b*, 1990; Hofman 1983). Through the analysis of the metabolic requirements of various organs in the body, we suggest the "expensive-tissue hypothesis" to explain how encephalized primates can have relatively large brains without correspondingly high basal metabolic rates.

1. We thank David Chivers and Ann MacLarnon for supplying unpublished gut weights and for drawing our attention to inaccuracies in the published primate gut weights in Chivers and Hladik (1980: table 6). We are grateful to the following people for reading earlier versions of this manuscript and/or discussing with us the ideas presented in it: Peter Andrews, Robert Barton, Robin Dunbar, Rob Foley, Maciej Henneberg, Katherine Homewood, Harry Jerison, Cathy Key, Ruth Mace, Katherine Milton, Sara Randall, Margaret Schoeninger, Francis Thackeray, Alan Walker, and Bernard Wood. We thank John Fleagle for making us aware that Sir Arthur Keith had pointed out the inverse relationship between brain size and stomach size in primates in 1891 (Keith 1891) and had lamented the fact that the findings in this obscure paper had gone, to his knowledge, entirely uncited (Keith 1950). An oral version of this paper was presented at the 1993 Paleoanthropology society meetings in Toronto, Canada.

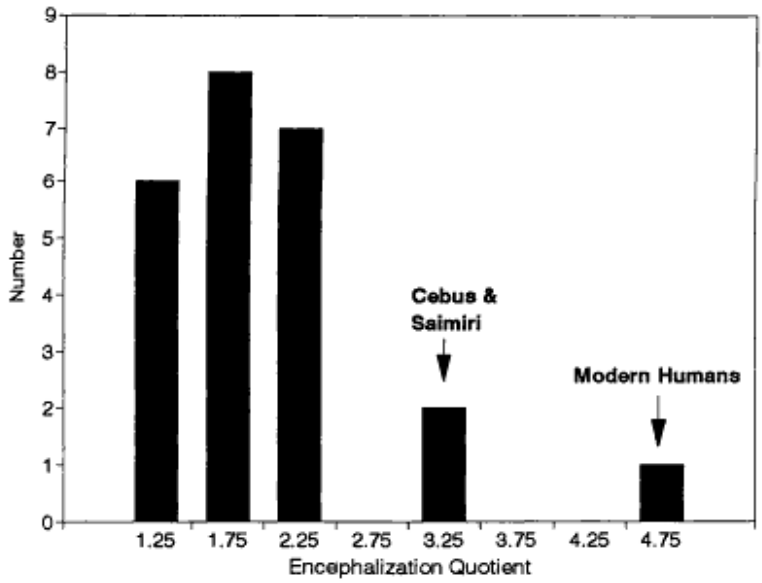

FIG. 1. *Encephalization quotients (genus averages) for humans and other primates* (n = 24) *(human body mass = 65 kg; brain mass = 1,300 g; other data from Aiello and Dean 1990).*

This hypothesis also provides an explanation for the apparent correlation between encephalization in the early hominids and the incorporation of increasingly large amounts of animal-derived food into the diet.

The Problem

Three factors combine to pose a major problem for the understanding of how encephalized primates, and particularly humans, can afford their relatively large brains. The first is encephalization itself. By definition, an encephalized primate has a larger-than-expected brain in relation to its body size. One of the most commonly used equations for the prediction of brain size for placental mammals (Martin 1983, 1990) is

$$\log_{10} E = 0.76 \log_{10} P + 1.77, \qquad (1)$$

where E is brain mass in milligrams and P is body mass in grams. In terms of this equation, modern humans have an encephalization quotient (ratio of observed to expected brain size [EQ]) of 4.6 while other primates average 1.9 ± 0.6 (fig. 1). This means that the average human has a brain that is 4.6 times the size expected for the average mammal and the average non-human primate anthropoid has a brain almost twice as large as that of the average mammal.

The second factor is the metabolic cost of the brain. On the basis of *in vivo* determinations, the mass-specific metabolic rate of the brain is approximately 11.2 $W.Kg^{-1}$ (watts per kilogram) (table 1; Aschoff, Günther, and Kramer 1971). This is nine times higher than the average mass-specific metabolic rate of the human body as a whole (1.25 $W.Kg^{-1}$). The majority of this high level of energetic expenditure, which is comparable to *in vivo* measurements of brain tissue from other mammalian species, appears to be associated with the ion pumping necessary to maintain the potentials across the axonal membranes. In addition, energy is used in the continual synthesis of neurotransmitters such as acetylcholine. Consequently, a large-brained mammal must be capable of continually supplying the brain with the high levels of substrate and oxygen required to fuel this expenditure, a task made more difficult by the inability of the brain to store significant energy reserves.

There is no doubt that any increase in brain tissue would represent a considerable energetic investment for the animal concerned. For example, according to equation 1, the average (65-kg) human has a brain 1.04 kg larger than would be expected for the average mammal of the same body mass (observed brain mass = 1,300 g; expected brain mass = 268 g) and 0.85 larger than would be expected for the average primate of the same body mass. Assuming for the moment that the metabolic cost of 11.2 $W.Kg^{-1}$ is constant for brain tissue in all mammals of comparable body mass, the inferred BMR for the expected brain mass in the average mammal of human body mass would be 3 watts. The observed BMR for the observed, much larger brain mass in humans is 14.6 watts.

Because the human brain costs so much more in energetic terms than the equivalent average mammalian brain, one might expect the human BMR to be correspondingly elevated. However, there is no significant

TABLE 1
Organ Mass and Metabolic Rate in Humans

Organ	Organ Mass (kg)	% Body Mass	Mass-Specific Organ Metabolic Rate ($W.Kg^{-1}$)	Total Organ Metabolic Rate (W)	% Total Body BMR
Brain	1.3	2.0	11.2	14.6	16.1
Heart	0.3	0.5	32.3	9.7	10.7
Kidney	0.3	0.5	23.3	7.0	7.7
Liver	1.4	2.2	12.2	17.1	18.9
Gastro-intestinal tract	1.1	1.7		13.4	14.8
Total	4.4	6.8		61.7	68.1
Skeletal muscle	27.0	41.5	0.5	13.5	14.9
Lung	0.6	0.9	6.7	4.0	4.4
Skin	5.0	7.7	0.3	1.5	1.7
Grand total	37.0	56.9		80.8	89.1

NOTE: Data for a 65-kg male with a BMR of 90.6 W (Aschoff, Günther, and Kramer 1971).

correlation between relative basal metabolic rate and relative brain size in humans and other encephalized animals (McNab and Eisenberg 1989). Mammalian BMRs are allometrically related to body mass by an equation of the form

$$\text{BMR (W)} = a \cdot \text{mass (kg)}^{0.75}. \quad (2)$$

Most interspecific studies have reported exponent values very close to 0.75, and this is generally accepted as the standard exponent for comparisons of species of differing body mass (Blaxter 1989, Bligh and Johnson 1973, Kleiber 1961, Schmidt-Nielsen 1984). Such analyses have produced similar estimates for the metabolic level (a in equation 2) that range from 3.3 to 4.1 (Blaxter 1989). One of the most widely used general relationships for mature placental mammals (eutherians) is that calculated by Kleiber (1961),

$$\text{BMR (W)} = 3.39 \cdot \text{mass (kg)}^{0.75}. \quad (3)$$

There is, however, considerable variation between taxonomic groups (Blaxter 1989, Huyssen and Lacy 1985, Peters 1983). For example, the reported metabolic levels of some insectivores (Blaxter 1989, Wheeler 1984) and mustelid carnivores (weasels) (Iversen 1972, Wheeler 1984) are as high as 9.5 and 7.5, respectively. In contrast, those of some chiropterans (bats) are as low as 2.0 to 2.5 (Poczopko 1971, Wheeler 1984). Although some eutherian taxa do therefore deviate markedly from the Kleiber relationship, this is not the case for primates, which, with a metabolic level of 3.36 (Blaxter 1989), display BMRs almost identical to those predicted by the Kleiber equation and other general relationships for eutherian mammals.

Far more experimental determinations have been made of human basal metabolism than for any other mammal (C. Schofield 1985). The extensive data available clearly demonstrate that, although influenced by factors such as age and sex (W. N. Schofield 1985), the BMRs of mature individuals are typical of primates and consequently eutherian mammals as a whole (table 2). In fact, the mean BMRs of mature men and women straddle the values predicted by both primate and eutherian equations for mammals of comparable body mass (fig. 2). Consequently, there is no evidence of an increase in basal metabolism sufficient to account for the additional metabolic expenditure of the enlarged brain. Where does the energy come from to fuel the encephalized brain?

The Solution

One possible answer to the cost question is that the increased energetic demands of a larger brain are compensated for by a reduction in the mass-specific metabolic rates of other tissues. For example, if a significant component of BMR is endogenous heat production specifically related to the thermoregulatory demands of the mammal, then any increased contribution made by brain metabolism to its thermal budget could allow a corresponding reduction in the requirement for dedicated thermogenesis elsewhere in the body.

An alternative and not necessarily contradictory possibility is that the expansion of the brain was associated with a compensatory reduction in the relative mass of one or more of the other metabolically active organs of the body. Although most studies of primate metabolism have focused on the energetic costs of encephalization, the brain is just one of several organs with high energetic demands. The heart, kidneys, and splanchnic organs (liver and gastro-intestinal tract) also make a substantial contribution to overall BMR (table 1). Determinations

TABLE 2
Observed and Predicted Basal Metabolic Rates for a 65-kg Human Compared with Other Primates and Eutherians

		Predicted 65-kg Mammal BMR			
		Other Primates ($3.36\ M^{0.75}$)		Eutherians ($3.39\ M^{0.75}$)	
Sex and Age	BMR (W)	W	Diff. (%)	W	Diff. (%)
Male					
18–30 yrs	80.914	76.916	+5.20	77.603	+4.25
30–60 yrs	78.391	76.916	+1.92	77.603	+1.02
Female					
18–30 yrs	70.208	76.916	−8.72	77.603	−9.53
30–60 yrs	66.528	76.916	−13.51	77.603	−14.27
All					
18–30 yrs	75.561	76.916	−1.76	77.603	−2.63
30–60 yrs	72.460	76.916	−5.79	77.603	−6.63

SOURCE: For humans, W. Schofield (1985).

of the oxygen consumption rates of these organs *in vivo* by perfusion experiments indicate that, together with the brain, they account for 60–70% of BMR despite making up less than 7% of total body mass. The heart and kidneys have mass-specific metabolic rates considerably higher than that of the brain, the energetic demands of which are comparable to those of the splanchnic tissues. The tissues which make up the remaining 93% of body mass display correspondingly low rates of energy turnover. For example, the *in vivo* mass-specific metabolic rate of resting human skeletal muscle is only about 5% of that of the brain, and consequently, although this tissue accounts for 41.5% of total body mass, it contributes only 14.9% of BMR on the basis of the data used here (table 1).

These differences in the contribution of various tissues to BMR are also reflected by measurements of the oxygen consumption rates of isolated tissues (table 3). Such *in vitro* determinations are known to be influenced by factors such as the mode of preparation of the tissues and the chemical composition of the suspending media, and therefore care is necessary in comparing the abso-

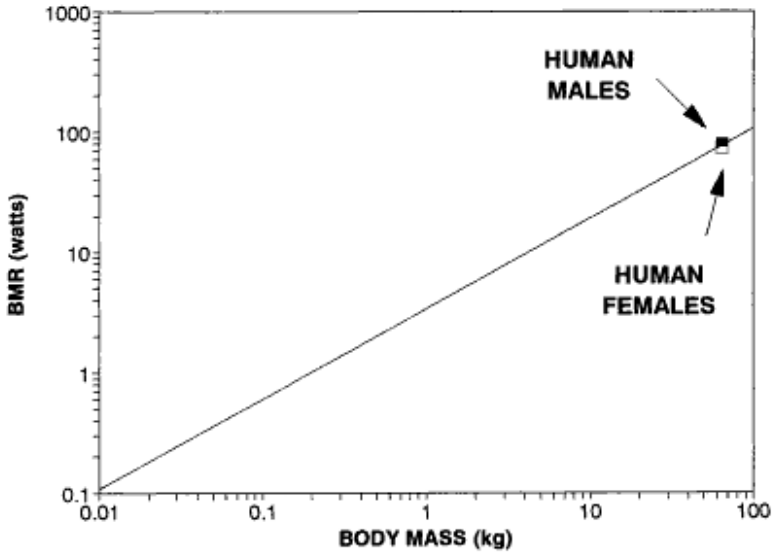

FIG 2. *Basal metabolic rate and body mass, showing that 65-kg human males and females (18–30 years old) span the best-fit line for all mammals (equation 3).*

TABLE 3
In vitro *Tissue Mass-specific Metabolic Rates*

Tissue	Mass-specific Metabolic Rate (W.Kg^{-1})			
	Mouse (13 g)	Rat (150 g)	Rat (242 g)	Dog (19.1 kg)
Brain	14.0	10.3	13.1	7.6
Heart	16.4 (117)	10.8 (105)	16.6 (126)	5.9 (78)
Kidney	22.0 (157)	23.0 (224)	24.9 (190)	13.8 (182)
Liver	16.3 (116)	11.2 (109)	11.6 (89)	11.2 (148)
Gastro-intestinal tract	17.4 (124)	5.6 (55)	10.9 (83)	3.8 (50)
Lung	3.8 (27)	7.0 (68)	–	3.0 (39)
Skeletal muscle	3.2 (23)	4.9 (48)	2.2 (17)	3.2 (42)
Skin	–	2.3 (23)	–	1.0 (13)
Bone	–	0.9 (8)	–	0.4 (5)

SOURCES: For mouse, Wheeler (1984); for 150-g rat, Field, Belding, and Martin 1939; for 242-g rat, Wheeler (1984) and unpublished data; for dog, Martin and Fuhrman (1955).
NOTE: Numbers in brackets represent the tissue mass-specific metabolic rate as a percentage of the mass-specific metabolic rate of the brain.

lute values reported by different studies. Also, the absolute metabolic rates of individual tissues of species of differing size cannot be directly compared because these parameters, like BMR itself, are allometrically related to body mass. The limited number of detailed studies conducted generally indicate that the mass-specific metabolic exponents of the different tissues are between 0 and −0.15 (Bertalanffy and Eastwick 1953, Grande 1980, Krebs 1950, Oikawa and Itazowa 1984, Wheeler 1984), and therefore cellular metabolism is less dependent on the size of the mammal than overall BMR, with its mass-specific exponent of around −0.25. However, when the different tissues are compared within a study, the general pattern of their relative metabolic rates is very similar to that observed for humans *in vivo*. As expected, an exception is the heart, which *in vivo* maintains high levels of contractile activity even in the resting mammal, resulting in much higher levels of oxidative metabolism than those measured in isolated cardiac muscle.

Therefore, both *in vivo* and *in vitro* data clearly demonstrate that, together with the brain, the heart, kidney, and splanchnic organs account for the majority of BMR. To determine whether increased encephalization is associated with a reduction in relative size of any of these other metabolically active tissues it is necessary to compare the observed mass of each organ in an adult human with that expected for the average primate of corresponding body mass.

The analysis is based on the organ masses of a 65-kg "standard" human male. Gastro-intestinal tract mass, excluding oesophagus and contents (food and digestive juices), has been estimated to be 1,150 g (Synder 1975). The liver is estimated as the difference between this figure and the splanchnic mass of 2.5 kg given by Aschoff, Günther, and Kramer (1971) and is consistent with other estimates of normal liver size in a "standard" individual (Synder 1975). Organ mass in adult humans varies with age, health, and nutritional status (Synder 1975), but data from complete dissections of individual cadavers (e.g., Mitchell et al. 1945, Forbes, Cooper, and Mitchell 1956) suggest that the general size relationships between organs shown in table 1 are reasonable reflections of the relationships in healthy individuals. It is important to note that this analysis is designed to reveal only general trends in observed size and metabolic relationships of human organs in relation to those that would be expected in the average primate of our body mass. It is not designed, and should not be interpreted, to represent a detailed size or metabolic analysis applicable at the individual level.

The organ masses that would be expected for the average primate of a human body mass (65 kg) were computed for the heart, liver, and kidneys on the basis of the least-squares equations for primates given in Stahl (1965). The correlation coefficients in these relationships are sufficiently high to guard against significant bias attributable to the use of least-squares regression rather than reduced-major-axis analysis (Aiello 1992). Expected brain mass and gut mass were derived from reduced-major-axis equations computed for this analysis. The relevant equations, along with sample sizes, correlation coefficients, and data sources, are given in figure 3.

The combined mass of the metabolically expensive tissues for the reference adult human is remarkably close to that expected for the average 65-kg primate (fig. 3, table 4), but the contributions of individual organs to this total are very different from the expected ones. Although the human heart and kidneys are both close to the size expected for a 65-kg primate, the mass of the splanchnic organs is approximately 900 g less than expected. Almost all of this shortfall is due to a reduction in the gastro-intestinal tract, the total mass of

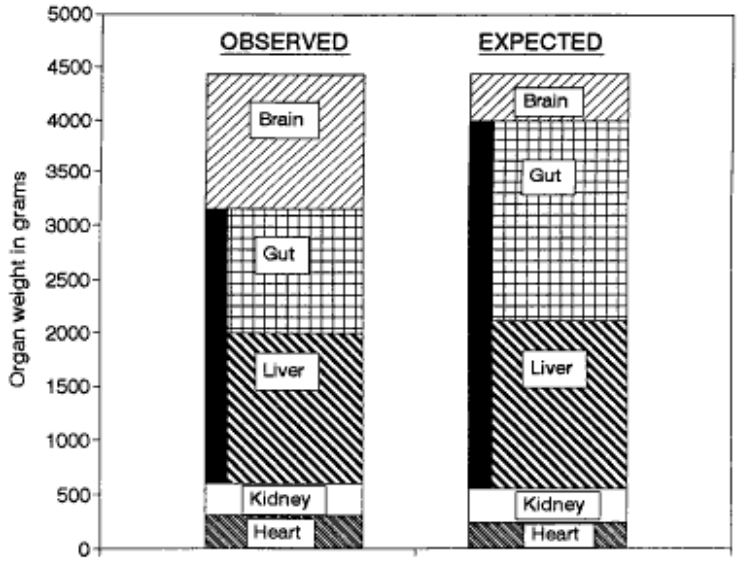

FIG. 3. *Observed and expected organ mass for a "standard" 65-kg human. Expected organ masses for heart, liver, and kidneys from Stahl (1965):* heart mass = $5.2M^{0.987}$ (n = 321, r = 0.99); liver mass = $32.2M^{0.94}$ (n = 293, r = 0.98); kidney mass (both kidneys together) = $6.3M^{0.87}$ (n = 268, r = 0.95). *Expected brain size is based on the reduced-major-axis equation computed for higher primates (excluding humans) from data in Stephan, Frahm, and Baron (1981):* brain mass = $log_{10}Bw = 0.72log_{10}M + 1.35$ (N = 26, r = 0.98). *Expected gut size is based on the reduced-major-axis equation computed for higher primates from data in Chivers and Hladik (1980) and Chivers, personal communication, 1990 (typesetting errors affecting data accuracy in their table 6 have been corrected, and new species have been added);* gut mass, $log_{10}GM = 0.853log_{10}M - 1.271$ (N = 22, r = 0.96). GM, *gut mass (kg);* W, *other-organ mass (g);* M, *body mass (kg);* n, *number of individuals;* N, *number of species;* r, *product-moment correlation coefficient.*

which is only about 60% of that expected for a similar-sized primate. Therefore, the increase in mass of the human brain appears to be balanced by an almost identical reduction in the size of the gastro-intestinal tract.

These relationships are size relationships rather than metabolic relationships. Whether the energetic saving attributable to the smaller gut is sufficient in itself to meet the metabolic demands imposed by the increased encephalization depends on the relative metabolic rates of the two tissues. Although no human data are available relating specifically to the *in vivo* oxygen consumption of the gastro-intestinal tract, the overall metabolic rate of the splanchnic organs is approximately 12.2 $W.Kg^{-1}$ (Aschoff, Günther, and Kramer 1971). If the mass-

TABLE 4
Observed and Expected Organ Metabolic Rates

Tissue	Mass (kg)				Metabolic Cost ($W.Kg^{-1}$)	Metabolic Increment (W)
	Observed	Expected	Observed-Expected			
Brain	1.300	0.450	+0.850		11.2	+9.5
Heart	0.300	0.320	−0.020		32.3	−0.6
Kidney	0.300	0.238	+0.062		23.3	+1.4
Liver	1.400	1.563	−0.163	−0.944	12.2	−11.5
Gastro-intestinal tract	1.100	1.881	−0.781			
Total	4.400	4.452				−1.2

SOURCE: Aschoff, Günther, and Kramer (1971).
NOTE: Expected metabolic rates are computed for a 65-kg human on the basis of the equations given in figure 3.

specific metabolic rates of the liver and smooth muscle of the gut contributing to this are comparable (and *in vitro* determinations of tissues from other mammalian species suggest that this is the case [table 3]), then the reduction in the size of the gut saves approximately 9.5 W. Consequently, the energetic saving attributable to the reduction of the gastro-intestinal tract is approximately the same as the additional cost of the larger brain (table 4). Therefore, if the changes in the proportions of the two organs were contemporary evolutionary events, there is no reason that the BMRs of hominids would ever have been elevated above those typical of other primates as a consequence of the energetic costs of encephalization.

Although this analysis is concerned primarily with the contribution of the metabolically active tissues to BMR, some consideration should be given to the significance of the costs of these organs in the context of the overall energy budget of the animal. Obviously, it is impossible to determine the total daily energy expenditure—the field metabolic rate (FMR)—of earlier hominids, but inferences about the likely levels of energy utilization can be made from measurements of modern humans and other living mammals. Calculations of FMR for 13 species of small mammal, the majority weighing less than 100 g, averaged 2.65 times BMR (Karasov 1992). The ratio is significantly lower in humans, ranging from 1.55 to 2.10 times BMR for individuals undertaking light and heavy occupational work respectively (FAO/WHO/UNU 1985). If the daily energy expenditure of earlier populations of *Homo sapiens* is taken as approximately 1.8 times BMR (the value estimated for subsistence farmers in developing countries today [FAO/WHO/UNU 1985]), then even if the metabolic rates of the brain and gut remain at their basal levels their combined contribution, which represents 31% of BMR, still accounts for a highly significant 17% of total energy requirements.

A significant proportion of FMR is attributable to the cost of activity (Karasov 1992), during which the energy demands of the skeletal musculature increase dramatically but those of the metabolically expensive organs, with the exception of the heart, remain close to their resting levels (Lehninger 1975). Another major component of FMR is an increment of heat production which occurs during the assimilation of nutrients, the summated effect above basal metabolism of which is termed the specific dynamic effect of food. The extent of this increase in energy expenditure depends on both the absolute quantity of food ingested and its composition. For example, for a range of mammalian carnivores, the average daily cost of assimilation has been calculated as approximately 15% of the total ingested metabolisable energy (Karasov 1992), which represents about 40% of BMR. The multiple causes of this substantial increase in energy expenditure are incompletely understood, but contributory factors include additional metabolic activity by the gut itself due to the energetic demands of processes associated with the transport of nutrients (Blaxter 1989). Since determinations of BMR are made specifically with the subject in a postabsorptive state, the rate of energy utilization by the gut will normally be higher than its basal level. Consequently, this organ will be responsible for an even more significant proportion of total energy expenditure than is indicated by its absolute contribution to BMR.

Evolutionary Implications

This analysis implies that there has been a coevolution between brain size and gut size in humans and other primates. The logical conclusion is that no matter what is selecting for brain-size increase, one would expect a corresponding selection for reduction in the relative size of the gut. This would be essential in order to keep the total body BMR at the typical level. If it was necessary for a primate to have a large gut, that primate would also be expected to have a relatively small brain.

This assumes that the primates were not balancing their energy budgets in other ways, such as opting for a relatively high BMR or altering the size and/or metabolic requirements of other tissues. A relatively high BMR would require a correspondingly high energy intake, and, unless the environmental conditions were unusual, this would not only require devoting a significantly larger percentage of the daily time budget to feeding behaviour but also put the animal in more intense competition for limited food resources. Further, it is unlikely that the size of other metabolically expensive tissues (liver, heart, or kidneys) could be altered substantially.

The extent to which the liver can be reduced in size during encephalization is probably constrained by the particular energy requirements of the brain, which uses glucose exclusively as its fuel. Since the brain effectively contains no energy reserves, it is critically dependent on the continual supply of glucose from the blood. If this falls appreciably below its normal concentration of around 4.5 mM for even relatively short periods, significant dysfunction of the central nervous system can result. A major role of the liver is to replenish and maintain these levels, both by releasing glucose from the breakdown of its glycogen stores, reserves of which can comprise up to 10% of total liver mass, and by manufacturing it from alternative energy reserves mobilised from elsewhere in the body. Consequently, the energy demands imposed by increased encephalization cannot exceed the capacity of the liver to store and ensure the uninterrupted supply of the glucose necessary to fuel this metabolism.

Since almost the entire mass of the heart consists of the rhythmically contracting cardiac muscle, it is difficult to envisage how any significant reduction in the size of this organ could take place without compromising its ability to maintain an adequate circulation of blood around the body. The maintenance of high tissue perfusion rates will be particularly important to the brain, which, for the reasons discussed above, requires a continuous supply of high levels of glucose and oxy-

gen. In specific relation to humans, if activities requiring a high aerobic scope, such as persistence hunting, were important in the mode of life of later hominids, then this would have been an additional selection pressure for high cardiovascular performance.

Along with the brain, the kidneys have an extremely high metabolic rate associated with high levels of active ion transport. The energetic process is not the formation of the primary urine itself but the subsequent resorption of water and solutes from this filtrate as it passes through the nephrons. Since the ability of the kidney to concentrate urine is related to both the level of active transport and the length of these structures (especially the loops of Henle), it is likely that any reduction in either its energetic expenditure or its size will reduce the maximum urine concentration it is capable of excreting. The production of a more dilute urine would have been a particular problem for hominids if they were exploiting relatively open equatorial habitats where drinking opportunities were scarce and thermoregulatory requirements were already placing considerable demands on their water budgets (Wheeler 1991).

Finally, a reduction in the relative mass of skeletal muscle could not be used to balance the energy budget in the same fashion as reduction in the mass of the expensive tissues, because the mass-specific BMR of muscle tissue is considerably lower than that of any of the expensive organs and the average mass-specific BMR of the body as a whole. Consequently, in order for a reduction in skeletal muscle mass to compensate for the increased energy expenditure of the enlarged human brain, approximately 19 kg of muscle, about 70% of the total, would have to be replaced by an equal amount of tissue with no metabolic cost at all.

If the hypothesis of coevolution is correct, what is essential for understanding how encephalized primates can afford large brains is identifying the factors that allow them to have relatively small guts. The gut is the only one of the expensive metabolic tissues that could vary in size sufficiently to offset the metabolic cost of the encephalized brain. The reason for this is that, although gut size is related to body size, its size and proportions are also strongly determined by diet (Chivers and Hladik 1980, 1984; Martin et al. 1985; MacLarnon et al. 1986*a*, *b*; Martin 1990). Gut size is associated with both the bulk and the digestibility of food (Milton 1986, 1993; Milton and Demment 1988). Diets characterized by large quantities of food of low digestibility require relatively large guts characterized by voluminous and elaborated fermenting chambers (stomach and/or small intestine). An extreme example is the artiodactyl ruminants (e.g., cows), which are folivores, usually subsisting almost entirely on grasses. Conversely, diets characterized by smaller quantities of food of high digestibility require relatively smaller guts and are characterized by simple stomachs and proportionately long small intestines (emphasizing absorption) (Chivers and Hladik 1980). Carnivores typify this pattern.

The association between gut size and diet also holds within primates (Chivers and Hladik 1984, Martin et al. 1985). For example, Milton (1987) has emphasized the relationship between the relatively small gut in *Cebus* and a high-quality and therefore reasonably easy-to-digest diet composed of sugary fruits and protein- and oil-rich seeds as well as soft-bodied grubs, cicadas, and small vertebrates. Searching for animal foods takes up about 40–50% of their feeding time budget. The relative gut size in this primate contrasts strongly with that of *Alouatta* (fig. 4) which eats a poorer-quality diet composed of a high percentage of leaves as well as both ripe and unripe fruits, a significant percentage of which are highly fibrous figs (Crockett and Eisenberg 1987, Milton 1988). The relationship between gut size and diet also holds within the Old World Colobinae, which differ from the rest of the anthropoid primates not only in their generally relatively larger guts but also in their exceptionally large stomachs (Chivers and Hladik 1980, Martin et al. 1985). *Presbytis rubicunda*, which has a high-quality diet, contrasts sharply in relative gut size with *P. cristatus*, which relies on a much poorer-quality diet. Within the Hylobatidae, *Hylobates lar*, which spends more time feeding on fruits than on leaves, has a relatively smaller gut than *H. syndactylus*, which spends more time feeding on leaves than on fruits (Milton 1987).

There is also a close relationship between relative gut size and relative brain size (fig. 4). Animals with relatively large guts also have relatively small brains, while animals with relatively small guts have relatively large brains. However, there also appears to be a grade relationship present. For a given relative brain size, the colobines have a relatively smaller gut than the cebids and hylobatids; they may have lower relative BMRs overall or guts with higher mass-specific metabolic rates, or their other expensive organs may be relatively larger and/or energetically more costly. The resolving power of these comparisons is limited by the small number of species for which gut data are available, the small number of individuals studied within each species, and the fact that brain and gut data do not come from the same individuals. Interpretation is also limited by the absence of data on the allometries of metabolic cost of individual organs in non-human primates. However, even with these limitations, there appears to be a linkage between diet and the relative sizes of the gastro-intestinal tract and the brain.

The relationship between gut size and brain size may help to answer the question why anthropoid primates have relatively larger brains than the average for other mammals without also having a relatively high BMR (Milton 1988, Armstrong 1990). The reduced-major-axis equation (fig. 3) for the relationship between gut mass and body mass in the anthropoid primates is

$$\log_{10} G = 0.853 \log_{10} P - 1.271, \qquad (4)$$

where G is gut mass and P is body mass, both expressed in kilograms. This equation has both a lower slope and a lower intercept than the equation which characterizes the relationship in non-primate mammals (Brody 1945),

$$\log_{10} G = 0.944 \log_{10} P - 1.127. \qquad (5)$$

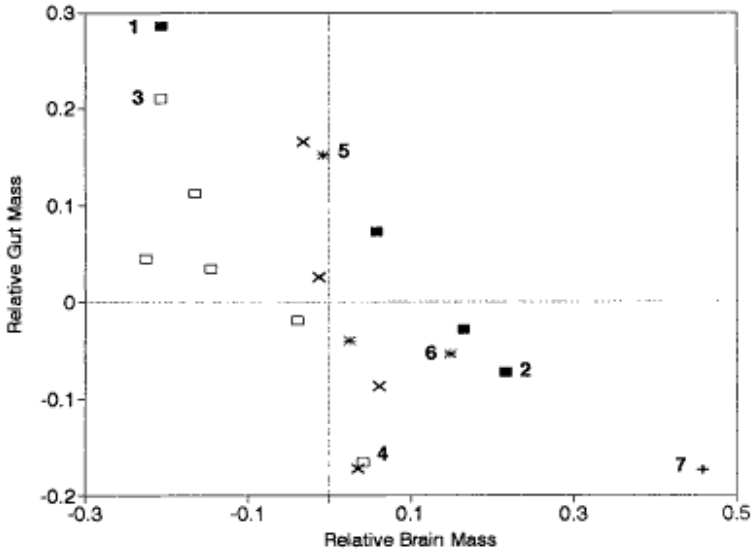

FIG. 4. *Relative brain mass versus relative gut mass in primates, determined on the basis of the higher-primate equations given in figure 3 and expressed as the residuals between the logged observed and expected sizes. The correlation of the residuals is* -0.69 ($n = 18$, $p < 0.001$, *one-tailed test*). Filled squares, *cebids;* open squares, *colobines;* stars, *hylobatids;* X's, *other catarrhines;* 1, Alouatta seniculus; 2, Cebus apella; 3, Presbytis cristatus; 4, P. rubicunda; 5, Hylobates syndactylus; 6, H. lar; 7, Homo sapiens.

These equations suggest that the average primate, with a larger relative brain size than the average mammal, also has a smaller relative gut size than the average mammal.

The relationship between relative brain size and diet is often mentioned in the literature on primate encephalization (e.g., Parker and Gibson 1979; Clutton-Brock and Harvey 1980; Gibson 1986; Milton 1987, 1988; MacNab and Eisenberg 1989) and is generally explained in terms of the different degrees of intelligence needed to exploit various food resources. For example, Parker and Gibson (1979; Gibson 1986) have argued that a relatively large brain and neocortical size correlates with omnivorous feeding in primates, which requires relatively complicated strategies for extracting high-quality foodstuffs. Alternatively, Clutton-Brock and Harvey (1980) have suggested that frugivores have relatively large brain sizes because they have relatively larger home ranges than folivores, necessitating a more sophisticated mental map for location and exploitation of the food resources. The results presented here suggest that the relationship between relative brain size and diet is primarily a relationship between relative brain size and relative gut size, the latter being determined by dietary quality (fig. 5). This would imply that a high-quality diet is necessary for encephalization, no matter what may be selecting

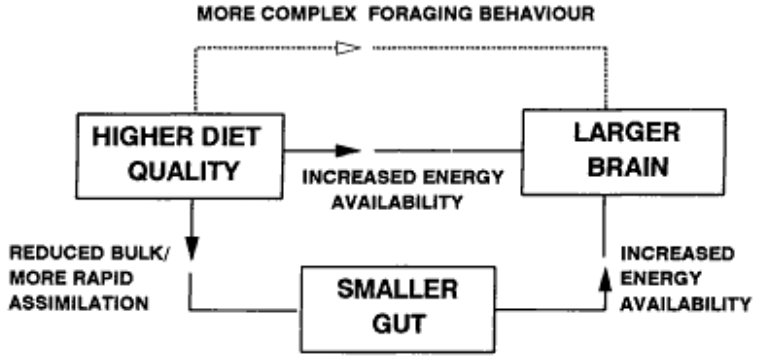

FIG. 5. *High-quality diet and increased encephalization.* Dashed line, *selection pressure;* solid lines, *relaxed constraints.*

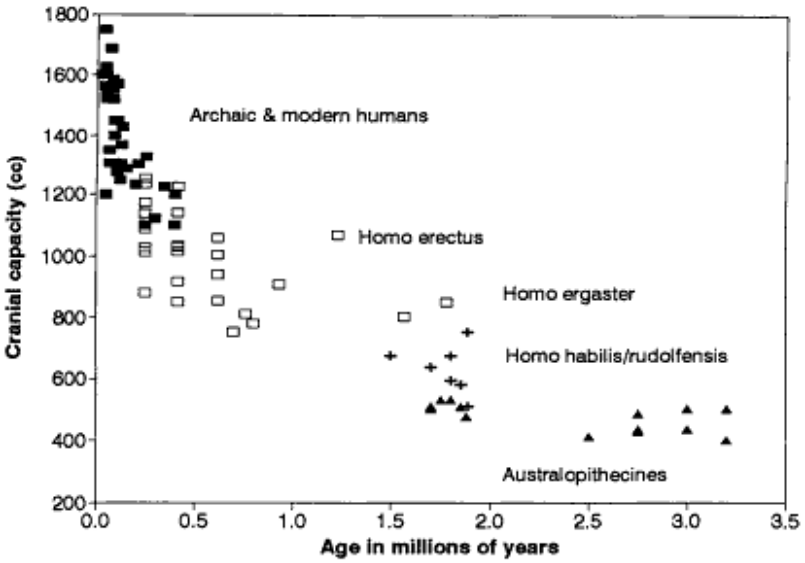

FIG. 6. *Increase in absolute hominoid cranial capacity over time. Brain sizes from Aiello and Dean (1990), ages from Aiello and Dunbar (1993).*

for that encephalization. A high-quality diet relaxes the metabolic constraints on encephalization by permitting a relatively smaller gut, thereby reducing the considerable metabolic cost of this tissue.

These results are compatible with the recent suggestion by Dunbar (1992, 1993, n.d.; Aiello and Dunbar 1993) that a large brain, and particularly a large neocortex ratio, is related primarily to group size in primates rather than to feeding strategy. It is certainly true, though, that a large brain size may have facilitated more complicated extractive foraging strategies (Dunbar n.d.) and acted as a secondary selection pressure for encephalization. A high-quality diet could also have benefited encephalization by directly increasing the total energy available to fuel an increased BMR. This would have applied, however, only if the quantities of high-quality food consumed were at least equal to those of the lower-quality food. In relation to humans this does not appear to be the case. Humans do not have a relatively high BMR, and, furthermore, Barton (1992) has demonstrated that they have a significantly lower daily food intake than non-human primates whose diet is of lower overall quality.

Brain-Size Change during Human Evolution

Over the past 4 million or so years the hominid brain has expanded from approximately 400 to 500 cc estimated for the australopithecines to 1,400 cc for modern humans (fig. 6). There have been two major periods of brain expansion. The first correlates with the appearance of the genus *Homo*, approximately 2 million years ago, when absolute brain size increased to an average of 654 cc (s.d. = 96.2, n = 8) in *H. habilis/rudolfensis* and approximately 850 cc in the earliest African *H. ergaster*. The second is coincident with the appearance of archaic *H. sapiens* in the latter half of the Middle Pleistocene, when brain size increased to its modern level (Leigh 1992, Rightmire 1981). This period of expansion probably represents an acceleration of an enlargement that had begun earlier in the Middle Pleistocene (Trinkaus and Wolpoff n.d.).

When brain size is corrected for body size, early hominid brain size falls either within or just above the upper range of the living primates (fig. 7). Even the most encephalized of the early hominids are closer in their relative brain sizes to the generic average EQs of the non-human primates, particularly *Cebus* and *Saimiri*, than they are to the EQs of modern humans. Both *Cebus* and *Saimiri* are relatively small-bodied primates. A considerable problem for the early hominids would have been to provide themselves, as a large-bodied species, with sufficient quantities of high-quality food to permit the necessary reduction of the gut. The obvious solution would have been to include increasingly large amounts of animal-derived food in the diet (Speth 1989, Milton 1987, 1988).

Although all hominids are more encephalized than the majority of living primate genera, the australopithecines show an overall lower encephalization than members of the genus *Homo*. They are similar in degree of encephalization to *Pan*, *Hylobates*, and *Saimiri*, which suggests that they had a diet at least equal in quality to that of these primates. *Gorilla* has one of the lowest levels of encephalization of any haplorhine primate, and

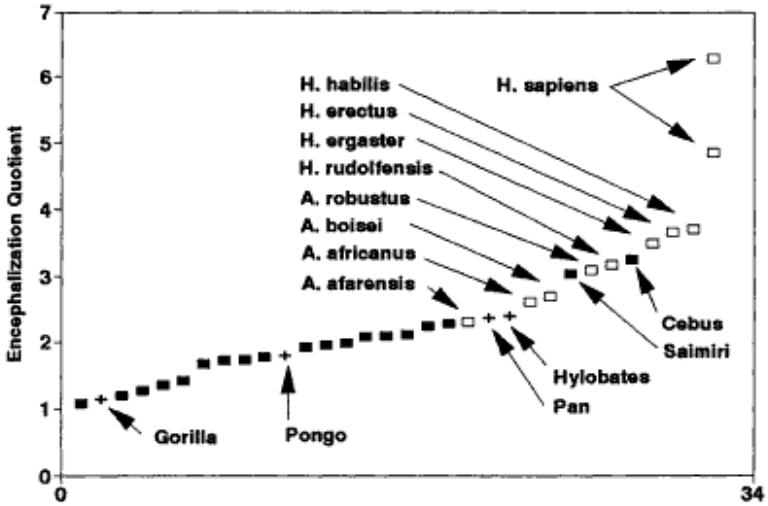

FIG. 7. *Encephalization quotients for hominids, by species* (open squares), *compared with other higher primates, by genus—apes* (pluses) *and monkeys* (solid squares)*—arranged in ascending order of magnitude. The lower EQ for modern humans is based on the body and brain masses used in this analysis; the higher is based on a body mass of 44 kg and a brain mass of 1,250 g (Harvey, Martin, and Clutton-Brock 1986). Hominid data from McHenry (1994).*

the much higher level of encephalization of all of the australopithecines suggests a diet of significantly higher quality than that of this genus. This suggestion of a relatively high-quality diet for all of the australopithecines, and particularly for the robust australopithecines, is consistent with evidence from dental microwear. Kay and Grine (1988) conclude that the microwear on the molars of the robust australopithecines resembles that of extant primates that eat hard food items, while that on the molars of the other australopithecines suggests that they subsisted more on leaves and fleshy fruits. It is interesting that *Cebus*, the most encephalized of living non-human primates, not only eats hard food items and closely resembles the robust australopithecines in its microwear pattern (Kay and Grine 1988) but also has a high-quality diet (Milton 1987) and resembles humans in its gut morphology (Martin et al. 1985, Milton 1987). Recent analysis of both the strontium-calcium and stable carbon isotope ratios of *Australopithecus robustus* from Swartkrans (Member 1) suggests an omnivorous rather than a strictly vegetarian diet for these hominids (Sillen 1992, Lee-Thorp, van der Merwe, and Brain 1994).

Because of their higher levels of encephalization, members of the genus *Homo* would be expected to have had an even higher-quality diet than the australopithecines. Sillen, Armstrong, and Hall (n.d.) have argued that the diet of early *Homo* from Swartkrans probably differed from that of the robust australopithecines in either the incorporation of more underground storage organs (soft bulbs, tubers, etc.) or the preferential consumption of animals having relatively high strontium-calcium ratios such as hyraxes. Meat consumption by early *Homo* might also be inferred from polish on Oldowan tools (Keeley and Toth 1981) and by cutmarks on bone (Potts and Shipman 1981, Shipman 1986, Bunn and Kroll 1986), but there is always a certain degree of uncertainty over which of the hominids, australopithecines or early *Homo*, actually made and used the tools. Evidence is stronger that early *H. erectus* (*H. ergaster*) was more predatory and, by inference, incorporated more animal products into its diet than the earlier hominids (Shipman and Walker 1989). Support for this interpretation rests primarily on the postcranial skeleton, which suggests a more efficient adaptation to rapid locomotion. Shipman and Walker also suggest that the Acheulian tool tradition might be interpreted as indicating greater reliance on and increased frequency of the processing of animal tissues.

It is difficult to infer relative gut size for the hominids, because, unlike the brain, the gut is not encased in a bony capsule whose volume can be measured. However, certain features of the postcranial skeleton of WT-15000 (*H. ergaster*) suggest that this hominid had a smaller relative gut size (consistent with its higher level of encephalization) than did the australopithecines, represented by AL-288-1 (*A. afarensis*). The large gut of the living pongids gives their bodies a somewhat pot-bellied appearance, lacking a discernible waist. This is because the rounded profile of the abdomen is continuous with that of the lower portion of the rib cage, which is shaped like an inverted funnel, and also because the lumbar region is relatively short (three to four lumbar vertebrae) (fig. 8). The narrowing of the upper portions of the thoracic cage is associated with the extremely powerful

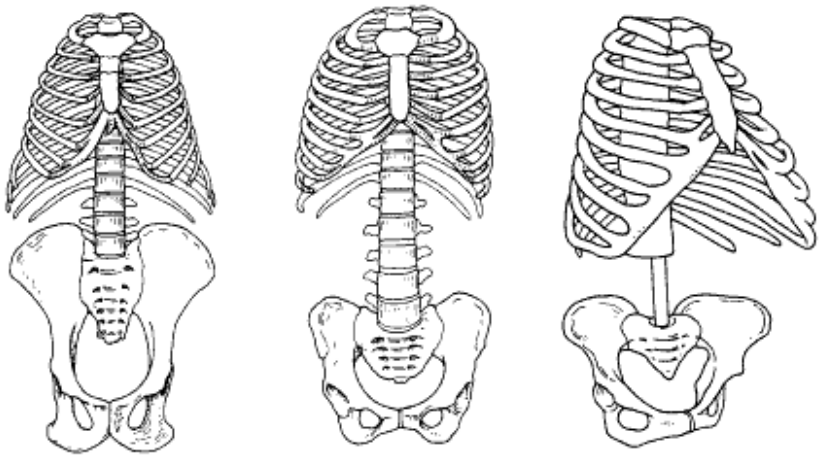

FIG. 8. *Trunks of a chimpanzee* (left), *a human* (center), *and* Australopithecus afarensis (right), *showing the protruding rib cage in the latter.* (A. afarensis *reconstruction after Schmid 1983, chimpanzee and human after Schultz 1950.)*

muscle complex of the pectoral girdle used during arboreal locomotion (Schmid 1991). The reconstructed rib cage of *A. afarensis* (Schmid 1983) indicates that these hominids retained a funnel-shaped thorax similar to that of the chimpanzee. *A. afarensis* differs from the pongids only in having a longer lumbar region (six lumbar vertebrae). Additional clues about the proportions of the abdominal organs of australopithecines are provided by the structure of the pelvis, which, because of their bipedal posture, provided some support to this region of the body. Both *A. afarensis* (Tague and Lovejoy 1986, Ruff 1991) and *A. africanus* (Robinson 1972) possessed wide pelves relative to their stature, the outwardly flared upper margins of which are consistent with the presence of a well-developed and protuberant abdomen (Schmid 1991).

Pongid and australopithecine trunk morphology contrasts with that of modern humans. The barrel-shaped thoracic cage and relatively smaller pelvis of *H. sapiens* border a narrower abdominal region with a distinct waist absent in the trunk of apes. *H. ergaster* is the first known hominid to approximate modern human body proportions (Ruff and Walker 1993). The inference is that it most probably also had a relatively smaller gut. Modern human trunk proportions in early *Homo* would have had additional significance if active hunting and/or long-distance migration was important to the ecology of these hominids. High levels of sustained activity require an extremely efficient cardiovascular system, the key components of which are located within the thoracic cage. In apes and australopithecines the construction of the shoulder girdle restricts the elevation of the upper portion of their funnel-shaped rib cages during respiration (Schmid 1991). Ventilation of the lungs was probably mainly dependent on the movements of the diaphragm and would therefore have been less effective than in *Homo*, in which the upper part of the rib cage can be raised to enlarge the thorax during inspiration. In addition to this physiological consideration, Schmid (1991) has identified biomechanical advantages of the *Homo* body form. A significantly narrower waist than in the australopithecines would have allowed the arms to swing more freely in the lowered position and permitted greater torsion in the abdominal region, both of which are essential in stabilising the upper body during bipedal running.

These observations are relevant to the first marked increase in hominid brain size. For the second increase, the introduction of cooking may have been an important factor. Cooking is a technological way of externalising part of the digestive process. It not only reduces toxins in food but also increases its digestibility (Stahl 1984, Sussman 1987). This would be expected to make digestion a metabolically less expensive activity for modern humans than for non-human primates or earlier hominids. Cooking could also explain why modern humans are a bit more encephalized for their relative gut sizes than the non-human primates (see fig. 4).

Conclusion

Although there is still much to learn about energy balances in humans and non-human primates, a picture is emerging that is consistent with a linkage between hominid diet and the relative sizes of the gastrointestinal tract and the brain. Our work complements that of Milton (1986, 1993; Milton and Demment 1988), which suggests that the emergence of the hominids, and particularly of *Homo*, was associated with the incorporation of higher-quality foodstuffs into the diet. A high-quality diet was probably associated with a reduction in

the size and therefore the energetic cost of the gut. If this is correct, encephalization in the hominids was able to proceed without placing any additional demands on their overall energy budgets. Furthermore, if the exploitation of these high-quality foods, such as animal products, nuts, or underground tubers, required more complex behaviours, then this also could have acted as one of the selection pressures for the observed increase in brain size. Further increases in brain size might well have been facilitated by the introduction of cooking to render food more digestible.

These conclusions are derived from the general observation that there is no significant correlation between relative basal metabolic rate and relative brain size in humans and other encephalized mammals. If an encephalized animal does not have a correspondingly elevated BMR, its energy budget must be balanced in some other way. The expensive-tissue hypothesis suggested here is that this balance can be achieved by a reduction in size of one of the other metabolically expensive organs in the body (liver, kidney, heart, or gut). We argue that this can best be done by the adoption of a high-quality diet, which permits a relatively small gut and liberates a significant component of BMR for the encephalized brain. No matter what was selecting for encephalization, a relatively large brain could not be achieved without a correspondingly increase in dietary quality unless the metabolic rate was correspondingly increased.

At a more general level, this exercise has demonstrated other important points. First, diet can be inferred from aspects of anatomy other than teeth and jaws. For example, an indication of the relative size of the gastrointestinal tract and consequently the digestibility of the food stuffs being consumed is provided by the morphology of the rib cage and pelvis. Second, any dietary inference for the hominids must be consistent with all lines of evidence. Third, the evolution of any organ of the body cannot profitably be studied in isolation. Other approaches to understanding the cost of encephalization have generally failed because they have tended to look at the brain in isolation from other tissues. The expensive-tissue hypothesis profitably emphasizes the essential interrelationship between the brain, BMR, and other metabolically expensive body organs.

Comments

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Aiello and Wheeler propose that a high-quality diet allows a larger percentage of an animal's total energy reserves to go to the brain than would otherwise be the case because the metabolically expensive gut is reduced in size. Their suggestion provides a solid lead into the question how nonhuman primates can afford to expend about twice as much and humans about four times as much energy on their brains as most other mammals (Armstrong 1985*a*, *b*, 1990).

Questions remain, however. Aiello and Wheeler propose that for human encephalization, the energy saving stemming from a reduced gut size is sufficient to eliminate the need for other forms of conservation. One of the attributes of the brain is that it utilizes glucose and does not switch to glycogen when reserves run low, in contrast to muscles, which can readily shift from one form of energy to another. Do the splanchnic organs resemble muscle in their use of glycogen, or are they, like the brain, restricted to glucose? If they use and store glycogen, part of their weight is in the form of prepackaged energy, and some sharpening of the analysis may be called for.

The differences between the expected and observed sizes of human organs and metabolic costs reported in the paper are based on primate data. Given that primates differ from other mammals both in having relatively big brains for their energy reserves and in utilizing a larger percentage of those energy reserves for their brains (Armstrong 1985*a*, *b*, 1990), does a change in relative gut size account for how primate encephalization differs from that in other mammals, or is some other mechanism or structural shift important here?

Hypotheses about functional biology that relate to a single species are weaker than those which can explain differences in many. The connection between diet and the expensive-brain hypothesis will be strengthened if Aiello and Wheeler can point to similar findings in other taxa. Birds with low metabolic rates have relatively smaller brains than those with standard metabolic rates, paralleling observations among mammals (Armstrong and Bergeron 1985). The relationship of owls to other birds, however, resembles that of primates to other mammals; owls have relatively large brains given their total energy supply. Do owls support their relatively big brains with reduced guts? A positive finding would strengthen the authors' hypothesis. In other situations, negative findings might also strengthen the hypothesis. Bat species differ in metabolism, diet, and encephalization, insectivorous bats having relatively smaller brains and lower metabolic rates than noninsectivorous ones. The differences in relative brain size disappear, however, when the differences in metabolism are taken into account; when the standard becomes total energy reserves rather than simple body weight, the bat species have equivalent degrees of encephalization (Armstrong 1983). In this case, one would not expect to find a difference in gut size between dietary groups of bats. Thus tests for the generality of the hypothesis may be found outside of the primate order.

Although the why of increased brain size is of general interest, hypotheses concerning this attribute will be weak until we come to understnd how brains can afford to increase in size and what structural modifications are correlated with that increase. This paper is a welcome addition to our knowledge.

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As Aiello and Wheeler point out, a few workers have suggested that brain size data reveal two periods of expansion over the past four million years of hominid evolution. However, an earlier survey of the literature failed to find wide support for a punctuated-equilibrium model of hominid brain size evolution; in fact, rates of evolutionary change in cranial capacity (millidarwins) suggest that brain enlargement in *Homo* appears to be autocatalytic, the data supporting a souped-up version of the gradualists' model (Falk 1987). Leigh (1992:11), examining trends in cranial capacity, concludes that "previously proposed punctuated equilibrium models do not adequately describe later hominid evolution." Furthermore, rates of brain size increase in *Homo erectus* and early *H. sapiens* cannot be statistically distinguished. In short, more data are sorely needed to assert that a burst of brain expansion coincided with the appearance of archaic *Homo*.

That said, it is nevertheless true that the past two million years have witnessed a dramatic increase in brain size in the genus *Homo*. Elucidation of the "prime movers" for this increase has become a favorite pastime in paleoanthropology (Falk 1992). Candidates include hunting, tool production, warfare, work, social intelligence, and language. The problem with these behavioral prime movers, however, is that they are highly speculative and do not lend themselves well to hypothesis testing. In contrast to most prime-mover theories, the present article is grounded in physiology and comparative anatomy. As a result, it is supported by quantified data and paves the way for collection of more data and further testing of related hypotheses. The expensive-tissue hypothesis also differs from the above conjectures in that it suggests a physiological/anatomical complex that acted as a *prime releaser* permitting selection for increased brain size rather than speculating about one hypothetical behavior that was the primary target of that selection (i.e., a prime mover).

In a nutshell, the expensive-tissue hypothesis proposes that a high-quality diet permitted a relatively smaller gut and thereby relaxed a metabolic constraint on brain size. Elsewhere, I have proposed another physiological/anatomical complex as a prime releaser of brain size in the genus *Homo*, namely, evolution of a network of cranial veins (a "radiator") that serves to cool the brain under conditions of hyperthermia (Falk 1990). Here I suggest that the two hypothetical releasers are compatible because cerebral metabolism, relative brain size, and thermolytic needs are all intertwined. If both releasers were instrumental during hominid evolution, perhaps the underlying behavioral factors (diet for metabolism, locomotion for vascular evolution) may be woven into a satisfying (if not falsifiable) scenario.

Brains are exquisitely heat-sensitive, and human brains have particularly great cooling needs. One reason for this is that the *ratio* of cerebral to body-resting metabolic rates increases with increased body size in mammals, and humans are relatively large mammals (Caputa 1981). (BMR decreases considerably with increasing body size in mammals whereas cerebral metabolic rate decreases only slightly with increasing body size.) A second reason that human brains have great cooling needs is that humans are highly encephalized—that is, they have relatively large brains generating potentially damaging heat given their body sizes. For example, as Aiello and Wheeler observe, the actual metabolic output for the human brain (14.6 W) is much larger than the 3 W expected for a mammal of similar body size.

The question, then, is not only "Where does the energy come from to fuel the encephalized brain?" but also Where do the resources come from to *cool* the encephalized brain? Aiello and Wheeler propose a shift to a high-quality diet (with a reduction of gut) as an answer to the first question. I propose refinement of bipedalism under hot savanna conditions (with a change in cranial vasculature) as an answer to the second (Falk 1990). Weaving these two together, we may now speculate about what early hominids were *doing* out there on the savanna—maybe they were "working out" and looking for veggie burgers/Big Macs! This fits with Wheeler's (1988) midday-scavenging hypothesis (which should perhaps be retitled "Stand Tall, Stay Cool, and Pig Out"). On a final note, convergent evolution for increased encephalization has occurred independently in certain whales and higher primates. The relatively large brains of whales generate a good deal of heat despite their aquatic habitats. In keeping with this, a recent report suggests that at least one species of whale has independently evolved a net of blood vessels that connect with the base of the skull and protect the brain from hyperthermic blood flow (Ford and Kraus 1992). In light of the expensive-tissue hypothesis, one wonders what gut/diet data might reveal about big-brained cetaceans. It remains to be seen if other physiological factors will be identified as potentially important for investigating hominid (brain) evolution. I hope so because, in my opinion, physiological hypotheses certainly beat storytelling.

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The hypothesis advanced by Aiello and Wheeler, although they do not explicitly say so, follows the old Fisherian theorem equating Darwinian fitness with energetic efficiency of reproduction (Fisher 1930). It does not pretend to identify the causes of the increase of the brain size in hominid evolution; it simply points to what the authors consider a *conditio sine qua non* for that increase. The logic is clear within the paradigms and numerical data sets employed.

Although it deals with evolution, the entire paper works on typological principles. Energy needs are predicted for a *typical* primate; humankind is represented

by a "standard" male, and it seems from the body weight of 65 kg that it is a "white" one. Variation in human brain size and body size is very considerable, producing an enormous number of combinations of brain, gut, and total body sizes and hence wide ranges of encephalization quotients. The average weight seems to be closer to 55 than to 65 kg (Henneberg 1990). The data presented here on the mass-specific metabolic rates of various organs vary considerably even if one allows for interspecies differences and discrepancies in laboratory techniques (compare table 1 and table 3). It is thus difficult to ascertain how reliable the estimates of "metabolic increments" in table 4 are and how many human individuals would conform to them.

There is little doubt that the absolute size of the hominid brain increased during the history of this lineage while reliance on higher-quality foods, especially meat, increased, leading to the reduction in the size of the gastrointestinal tract. Whether this concurrence indicates interdependence is another matter. The changes in BMR caused by the 850 g increase in brain size are small—9.5 W, corresponding to 10.5% of the total BMR, as indicated by the authors, or to 5.8% of the FMR, calculated as 1.8BMR. Thus a simple drop in FMR to 1.7BMR would more than compensate for increased brain energy consumption. The amount of energy in question equals that expended during 45 minutes' leisurely walking (4 km/hr.) or the change in BMR accompanying change in body mass by 6.8 kg—less than the difference between the "average for humankind" (55–58 kg [Henneberg 1990]) and the authors' assumed 65 kg. A change of a few degrees in the temperature of the immediate environment might save the required amount of energy, and so would a moderate decrease in habitual food intake—dieting individuals can reduce their resting energy expenditure by as much as 30% (Lamb 1984). It seems that extending the time taken up by sleep would also do the trick. The postulated increase in energy requirement of the larger brain could be absorbed in numerous ways other than the reduction in the size of the gastrointestinal tract.

The question remains, Why do we need a larger brain? Must it be absolutely larger, as in a poor gorilla scoring so abysmally on the encephalization quotient and yet considered intellectually closer to humans than cebids, or simply relatively larger as indicated by that quotient? In most measures of encephalization, brain size is expressed as a (variously calculated) fraction of body size. A larger gut contributes to the increase in body size not only directly but also through the requirement of increased muscle and skeletal mass to carry it around. Thus a larger gut means considerably greater body mass and hence a larger denominator for the encephalization quotient and a smaller quotient—simple arithmetic rather than some biological phenomenon. Is bigger really better?

The threefold increase in hominid brain size since the Pliocene is paralleled by a 3.2 times increase in brain size in equids (from 270 g in *Pliohippus* to 870 g in modern horse [Jerison 1973]) and does not seem exceptional. The uniqueness of hominid evolution rests in the lack of expected increase in body size—thus a reduction of body size relative to brain size. This overall reduction results from externalization of functions. Aiello and Wheeler correctly point to cooking as an example, but the list is much longer. Externalization leads to a reduction in the overall energy requirements of the human body—the amount of muscle and consequently the robusticity of the skeleton and the size of the viscera servicing the body decrease or, rather, for most of hominid evolution do not increase at a rate commensurate with the increase of the brain. From the terminal Pleistocene, however, until several hundred years ago, the overall size of the human body actually decreased (Frayer 1984, Jacobs 1985). It seems that this general "structural reduction" of the human body is responsible for our large encephalization quotient.

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This intriguing paper ought to provide considerable fodder for thought, renewed testing, and, ideally, synthesis with other aspects of possible brain-behavior-growth constraints. As intriguing as it is, however, I remain skeptical that these economics-based models (including Dunbar's ideas regarding the neocortex and language as a cheap form of social grooming [see Dunbar 1993 and my response]) get us any nearer to understanding the relationships between brains and behavior that might have been targets for past selection pressures. My problem ever since 1966, when I first published on the question of brain size in human evolution, has been that I cannot see the brain as a unitary organ with a simple behavioral task to accomplish such as "intelligence," "language," "adaptive behavior," or any other such pedagogical fig leaf to cover our ignorance about how the brain evolved. To me the brain is composed of a multitude of parts that serve numerous behavioral functions that can have life-or-death consequences depending on how circuits are activated or inhibited, information is processed, and action patterns are manifested in environments with complex interdependencies between the social and material. Can such macroevolutionary models possibly account for nitty-gritty real-life selection walks that particular hominid groups took through a million or so years (Holloway 1979:84–85)?

I wonder if the authors should be so certain that "whatever the selection pressures" the evolution of primate brains had to follow this particularly interesting set of constraints. Where are the data that will show variation in these parameters in a population and indicate which variations are favored? Will the application of these economic models explain why chimpanzees (bonobos and troglodytes), gorillas, orangutans, etc., behave as they do (since the only neural variate ever discussed is total brain size)? Can we think a bit more deeply about

just what brain size is? Are all neural tissues equally as energy-hungry and "expensive"?

Having railed against viewing brain size as the end-all of the neural substrate underlying behavior that varies and is eventually selected for (or against) all these years, I am chagrined to admit that I certainly haven't come any closer to something more substantive than notions of "reorganization." I cannot help but feel that we are burdened by our fixation on what we can easily measure, brain size, and overlook the relationships that have emerged over the past 50 years between neural nuclei and their fiber tracts and behavior. Aiello and Wheeler offer a different and intriguing scenario here, and I look forward to hearing more.

The extrapolation to feeding adaptations from rib cage and pelvic morphology (i.e., between *A. afarensis* and *Homo*) is interesting, but I wonder how feeding strategies and gut size can be related to metabolic constraints and brain size within the hominids. As I see the record, there are times when brain size seems to have increased without much concomitant body-size increase (e.g., *Homo erectus* to archaic *Homo*, 800–900 ml to about 1,200–1,300 ml) or when the brain-size increase might be related (at least partially) allometrically to body size (e.g., *Homo habilis* to *H. rudolfensis* or *Australopithecus afarensis* to *A. africanus* or even, possibly, archaic *Homo* to *H. neanderthalensis*). The record suggests to me that there was plenty of heterogeneity of possible cause-effect relationships between brains, bodies, feeding, and behaviors within any ongoing evolutionary period of the past 2 million years (see, e.g., Holloway 1980: 119). I sincerely doubt that feeding and brain-gut-size interdependencies can explain these interdependent changes.

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Aiello and Wheeler's expensive-tissue hypothesis is a multifacted model of how *Homo* spp. could afford to increase significantly their cranial capacity beyond that of their phylogenetic predecessors, the australopithecines. The authors explain how (in a physiological, metabolic, and anatomical sense) the members of genus *Homo* accomplished this transformation. The hypothesis rests on a critical assumption that Aiello and Wheeler readily admit cannot be directly demonstrated—that the changes in the proportions of the two organs (brain and gut) were contemporary evolutionary events. In their model, as the brain enlarges, the gut is reduced in size. A linchpin in this "evolutionario" is a change in the quality of hominid diets, with animal-derived constituents becoming increasingly important. They should be complimented for attempting this interesting synthesis, although at times it seems that everything including the kitchen sink (or perhaps stove, since the suggestion is also made that cooked food may have played a role in encephalization) has been added to this "recipe" for a bigger brain. They suggest that earlier explanations are insufficient because others "have tended to look at the brain in isolation from other tissues," but, as they note, these other efforts have addressed the why question rather than the how question (Aiello and Dunbar 1993, Byrne and Whiten 1988, Milton 1979, and others).

In discussing the pattern of changes in brain size in human evolution, Aiello and Wheeler suggest two major periods in which this occurred. One corresponds to the appearance of the earliest members of genus *Homo*, whether *habilis* or *rudolfensis*, and later *ergaster*. The second period is associated with archaic *H. sapiens*. Leaving aside the issue of just how many species are *really* represented in the first period (cf. Foley 1991), it appears that the earliest members of *Homo* were not larger in body size than australopithecines and did not have modern limb proportions (Johanson et al. 1987). It is not at all clear how much animal-derived food was in their diets or whether this was vertebrate or invertebrate, and it is perhaps problematic to launch the coevolution of gut and brain on such a tentative foundation. However, with species like *ergaster* and later hominids Aiello and Wheeler are on much firmer ground with respect to modern morphology and dietary patterns and composition.

In advancing their case for "active hunting and/or long-distance migration" they suggest that the reshaping of the rib cage from a funnel-shaped (pongid and australopithecine) to a more barrel-shaped modern appearance would enhance the cardiovascular system and more efficiently ventilate the lungs. This sounds a bit like the "notion of progress" and is not critical to the hypothesis as presented. Alternatively, such a change may be a function of the change from quadrupedal to bipedal locomotion, as suggested by Hunt (1994). Field biologists who witness episodes of sustained locomotion and especially arboreal hunting by wild chimpanzees would not doubt their cardiovascular fitness or their respiratory functioning (Stanford et al. 1994).

Aiello and Wheeler come to their inference of coevolution of brain size and gut size by a process of elimination. That is, they examine other "expensive tissue" (heart, kidney, liver) and conclude that size reduction in any of them would be too risky, whereas the gut has more flexibility in its size (contingent on the necessary dietary changes, of course). In effect, the gut coevolves by default, a less than satisfactory evolutionary explanation and one that needs to be addressed if this hypothesis is to be developed further.

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How humans can afford their large brains has long been a question of interest. Humans are regarded as having a small gut for their body mass, an unusually large brain, and a normal metabolic level—a set of conditions which

appears to pose a paradox, since brain tissue is regarded as energetically expensive. Aiello and Wheeler describe the case of the incredible shrinking gut as a solution to this apparent paradox, but the functional mechanism linking these phenomena in an evolutionary pathway is not made clear. Though available data indicate a small gut in humans (e.g., Martin 1981, Milton and Demment 1988), measurements of human gut proportions often appear to have been made on individuals from Western nations eating refined Western diets. Speculations on human gut proportions and gut size in humans and other primates should be advanced with caution, as work on other animal species shows that different sections of the gut can rapidly alter in response to changing dietary conditions, even within the life span of the individual (e.g., Gross, Wang, and Wunder 1986). Some non-Western rural human populations which consume large amounts of dietary fiber are estimated to obtain as much as 10% of their total caloric intake each day from the volatile fatty acids produced in cecal and colon fermentation; in contrast, this figure for the low-fiber Western diet is around 0.7% (Van Soest et al. 1982, Milton 1986). This magnitude of difference suggests that some human populations may have considerably larger colons than others and thus perhaps a large overall gut. Would Aiello and Wheeler then predict a correspondingly smaller brain size in such populations? I doubt it. If I remember correctly, the human brain and nervous system are estimated to account for only some 20% of daily energy turnover, leaving a robust 80% to take care of other business.

However, even if some human populations do have larger colons and guts, I would still predict that modern humans as a species have a small gut for their body mass. In my opinion, when using an evolutionary perspective it is always best to try to account for both the how *and* the why, since the two are intertwined (Milton 1988). As is pointed out by Jerison (1973), primates appear to have been relatively large-brained mammals since the inception of the order, which suggests that they have long tended to seek behavioral (brain-based) solutions to their dietary problems and thus have long been able to "afford" the mental solution—that is, afford to have a somewhat large brain relative to body mass. I have proposed that this came about because the ancestral lineage ultimately leading to Primates was somehow able to enter the as-yet-unfilled arboreal plant-based dietary niche provided by tropical-forest angiosperm trees and vines and then radiate in such a way as eventually to control a large proportion of the highest-quality plant foods (new leaves, ripe fruits, and flowers) in this arboreal environment (Milton 1987:94–95). Entry into this dietary niche appears to place considerable pressure on the feeder to lower the costs associated with procurement of these patchily distributed plant foods—a solution which in our order appears to have been resolved in large part by the development of cerebral complexity, with the attendant behavioral plasticity, memory, learning, and social skills required to lower food acquisition costs and improve foraging returns (see, e.g., Milton 1979, 1981, 1987, 1988, 1993). Manual dexterity and the use of the hand in preparing food and in feeding are also important Primate traits which serve to broaden the overall Primate dietary niche and contribute to foraging success (see, e.g., Gibson 1986). It isn't so much that guts shrank, giving Primates extra metabolic scope to afford their brains; rather, it would appear that the development of the brain in direct association with an unusually high-quality diet and the foraging skills required to obtain it may gradually have facilitated some reduction of overall gut mass. This is an important distinction.

How did the human genus break into its unusually profitable dietary niche—one which I have termed "the niche of the cultural omnivore" (Milton n.d.)—so that it could get by with a smaller gut? Let's imagine a protohuman ancestor living in a changing environment in which, for whatever reason, higher-quality plant foods become increasingly difficult to obtain. There are two principal solutions to this problem. One is to turn to lower-quality foods that are relatively abundant but fairly easy to obtain (thereby, in the hominoid lineage, with its characteristic hominoid gut morphology, sacrificing mobility and many behavioral aspects (e.g., orangutans and gorillas relative to chimpanzees); the other is to hold the line with regard to dietary quality and find some way to cover the increasing costs of procuring rare but far more nutritionally concentrated, high-quality dietary items (for discussion see Demment 1983; Milton 1986, 1987, 1988, 1993; Milton and Demment 1988).

Obviously, it is this second solution which was favored in our lineage. We find crude stone tools and reduced dentition as characteristic traits of early members of our genus—traits which testify to the increasingly important role of *technology* in terms of the ancestral human diet (Milton 1987, 1993; Milton and Demment 1988). The reduced dentition of early humans indicates that technology had begun to intervene in human dietary behavior, in effect placing a buffer or barrier between human dental morphology and the human gut (*and thus selection pressures*) and foods consumed. Stone tools could have facilitated access to formerly unavailable foods, both plant and animal, or upgraded existing food quality. Elsewhere (Milton 1987) I have discussed the probable role of meat eating in human evolution, pointing out that though animal protein is an excellent amino-acid source for humans, it is less desirable as an energetic substrate.

Rather than suggesting, as Aiello and Wheeler do, that the large brain in our lineage relates to group size rather than feeding strategy (an argument which seems forced in light of their paper's content), I would argue that early human sociality as well as group size is best viewed as another type of dietary tool. In the genus *Homo*, a division of labor and food sharing appear to have been the social tools contributing to dietary sufficiency (e.g., Milton 1987). Humans in modern technological societies often forget just how problematic getting one's daily food can be, but, as Richards (1948) noted long ago, it is food not sex that makes the world go round. Though

reduction of gut mass may well free up some energy to support other organs, the brain is lost without a constant and dependable energetic substrate. Thus it would be evolutionarily irresponsible to cast off gut tissue until mental complexity was sufficiently developed to more or less ensure dietary quality. Indeed, only when selection is relaxed should any reduction in gut size occur. The gut is certainly an important part of the evolutionary picture both for nonhuman Primates and for humans, but it seems pointless to try to view gut changes apart from the foraging strategy and dietary niche in which they are evolutionarily embedded.

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Organisms can't afford long-term debt. Since the recognition that the costs of encephalization are especially high, therefore, metabolic constraints have been a potential source of explanation of variation in brain size. But if the idea that increased expenses must be met by reduced costs is old news, Aiello and Wheeler's presentation of the expensive-tissue hypothesis is nevertheless novel. The key argument is that there are few dimensions of freedom in primate energy budgets and that of these only gut cost is likely to vary enough to account for the observed differences in brain size. They find that relative gut size varies inversely with relative brain size and infer that only species with cheap guts can afford large brains and tend to have them.

Because it provides a clear account of both the nature and direction of causation, this is a very valuable hypothesis. The evidence for a dietary explanation of hominid brain expansion is compelling. The hypothesis deserves rigorous examination to test points of weakness. We suggest two.

First, there appears to be a hidden assumption that the energy budget of an animal at basal metabolic rate (BMR) is constrained—that the BMR is the minimal rate of energy turnover at which organs can be maintained. If metabolic rate could fall below BMR, however, and certain organs could survive, then the observed pattern of energy distribution among organ systems at BMR is not mandatory. It is only if BMR is constrained that the Aiello-Wheeler logic works. Is it true, therefore, that BMR is constrained—that it is the minimal possible rate? No. BMR is formally measured on waking subjects. During sleep, metabolic rate falls by about 10% (Blaxter 1989). It also falls in other contexts, such as during starvation. It can differ between human populations by as much as 17% (Blaxter 1989:144). Such variation means that the amount of energy consumed by different organs operating at BMR is higher than that dictated by survival. Therefore, it is illegitimate to infer that the observed distribution of energy towards different organs at BMR represents the minimal levels needed by those organs. For some such organs the energy turnover at BMR may be the minimum; for others it may not be.

One escape from this line of thinking could be to suggest that the BMR represents not the minimal but an average metabolic rate, a rate which shows how animals distribute energy to organs at a typical working level. But this escape would be a false one, because as soon as we think in terms of actual expenditures of energy we must acknowledge that BMR does not predict the total energy intake (daily energy expenditure). How high the average metabolic rate is above BMR varies between species in ways not predicted by BMR itself.

We conclude that BMR cannot in theory be used to index the total or average or minimum amount of energy flowing through the system or to different organs. This doesn't mean that Aiello and Wheeler's conclusions are wrong, but it does mean that there are logical and empirical issues missing from the argument. A key question is how much potential variation there is in the metabolic rates of organs operating at BMR.

Second, Aiello and Wheeler have been forced to assume that the metabolic costs of organs scale isometrically with their weights, but this relationship is unknown. What is the true cost of evolving a larger brain, and how might this cost be supported? Using their figure 3, we can infer that a standard human needs only about 5% more daily energy to maintain its enlarged brain over that of a small-brained individual of equivalent body size. Could larger brains be maintained without gut-size reduction by dietary compensation? It is curious that of the expensive organ systems only the brain scales with an exponent substantially less than 1 (Peters 1983). If larger species find the energy to support these relatively larger organs, why can't they support larger, less costly (in terms of mass-specific energy) brains?

In sum, this is an exciting and stimulating result. We look forward to seeing the Aiello-Wheeler logic fleshed out with better data and applied to taxa such as bats (do fruit bats have smaller guts than insectivorous bats?), cetaceans (are odontocetes smaller-gutted than mysticetes?), and rodents. We also look forward to the resolution of what appears, on the face of it, to be a problem. How do Aiello and Wheeler reconcile their conclusion with the allometry of adult brain mass in mammals, birds, and reptiles? In all three groups, gut mass scales isometrically with body mass. Yet in mammals brain mass scales to the 0.75 exponent, compared with a 0.56 exponent for birds and reptiles (Martin 1981). An explanation of these patterns in terms of the expensive-tissue hypothesis would be a substantial achievement.

Reply

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The expensive-tissue hypothesis is a hypothesis to explain the coevolution of the brain, the digestive sys-

tem, and diet. We are pleased to note that the majority of the commentators find it novel, interesting, and relevant and also that they recognize that we are not claiming that dietary change and the associated change in gut size were necessarily "prime movers" in hominid encephalization. As Falk has clearly stated, we view these factors, for the most part, as "prime releasers" which make available the not inconsiderable energy resources that are a necessary concomitant of encephalization.

Holloway comments that the expensive-tissue hypothesis does not get us any nearer to understanding the relationship between brains and behaviour, but that was not our intention. Nonetheless, Milton has proposed that increased complexity of foraging behaviour associated with the change in diet could be a "prime mover" for the increase in brain size in primates, a view also expressed in other guises by, for example, Parker and Gibson (1979; Gibson 1986) and Clutton-Brock and Harvey (1980). Milton's comments here give the impression that we reject this view; on the contrary, it was not our intention to come down on the side of any one of the various hypotheses that are current in the literature. Indeed, in figure 5 we clearly indicate that there may have been a causal connection between more complex foraging strategies and brain size increase, but we also realize that the causal nature of such relationships is always difficult to determine. We certainly do not want to give the impression that we are suggesting that gut changes should be viewed "apart from the foraging strategy and dietary niche in which they are evolutionarily embedded." But we also want to make clear that our hypothesis does not require dietary change to be the "prime mover" or even one of the "prime movers" for encephalization. It clearly would also be compatible with group-size hypotheses (Dunbar 1992, 1993; Aiello and Dunbar 1993), social-intelligence hypotheses (Byrne and Whiten 1988), or, perhaps more realistic, a combination of causes. Our main point is that the reduction in gut size is a concomitant of a change to higher-quality diets. The lower energy requirements of smaller guts were a releaser that energetically permitted an associated increase in the size of the brain.

Although we consider the relationship between a high-quality diet and and a relatively small gut to be an important concomitant of encephalization, we also do not want to give the impression that it is the only prime releaser. Other factors, anatomical as well as energetic, have almost certainly constrained brain size during hominid evolution. For example, as is noted by Falk, the problem of supplying the brain with the high levels of chemical energy it requires is intimately linked with that of removing the resultant heat from this extremely temperature-sensitive organ. In this context, it has been proposed that the thermal protection provided by a naked skin and its associated sweat glands stabilising the temperature of the arterial blood supply to the brain (Wheeler 1984) and the elaboration of emissary veins affording cooling to the delicate outer layer of the cortex (Falk 1990) were crucial factors in allowing the expansion of the brain that has taken place during the evolution of the genus *Homo*.

A key point raised in detail by some of the commentators (Hennenberg, Wrangham et al.) is whether whole-body BMR is constrained to the extent that encephalization would require the compensatory reduction in size of another metabolically expensive organ. This is a valid question. It is widely assumed that the total energy available to and utilized by organisms is an important limiting factor to survival and reproductive success. Indeed, this forms the basis of much current evolutionary and ecological theory. Consequently, we have endeavoured to show that the cost of encephalization is a significant component not only of BMR itself but also of the total energy budget of humans. The extra cost for humans appears to be in the range of a 5% increase in total metabolic energy requirements. We would argue, contra Henneberg and Wrangham et al., that this value is not insignificant. An individual with an increased energy budget will be at a significant disadvantage in terms of competition and reproductive success.

As we indicated, we concur with these commentators that, in theory at least, the cost of the additional brain tissue could have been met by strategies other than a reduction in gut size. For example, if sufficient dietary resources were available, overall BMR could have been correspondingly increased and/or the energetic costs associated with other components of the energy budget reduced. What strategy would maximise the reproductive success of organisms would depend on the overall ecological context in which they lived. It is quite possible that other taxonomic groups have solved the problem of the energetic costs associated with encephalization in other ways than by a reduction in the size of the gut. For example, it is possible at least in part that the cost of the larger than average brains of mustelids (Jerison 1990) is reflected in their higher than average BMR (Wheeler 1984, Iverson 1972); whether there are also corresponding reductions in the sizes of other expensive organs is currently unknown. Several commentators (Armstrong, Falk, Wrangham et al.) note the potential value of extending the analyses to other taxonomic groups (e.g., cetaceans, bats, birds), and we recognize that, where adequate data sets can be obtained, this is fertile ground for much future research.

Regardless of what strategies are ultimately shown to have been adopted by other groups and could *potentially* have been used by hominids, our central point remains valid; humans possess a relatively large brain and a relatively small gut and also have no corresponding increase in BMR. Consequently, there is no need to look for alternative explanations (such as reduced activity, increased sleep, and reduced dietary intake, as suggested by Henneberg and Wrangham et al.) for how the energetic costs associated with human encephalization have been met. In humans both organ weight and *in vivo* organ metabolic data strongly support the hypothesis that the increased metabolic cost of the large human brain was met specifically by a decrease in the size of the gastrointestinal tract. The conclusion that can be drawn from this is

that other solutions were not as viable in the adaptive context that confronted our evolutionary ancestors. But our data also suggest that some of the other factors may be important in offsetting the costs of encephalization in other primates. Although there is a strong inverse relationship between relative brain size and relative gut size across primates (fig. 4), there is also some evidence for grade relationships in these data. For example, the colobines have a relatively smaller combined brain and gut mass for their total body mass than other primates, and this could be a reflection of a lower than average BMR.

Some of the commentators express concern over the quality of the data used in this analysis (Henneberg, Wrangham et al., Milton). We fully recognize that the data set is not ideal. In the interspecific primate comparison we have clearly noted the problems with the data but nonetheless have been impressed by the negative relationship between relative brain size and relative gut size. At the same time we have specifically avoided taking interpretations of this relationship to too fine a level of detail. Perhaps a more serious concern is potential variation in the data due to real differences in organ sizes and body masses within humans and other primate taxa. To our knowledge data sets do not currently exist which would allow us to test the intraspecific variation in BMR and relative organ size. However, it should be recognized that the expensive-tissue hypothesis rests squarely on the existence of such variation. In particular, within species we would expect that encephalized individuals deviating from the ideal brain/gut-size relationships would also deviate in other aspects of their energy budgets. Depending on the environmental conditions in which they found themselves, we would further expect that this deviation would have had adverse consequences for their reproductive success. On the individual level this would be the selective mechanism driving the observed between-taxa relationships.

In relation to data quality, Henneberg also notes the high variability in the mass-specific metabolic rates for individual organs across species (tables 1 and 3) and accordingly questions the reliability of our resulting estimates of the metabolic balance in table 4. It should be noted, however, that the highly variable *in vitro* data (table 3) were presented for illustrative purposes only, allowing the relative costs of the different tissues to be compared *within* a species. It is important to emphasize that the quantitative analysis which shows that the metabolic cost of the human brain is balanced by the reduced metabolic cost of the small human gut (table 4) was derived exclusively from *in vivo* measurements of human subjects (table 1). Possible interspecific variation in mass-specific metabolic cost does not bear on this conclusion.

Wrangham et al. also wonder how we reconcile our conclusions with the different organ allometries, particularly brain and gut allometries, in mammals, birds, and reptiles. We do not have the data at present to answer this question, but table 5 suggests that birds, at least, have adopted a very different energy strategy from primates. For example, the expensive tissues of an average 500-g bird make up a smaller percentage of body mass than do the same tissues in a similarly sized primate. The bird has a much smaller brain than a similarly sized primate and also a smaller gut while having considerably larger kidneys and heart. These relationships obtain throughout the relevant body-mass ranges. If these size relationships mirror the actual metabolic costs of the tissues in birds as they do in humans, we can postulate that the demands of the very different lifestyle and locomotor pattern of birds govern their different organ allometries. The principle is the same, however. All organisms have to accommodate the relative costs of their expensive tissues within both their BMR and their total energy budgets. It is highly possible that the energetic demands of flight, as well as the effect of these demands on, for example, the size of the heart, have precluded any degree of encephalization comparable to that found in primates.

TABLE 5
Mass (g) of the Expensive Tissues for a 500-g Primate and a Bird

	Primate	Bird	Bird/Primate
Heart	2.62	4.26	1.63
Liver	16.78	17.97	1.07
Kidney	3.45	4.62	1.34
Brain	13.59	4.75	0.35
Gut	29.66	3.54	0.12
ET/BM	0.13	0.07	
B&G/ET	0.65	0.24	
B&G/BM	0.09	0.02	

NOTE: ET/BM = mass of the expensive tissues as a proportion of body mass; B&G/ET = mass of the brain and gut as a proportion of the mass of the expensive tissues; B&G/BM = mass of the brain and gut as a proportion of body mass; primate predictions based on the equations presented here, bird predictions on equations in Peters (1983).

Wrangham et al. also raise the interesting question of why among the expensive organs only brain mass, like BMR, scales with an exponent significantly less than unity. However, the important relationships are actually those between body size and the total metabolic costs of the expensive organs, not just their masses. In the case of the brain the metabolic rate and the organ mass scale with rather similar exponents (0.69 and 0.76, respectively), since the mass-specific metabolic rate of this organ is not strongly related to body size (Grande 1980). In contrast, although the size of other expensive organs may scale with exponents closer to 1, their mass-specific metabolic rates may decline more rapidly with increasing body size, also resulting in an overall exponent for the total metabolic cost of the organ close to the 0.75 of BMR itself. For example, liver mass scales with an exponent of about 0.87 (Peters 1983), but its mass-specific metabolic rate exponent is −0.12 (Grande 1980), giving a combined exponent of 0.75 for the total

energetic cost of this organ. However, whether the allometric relationships of gut size and metabolism follow a similar pattern to those of the liver is currently uncertain because of the lack of good data sets relating mass-specific tissue metabolic rate to body size.

Armstrong further raises the question of substrate utilization patterns by the brain and other organs and their relationship to our analysis. Specifically, she asks whether glycogen is stored in gut tissue and, if so, whether this significantly adds to the mass of the tissue and thereby confounds the analysis. We do not feel that this is a relevant concern. Our analysis is based on rates of energy utilization by the various organs expressed as the mass-specific organ metabolic rate. Glycogen would be metabolically inactive, and if it were stored in the gut its weight would be taken care of in the computation of the mass-specific metabolic rate for that tissue. The analysis would therefore not be influenced by the specific substrates being metabolized.

Some commentators (Falk, Marchant, Holloway, Henneberg) have brought up points that are specifically relevant to hominid evolution. Falk has argued that brain expansion in the hominids may have been gradual rather than punctuated after the appearance of *Homo;* Marchant suggests that early *Homo,* particularly *H. habilis sensu stricto,* may not have had a higher-quality diet than the australopithecines, and both Holloway and Henneberg point out that an increase in absolute brain size in the hominids may accompany an increase in body size or may be independent of it. These are all evolutionary details that are subject to debate stemming from the ambiguity of relatively poor data. They do not affect the basic issue that when the brain expanded *in relation to body size* the energetic balance would have had to be adjusted. The question that is still open is when in our evolutionary history this may have happened. We suggest that the change in body proportions in *H. ergaster* in relation to the australopithecines may have marked a major shift to a higher-quality food and correspondingly smaller guts. The fact that the change in the shape of the rib cage in *Homo* may also be associated with the adoption of fully bipedal locomotion (Marchant) does not seriously affect this notion. Our point is that in relation to body size the space available in the australopithecine pelvic region (specifically in *Australopithecus afarensis* and *A. africanus*) would accommodate larger guts in relation to body size than the corresponding space in *H. ergaster.*

We feel that the evidence provided to support the expensive-tissue hypothesis is sufficient to show that in humans and primates there has been a coevolution of the brain and the digestive system. We do not claim that a reduction in the size of the gut is the only way to balance the high energy requirements of a relatively large brain; rather, we suggest that it is the most probable means by which humans have accomplished this. There is no doubt that the hypothesis would benefit by further development and testing, and we hope that this contribution will have stimulated others to follow its lead.

References Cited

AIELLO, L. C. 1992. Allometry and the analysis of size and shape in human evolution. *Journal of Human Evolution* 22:127–47.

AIELLO, L. C., AND M. C. DEAN. 1990. *An introduction to human evolutionary anatomy.* London: Academic Press.

AIELLO, L. C., AND R. I. M. DUNBAR. 1993. Neocortex size, group size, and the evolution of language in the hominids. CURRENT ANTHROPOLOGY 34:184–93.

ARMSTRONG, E. 1982. A look at relative brain size in mammals. *Neuroscience Letters* 34:101–4.

———. 1983. Relative brain size and metabolism in mammals. *Science* 220:1302–4.

———. 1985*a*. Relative brain size in monkeys and prosimians. *American Journal of Physical Anthropology* 66:263–73.

———. 1985*b*. "Allometric considerations of the adult mammalian brain, with special emphasis on primates," in *Size and scaling in primate biology.* Edited by W. L. Jungers, pp. 115–46. New York: Plenum Press.

———. 1990. Brains, bodies, and metabolism. *Brain, Behaviour, and Evolution* 36:166–76.

ARMSTRONG, ESTE, AND R. BERGERON. 1985. Relative brain size in birds. *Brain, Behavior, and Evolution* 26:141–53. [EA]

ASCHOFF, J., B. GÜNTHER, AND K. KRAMER. 1971. *Energiehaushalt und Temperaturregulation.* Munich: Urban and Schwarzenberg.

BARTON, R. A. 1992. Allometry of food intake in free-ranging anthropoid primates. *Folia Primatologica* 58:56–59.

BERTALANFFY, L. VAN, AND R. R. EASTWICK. 1953. Tissue respiration of musculature in relation to body size. *American Journal of Physiology* 173:58–60.

BLAXTER, K. 1989. *Energy metabolism in animals and man.* Cambridge: Cambridge University Press.

BLIGH, J., AND K. G. JOHNSON. 1973. Glossary of terms for thermal physiology. *Journal of Applied Physiology* 35:941–60.

BRODY, S. 1945. *Bioenergetics and growth.* Baltimore: Reinhold.

BUNN, H. T., AND E. M. KROLL. 1986. Systematic butchery by Plio/Pleistocene hominids at Olduvai Gorge, Tanzania. CURRENT ANTHROPOLOGY 27:531–42.

BYRNE, R., AND A. WHITEN. Editors. 1988. *Machiavellian intelligence.* Oxford: Clarendon.

CHIVERS, D. J., AND C. M. HLADIK. 1980. Morphology of the gastrointestinal tract in primates: Comparisons with other mammals in relation to diet. *Journal of Morphology* 166:337–86.

———. 1984. "Diet and gut morphology in primates," in *Food acquisition and processing in primates.* Edited by D. J. Chivers, B. A. Wood, and A. Bilsborough, pp. 213–30. New York: Plenum Press.

CLUTTON-BROCK, T. H., AND P. H. HARVEY. 1980. Primates, brains, and ecology. *Journal of Zoology, London* 190:309–23.

CRAWFORD, M. A. 1992. The role of dietary fatty acids in biology: Their place in the evolution of the human brain. *Nutrition Reviews* 50:3–11.

CROCKETT, C. M., AND J. F. EISENBERG. 1987. "Howlers: Variations in group size and demography," in *Primate societies.* Edited by D. L. Cheney, R. M. Seyfarth, R. W. Wrangham, and T. T. Struhsaker, pp. 54–68. Chicago: University of Chicago Press.

DEMMENT, M. 1983. Feeding ecology and the evolution of body size in baboons. *African Journal of Ecology* 21:219–33.

DUNBAR, R. I. M. 1992. Neocortex size as a constraint on group size in primates. *Journal of Human Evolution* 22:469–93.

———. 1993. Co-evolution of neocortex size, group size, and language in humans. *Behavioral and Brain Sciences* 16:681–735.

———. n.d. Neocortex size and group size in primates: A test of the hypothesis. *Journal of Human Evolution.* In press.

FALK, DEAN. 1987. Hominid paleoneurology. *Annual Review of Anthropology* 16:13–30. [DF]

———. 1990. Brain evolution in *Homo:* The "radiator" theory. *Behavioral and Brain Sciences* 13:333–81.

———. 1992. *Evolution of the brain and cognition in hominids (The Sixty-Second James Arthur Lecture).* New York: American Museum of Natural History. [DF]

FAO/WHO/UNU. 1985. *Energy and protein requirements: Report of a joint expert consultation.* (WHO Technical Report Series 724.) Geneva: World Health Organisation.

FIELD, J., H. S. BELDING, AND A. W. MARTIN. 1939. An analysis of the relation between basal metabolism and summated tissue respiration in the rat. I. The post-pubertal albino rat. *Journal of Cellular and Comparative Physiology* 14:143–57.

FISHER, R. A. 1930. *The genetical theory of natural selection.* Oxford: Oxford University Press. [MH]

FOLEY, ROBERT. 1991. How many species of hominid should there be? *Journal of Human Evolution* 20:413–29. [LFM]

FOLEY, R. A., AND P. C. LEE. 1991. Ecology and energetics of encephalization in hominid evolution. *Philosophical Transactions of the Royal Society, London* B 334:223–32.

FORBES, R. M., A. R. COOPER, AND H. H. MITCHELL. 1953. The composition of the adult human body as determined by chemical analysis. *Journal of Biological Chemistry* 302:359–66.

FORD, T. J. AND S. D. KRAUS. 1992. A rete in the right whale. *Nature* 359:680. [DF]

FRAYER, D. 1984. "Biological and cultural change in the European Late Pleistocene and Early Holocene," in *The origins of modern humans: A world survey of the fossil evidence.* Edited by F. H. Smith and F. Spencer, pp. 211–50. New York: Alan R. Liss. [MH]

GIBSON, K. R. 1986. "Cognition, brain size, and the extraction of embedded food resources," in *Primate ontogeny, cognition, and social behavior.* Edited by J. G. Else and P. C. Lee, pp. 93–105. Cambridge: Cambridge University Press.

GRANDE, F. 1980. "Energy expenditure of organs and tissues," in *Assessment of energy metabolism in health and disease.* Edited by S. Calvert, pp. 88–92. Columbus: Ross Laboratories.

GROSS, J., Z. WANG, AND B. A. WUNDER. 1986. Adaptations to food quality and energy need changes in gut morphology and capacity of *Microtus orchrogaster. Journal of Mammalogy* 66:661–69.

HARVEY, R. H., R. D. MARTIN, AND T. H. CLUTTON-BROCK. 1986. "Life histories in comparative perspective," in *Primate societies.* Edited by B. B. Smuts, D. L. Cheney, R. M. Seyfarth, R. W. Wrangham, and T. T. Struhsaker, pp. 181–96. Chicago: University of Chicago Press.

HENNEBERG, M. 1990. Brain size/body weight variability in *Homo sapiens:* Consequences for interpreting hominid evolution. *Homo* 39:121–30. [MH]

HOFMAN, M. A. 1983. Energy metabolism, brain size, and longevity in mammals. *Quarterly Review of Biology* 58:495–512.

HOLLOWAY, R. L. 1979. "Brain size, allometry, and reorganization: Toward a synthesis," in *Development and evolution of brain size: Behavioral implications.* Edited by M. E. Hahn, C. Jensen, and B. Dudek, pp. 59–88. New York: Academic Press. [RLH]

———. 1980. Within-species brain-body weight variability: A reexamination of the Danish data and other primate species. *American Journal of Physical Anthropology* 53:109–21. [RLH]

HUNT, KEVIN D. 1994. The evolution of human bipedality: Ecology and functional morphology. *Journal of Human Evolution* 26:183–202. [LFM]

HUYSSEN, V., AND R. C. LACY. 1985. Basal metabolic rates in mammals: Taxonomic differences in the allometry of SMR and body mass. *Comparative Biochemistry and Physiology* 81A:741–54.

IVERSEN, J. A. 1972. Basal energy metabolism of mustelids. *Journal of Comparative Physiology* 81A:341–44.

JACOBS, K. H. 1985. Climate and hominid postcranial skeleton in Würm and Early Holocene Europe. CURRENT ANTHROPOLOGY 26:512–14. [MH]

JERISON, H. J. 1973. *Evolution of the brain and intelligence.* New York: Academic Press. [MH, KM]

———. 1990. Welcome light on a hot topic. *Behavioral and Brain Sciences* 13:360.

JOHANSON, DONALD C., ET AL. 1987. New partial skeleton of *Homo habilis* from Olduvai Gorge, Tanzania. *Nature* 327:205–9. [LFM]

KARASOV, W. H. 1992. Daily energy expenditure and the cost of activity in mammals. *American Zoologist* 32:238–48.

KAY, R. F., AND F. E. GRINE. 1988. "Tooth morphology, wear, and diet in *Australopithecus* and *Paranthropus,*" in *Evolutionary history of the "robust" australopithecines.* Edited by F. E. Grine, pp. 427–48. New York: Aldine de Gruyter.

KEELEY, L. M., AND N. TOTH. 1981. Microwear polishes on early stone tools from Koobi Fora. *Nature* 297:676–78.

KEITH, A. 1891. Anatomical notes on Malay apes. *Proceedings of the Singapore Branch of the Royal Asiatic Society* 23:77–89.

———. 1950. *An autobiography.* New York: Philosophical Library.

KLEIBER, M. 1961. *The fire of life.* New York: Wiley.

KREBS, H. A. 1950. Body size and tissue respiration. *Biochimica et Biophysica Acta* 4:249–69.

LAMB, D. R. 1984. *Physiology of exercise: Responses and adaptations.* New York: Macmillan. [MH]

LEE-THORP, J. A., N. J. VAN DER MERWE, AND C. K. BRAIN. 1994. Diet of *Australopithecus robustus* at Swartkrans from stable carbon isotopic analysis. *Journal of Human Evolution* 27:361–72.

LEHNINGER, A. L. 1975. *Biochemistry.* New York: Worth.

LEIGH, S. R. 1992. Cranial capacity evolution in *Homo erectus* and early *Homo sapiens. American Journal of Physical Anthropology* 87:1–14.

MC HENRY, H. 1994. Behavioral ecological implications of early hominid body size. *Journal of Human Evolution* 27:77–87.

MAC LARNON, A. M., D. J. CHIVERS, AND R. D. MARTIN. 1986*a*. "Gastro-intestinal allometry in primates including new species," in *Primate ecology and conservation.* Edited by J. G. Else and P. C. Lee, pp. 75–85. Cambridge: Cambridge University Press.

———. 1986*b*. "Some aspects of gastro-intestinal allometry in primates and other mammals," in *Définition et origines de l'homme.* Edited by M. Sakka, pp. 293–302. Paris: Editions du CNRS.

MAC NAB, B. K., AND J. F. EISENBERG. 1989. Brain size and its relation to the rate of metabolism in mammals. *American Naturalist* 133:157–67.

MARTIN, A. W., AND F. A. FUHRMAN. 1955. The relationship between summated tissue respiration and metabolic rate in the mouse and dog. *Physiological Zoology* 28:18–34.

MARTIN, R. D. 1981. Relative brain size and metabolic rate in terrestrial vertebrates. *Nature* 293:57–60.

———. 1983. *Human brain evolution in an ecological context: 52nd James Arthur Lecture on the Evolution of the Human Brain.* New York: American Museum of Natural History.

———. 1990. *Primate origins and evolution.* London: Chapman and Hall.

MARTIN, R. D., D. J. CHIVERS, A. M. MAC LARNON, AND C. M. HLADIK. 1985. "Gastrointestinal allometry in primates and other mammals," in *Size and scaling in primate biology.* Edited by W. L. Jungers, pp. 61–89. New York: Plenum Press.

MILTON, K. 1979. Spatial and temporal patterns of plant foods in tropical forests as a stimulus to intellectual development in primates (abstract). *American Journal of Physical Anthropology* 50:464–65.

———. 1981. Diversity of plant foods in tropical forests as a stimulus to mental development in primates. *American Anthropologist* 83:534–48. [KM].

———. 1986. Features of digestive physiology in Primates. *News in Physiological Sciences* 1:76–79.

———. 1987. "Primate diets and gut morphology: Implications for hominid evolution," in *Food and evolution: Toward a theory of human food habits.* Edited by M. Harris and E. B. Ross, pp. 96–116. Philadelphia: Temple University Press.

———. 1988. "Foraging behaviour and the evolution of primate intelligence," in *Machiavellian intelligence.* Edited by R. Byrne and A. Whiten, pp. 285–409. Oxford: Oxford Science Publications.

———. 1993. Diet and primate evolution. *Scientific American* 269(2):86–93.

———. n.d. "Food and diet," in *The encyclopedia of cultural anthropology*. Edited by D. Levinson and M. Ember. Lakeville, Conn.: American Reference Publishing. In press. [KM].

MILTON, K., AND M. DEMMENT. 1988. Digestive and passage kinetics of chimpanzees fed high and low fiber diets and comparison with human data. *Journal of Nutrition* 118:285–305.

MITCHELL, H. H., T. S. HAMILTON, F. R. STEGGERDA, AND H. W. BEAN. 1945. The chemical composition of the adult human body and its bearing on the biochemistry of growth. *Journal of Biological Chemistry* 158:625–37.

OIKAWA, S., AND Y. ITAZOWA. 1984. Allometric relationship between tissue respiration and body mass in the carp. *Comparative Biochemistry and Physiology* 77A:415–18.

PARKER, S. T., AND K. R. GIBSON. 1979. A developmental model for the evolution of language and intelligence in early hominids. *Behavioral and Brain Sciences* 2:367–407.

PETERS, R. H. 1983. *The ecological implications of body size*. Cambridge: Cambridge University Press.

POCZOPKO, P. 1971. Metabolic levels in adult homeotherms. *Acta Theriologica* 16:1–21.

POTTS, R., AND P. SHIPMAN. 1981. Cutmarks made by stone tools on bones from Olduvai Gorge, Tanzania. *Nature* 291:577–80.

RICHARDS, AUDREY. 1948. *Hunger, work, and sex in a savage tribe*. Westport: Greenwood Press. [KM]

RIGHTMIRE, G. P. 1981. Patterns in the evolution of *Homo erectus*. *Paleobiology* 7:241–46.

ROBINSON, J. T. 1972. *Early hominid posture and locomotion*. Chicago: University of Chicago Press.

RUFF, C. B. 1991. Climate and body shape in hominid evolution. *Journal of Human Evolution* 21:81–105.

RUFF, C. B., AND A. WALKER. 1993. "Body size and body shape," in *The Nariokotome* Homo erectus *skeleton*. Edited by A. Walker and R. E. Leakey. Cambridge: Harvard University Press.

SCHMID, P. 1983. Eine Rekonstruktion des Skelettes von A.L. 288-1 (Hadar) und deren Konsequenzes. *Folia Primatologica* 40:283–306.

———. 1991. "The trunk of the australopithecines," in *Origine(s) de la bipédie chez les Hominidés*. Edited by Y. Coppens and B. Senut, pp. 225–34. Paris: Editions du CNRS.

SCHMIDT-NIELSEN, KNUT. 1984. *Scaling: Why is animal size so important?* Cambridge: Cambridge University Press.

SCHOFIELD, C. 1985. An annotated bibliography of source material for basal metabolic rate data. *Human Nutrition: Clinical Nutrition* 39C, suppl. 1:42–91.

SCHOFIELD, W. N. 1985. Predicting basal metabolic rate: New standards and review of previous work. *Human Nutrition: Clinical Nutrition* 39C, suppl. 1:5–41.

SHIPMAN, P. 1986. Scavenging or hunting in early hominids. *American Anthropologist* 88:27–43.

SHIPMAN, P., AND A. WALKER. 1989. The costs of becoming a predator. *Journal of Human Evolution* 18:373–92.

SILLEN, A. 1992. Strontium-calcium ratios (Sr/Ca) of *Australopithecus robustus* and associated fauna from Swartkrans. *Journal of Human Evolution* 23:495–516.

SILLEN, A., R. ARMSTRONG, AND G. HALL. n.d. Strontium-calcium ratios (Sr/Ca) and $^{87}Sr/^{86}Sr$ of *Australopithecus robustus* and *Homo* sp. from Swartkrans. *Journal of Human Evolution*. In press.

SPETH, J. D. 1989. Early hominid hunting and scavenging: The role of meat as an energy source. *Journal of Human Evolution* 18:329–44.

STAHL, A. B. 1984. Hominid dietary selection before fire. CURRENT ANTHROPOLOGY 25:151–68.

STAHL, W. R. 1965. Organ weights in primates and other mammals. *Science* 150:1039–42.

STANFORD, CRAIG B., JANETTE WALLIS, HILALI MATAMA, AND JANE GOODALL. 1994. Patterns of predation by chimpanzees on red colobus monkeys in Gombe National Park, 1982–1991. *American Journal of Physical Anthropology* 94:213–28. [LFM]

STEPHAN, H., H. FRAHM, AND G. BARON. 1981. New and revised data on volume of brain structures in insectivores and primates. *Folia Primatologica* 35:1–29.

SUSSMAN, R. W. 1987. "Morpho-physiological analysis of diets," in *The evolution of human behavior: Primate models*. Edited by W. G. Kinzey, pp. 151–79. Albany: State University of New York Press.

SNYDER, W. S. 1975. *International Commission on Radiological Protection (ICRP) report on the Task Force on Reference Man*. Oxford: Oxford University Press.

TAGUE, R. G., AND C. O. LOVEJOY. 1986. The obstetric pelvis of A.L. 288-1 (Lucy). *Journal of Human Evolution* 15:237–55.

TRINKAUS, E., AND M. H. WOLPOFF. n.d. Brain size in posthabiline archaic *Homo*. MS.

VAN SOEST, P. J., T. FOOSE, J. JERACI, K. WRICK, AND F. EHLE. 1982. Comparative fermentation of fibre in man and other animals. Paper presented to the International Symposium on Dietary Fiber, Palmerston North, New Zealand, May 22–28.

WHEELER, P. E. 1984. An investigation of some aspects of the transition from ectothermic to endothermic metabolism in vertebrates. Ph.D. diss., University of Durham, Durham, U.K.

———. 1988. Stand tall and stay cool. *New Scientist* 12:62–65. [DF]

———. 1991. The influence of bipedalism on the energy and water budgets of early hominids. *Journal of Human Evolution* 21:107–36.