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Neural Capital and Life span Evolution among Primates and Humans

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Introduction

This paper presents a theory of brain and life span evolution and applies it to both the primate order, in general, and to the hominid line, in particular. To address the simultaneous effects of natural selection on the brain and on the life span, it extends standard life history theory (LHT) in biology, which organizes research into the evolutionary forces shaping age-schedules of fertility and mortality (Cole 1954; Gadgil and Bossert 1970; Partridge and Harvey 1985). This extension, *the embodied capital theory* (Kaplan and Robson 2001 b; Kaplan 1997; Kaplan et al. 2000), integrates existing models with an economic analysis of capital investments and the value of life.

The chapter begins with a brief introduction to embodied capital theory and then applies it to understanding major trends in primate evolution and the specific characteristics of humans. The evolution of brain size, intelligence and life histories in the primate order are addressed first. The evolution of human life course is then considered, with a specific focus on the relationship between cognitive development, economic productivity and longevity. It will be argued that the evolution of the human brain entailed a series of co evolutionary responses in human development and aging, resulting in a highly structured species-typical life span that can vary within a limited range.

The Embodied Capital Theory of Life History Evolution

According to the theory of evolution by natural selection, the evolution of life is the result of a process in which variant forms compete to harvest energy from the environment and convert that energy into replicates of those forms. Those forms that can capture more energy than others and can convert the energy they acquire more efficiently into replicates than others become more prevalent through time. This simple issue of harvesting energy and converting energy into offspring generates many complex problems that are time-dependent.

Two fundamental tradeoffs determine the action of natural selection on reproductive schedules and mortality rates. The first tradeoff is between current and future reproduction. By growing, an organism can increase its energy capture rates in the future and thus increase its future fertility. For this reason,

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organisms typically have a juvenile phase in which fertility is zero until they reach a size at which some allocation to reproduction increases lifetime fitness more than growth. Similarly, among organisms that engage in repeated bouts of reproduction (humans included), some energy during the reproductive phase is diverted away from reproduction and allocated to maintenance so that they can live to reproduce again. Natural selection is expected to optimize the allocation of energy to current reproduction and to future reproduction (via investments in growth and maintenance) at each point in the life course so that genetic descendents are maximized (Gadgil and Bossert 1970). Variation across taxa and across conditions in optimal energy allocations is shaped by ecological factors, such as food supply, disease and predation rates.

A second fundamental life history tradeoff is between offspring number (quantity) and offspring fitness (quality). This tradeoff occurs because parents have limited resources to invest in offspring and each additional offspring produced necessarily reduces average investment per offspring. Most biological models (Lack 1954; Lloyd 1987; Smith and Fretwell 1974) operationalize this tradeoff as number vs. survival of offspring. However, parental investment may affect not only survival to adulthood but also the adult productivity and fertility of offspring. This is especially true of humans. Thus, natural selection is expected to shape investment per offspring and offspring number so as to maximize offspring number times their average lifetime fitness.

The embodied capital theory generalizes existing LHT by treating the processes of growth, development and maintenance as investments in stocks of somatic or embodied capital. In a physical sense, embodied capital is organized somatic tissue – muscles, digestive organs, brains, etc. In a functional sense, embodied capital includes strength, speed, immune function, skill, knowledge and other abilities. Since such stocks tend to depreciate with time, allocations to maintenance can also be seen as investments in embodied capital. Thus, the present-future reproductive trade-off can be understood in terms of optimal investments in own embodied capital vs. reproduction, and the quantity quality tradeoff can be understood in terms of investments in the embodied capital of offspring vs. their number.

The Brain as Embodied Capital

The brain is a special form of embodied capital. Neural tissue is involved in monitoring the organism's internal and external environment and organizing physiological and behavioral adjustments to those stimuli (Jerison 1976). Portions (particularly the cerebral cortex) are also involved in transforming present experiences into future performance. Cortical expansion among higher primates, along with enhanced learning abilities, reflects increased investment in transforming present experience into future performance (Armstrong and Falk 1982; Fleagle 1999).

The action of natural selection on neural tissue involved in learning and memory should depend on costs and benefits realized over the organism's life-

time. Three kinds of costs are likely to be of particular importance. First, there are the initial energetic costs of growing the brain. Among mammals, those costs are largely born by the mother. Second, there are the energetic costs of maintaining neural tissue. Among infant humans, about 65% of all resting energetic expenditure supports maintenance and growth of the brain (Holliday 1978). Third, certain brain capacities may actually decrease performance early in life. Specifically, the ability to learn and increased behavioral flexibility may entail reductions in "pre-programmed" behavioral routines. The incompetence with which human infants and children perform many motor tasks is an example.

Some allocations to investments in brain tissue may provide immediate benefits (e.g., perceptual abilities, motor coordination). Other benefits of brain tissue are only realized as the organism ages. The acquisition of knowledge and skills has benefits that, at least in part, depend on their impact on future productivity. Figure 1 illustrates two alternative cases, using as an example the difficulty and learning-intensiveness of the organism's foraging niche. In the easy feeding niche, where there is little to learn and little information to process, net productivity (excess energy above and beyond maintenance costs of brain and body) reaches its asymptote early in life. There is a relatively small impact of the brain on productivity late in life (because there is little to learn), but there are higher costs of the brain early in life. Unless the life span is exceptionally long, natural selection will favor the smaller brain.

In the difficult food niche, the large-brain creature is slightly worse off than the small-brain one early in life (because the brain is costly and learning is taking place), but much better off later in life. The effect of natural selection will depend upon the probabilities of reaching the older ages. If those probabilities are sufficiently low, the small brain will be favored; if they are sufficiently high, the large brain will be favored. Thus, selection on learning-based neural capital

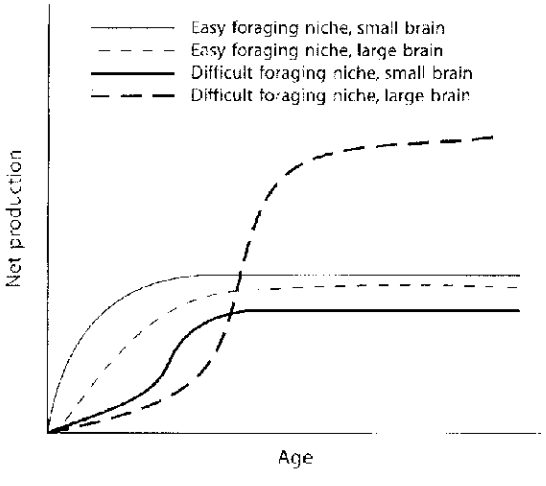

Fig. 1. Age specific effects of brains on net production: easy and difficult foraging niches

depends not only on its immediate costs and benefits but also upon mortality schedules that affect the expected gains in the future.

Selection on Mortality Schedules

In standard LHT models, mortality is generally divided into two types: 1) extrinsic mortality (i.e., mortality, such as predation or winter, that is imposed by the environment and is outside the control of the organisms) and 2) intrinsic mortality (hazards of mortality over which the organism can exert some control over the short run or that are subject to selection over the longer periods). In most models of growth and development, mortality is treated as extrinsic (Charnov 1993; Kozlowski and Wiegert 1986) and therefore as a causal agent, not subject to selection. Models of aging and senescence (Promislow 1991; Shanley and Kirkwood 2000) typically focus on aging-related increases in intrinsic mortality. Extrinsic mortality, in turn, is thought to affect selection on rates of aging, with higher mortality rates favoring faster aging.

This distinction between types of mortality is problematic. Organisms can exert control over virtually all causes of mortality in the short or long run. Susceptibility to predation can be affected by vigilance, choice of foraging zones, travel patterns and anatomical adaptations, such as shells, cryptic coloration and muscles facilitating flight. Each of those behavioral and anatomical adaptations has energetic costs (lost time foraging, investments in building and maintaining tissue) that reduce energy available for growth and reproduction. Similar observations can be made regarding disease and temperature. The extrinsic mortality concept has been convenient because it has provided a causal agent for examining other life history traits, such as age of first reproduction and rates of aging. However, this has prevented the examination of how mortality rates themselves evolve by natural selection.

Since all mortality is, to some extent, intrinsic or "endogenous", a more useful approach is to examine the functional relationship between mortality and effort allocated to reducing it (see Fig. 2). Exogenous variation can be thought of in terms of varying "assault" types and varying "assault" rates of mortality hazards. For example, warm, humid climates favor the evolution of disease organisms and therefore increase the assault rate and diversity of diseases in organisms living in those climates. Such exogenous variation would affect the functional relationship between actual mortality hazards and endogenous effort allocated to reducing it. The outcome mortality rate is neither extrinsic nor intrinsic.

Kaplan and Robson (2001 a, b) developed formal models to analyze the simultaneous effects of natural selection on both investments in capital and in reducing mortality. As a first step, it is useful to think of capital generally (interpreted as the bundle of functional abilities embodied in the soma). Organisms generally receive some energy from their parents (e.g., in the form of energy stored in eggs) to produce an initial stock of capita. Net energy acquired from the environment at each age grows as a function of the capital stock, with

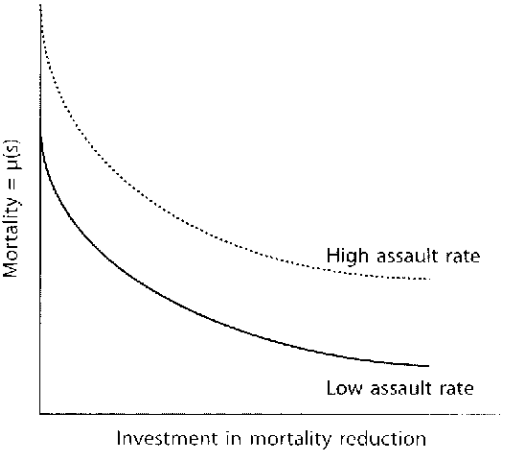

Fig. 2. Mortality rate as a function of investments

diminishing returns to capital (as illustrated in Fig. 3). This energy can be used in three ways that are endogenous and subject to selection. It can be reinvested in increasing the capital stock (e.g., growth of the body or brain). Some energy may also be allocated to reducing mortality (for example in the form of increased immune function, as illustrated above in Fig. 2). The probability of reaching any age will be a function of mortality rates at each earlier age. Finally, energy can also be used for reproduction, which is the net excess energy available after allocations to capital investments and mortality reduction. An optimal life history program would optimize allocations to capital investments, mortality reduction, and reproduction at each age so as to maximize total energy allocations to reproduction over the life course. This, of course, depends both on reproductive allocations and on survival.

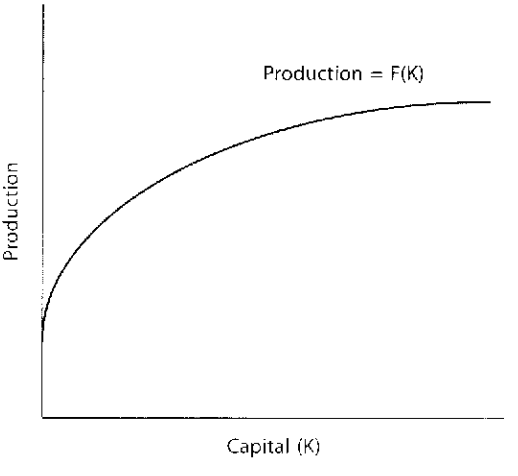

Fig. 3. Production as a function of the capital stock. At each point in time, an individual has an embodied capital stock, K, that produces a stream of energy output, $F(K)$. The stock grows during development

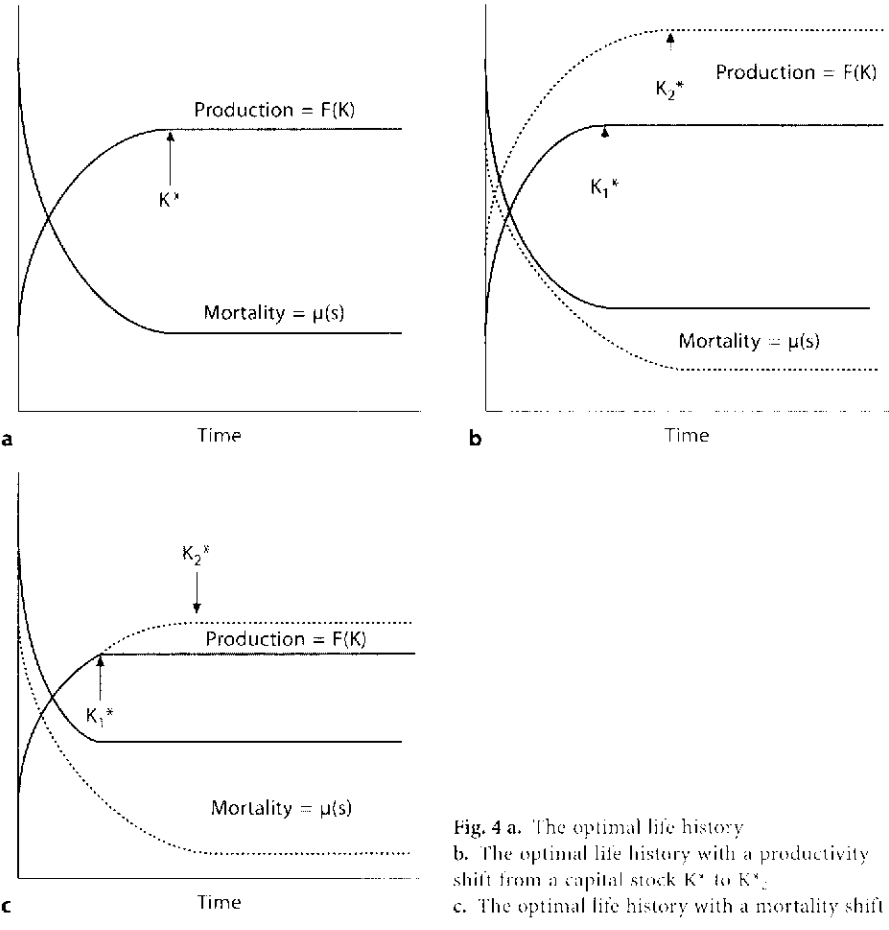

Fig. 4 a. The optimal life history
b. The optimal life history with a productivity shift from a capital stock K^* to K^*_2
c. The optimal life history with a mortality shift

The results of the analysis, which are presented and proven formally in Kaplan and Robson (2001 a), are illustrated in Figures 4 a–c. During the capital investment period, the value of life, which is equal to total expected future net production, is increasing with age, since productivity is growing with increased capital. The optimal value of investment in mortality reduction also increases, since the effect of a decrease in mortality increases as capital increases (illustrated in Fig. 4 a). At some age, a steady state is reached when capital is at its optimum level, and both capital and mortality rates remain constant.

Figures 4 b and 4 c show two important comparative results. In Figure 4 b, the impact of a change in productivity is shown. Some environmental change that increases productivity (holding the marginal value of capital constant) has two reinforcing effects: it increases the optimal level of both capital investment (and hence the length of the investment period) and efforts to reduce mortality. Figure 4 c shows the impact of a reduction in mortality rates, again with two

effects. It both increases the optimal capital stock (because it increases the expected length of life and hence the time over which it will yield returns) and produces a reinforcing increase in effort at reducing mortality, since the impact of a decrease in mortality is greater as mortality rates decrease.

Finally, the model shows that a shift in productivity from younger to older ages (for example, an increased reliance on learning that lowers juvenile energy production but increases adult production) increases the value of living to older ages and therefore the optimal effort at reducing mortality. This has the effect of increasing expected life span. Our theory is that brain size and longevity coevolve for the following reasons. Ecological conditions favoring large brains also select for greater endogenous investments in staying alive. As the stock of knowledge and functional abilities embodied in the brain grow with age, so too does the value of the capital investment. This favors greater investments in health and mortality avoidance. In addition, holding the value of the brain constant, ecological conditions that lower mortality select for increased investment in brain capital for similar reasons; an increased probability of reaching older ages increases the value of investments whose rewards are realized at older ages. The next section applies this logic to the brain and life span evolution in the primate order.

Brain and Life Span Evolution among Primates

The Theoretical and Empirical Model

Relative to other mammalian orders, the primate order can be characterized as slow-growing, slow reproducing, long-lived and large-brained. The radiation of the order over time has involved a series of four directional grade shifts towards slowed life histories and increased encephalization (i.e., brain size relative to body size). Even the more "primitive" prosimian primates are relatively long-lived and delayed in reaching reproductive maturity compared to mammals of similar body size, which suggests the same of early primate ancestors. Austad and Fischer (1991, 1992) relate this evolutionary trend in the primates to the safety provided by the arboreal habitat and compare primates to birds and bats, which are also slow-developing and long-lived for their body sizes. Thus, the first major grade shift that separated the primate order from other mammalian orders was a change to a lowered mortality rate and the subsequent evolution of slower senescence rates, leading to longer life spans and slightly larger brains.

The second major grade shift occurred with the evolution of the anthropoids (the lineage containing monkeys, apes and humans), beginning about 35 mya. Its major defining characteristic is the reorganization of the sensory system, one dominated by binocular, color vision and associated with hand-eye coordination as opposed to olfaction and hearing. These sensory changes co-occurred with an increased emphasis on plant foods (especially hard seeds and fruits), as opposed to insects (Benefit 2000; Fleagle 1999). The grade shift is also

seen in brain size and life history. Regressions of log brain size on log body size (Barton 1999), as well as log maximum life span on body size (Allman et al. 1993), show significant differences in intercept between strepsirhine (including most prosimians) and haplorhine (including all anthropoids and a few prosimians) primates. Relative to prosimians, anthropoids also have lower metabolic rates and longer gestation times (Martin 1996).

The evolution of monkey and ape dietary adaptations in the Miocene and Pliocene appears to be based on an early adaptation for both groups to feed on hard seeds and green fruits (Benefit 2000). In the Late Miocene/Early Pliocene, cercopithecoids, which had been semi-terrestrial, cursorial, hard seed and green fruit eaters much like modern vervet monkeys, evolved new digestive adaptations allowing the colobines to digest mature leaves. Cercopithecoids also began to more directly compete with apes in both terrestrial and arboreal habitats. Miocene apes were highly diverse and found in many habitats but were essentially agile arboreal quadrupeds. By the Late Miocene apes had fully developed their characteristic shoulder girdle morphology, allowing suspension below branches that gave special access to ripe fruits for larger bodied animals. This dietary shift to dependence on ripe fruits, based on the morphological adaptation of arm suspension, moved apes into a new grade with an emphasis on feeding higher in the food pyramid on very nutritious food packets high in energy but spatially and temporally dispersed in an arboreal habitat. This new grade reduced direct competition with monkeys, ceded open terrestrial habitat to them, and greatly reduced the number and diversity of ape species. At the same time it put a premium on acquired knowledge about the location of ripe fruits and for skills for more complex extractive foraging of embedded and protected, high energy and fatty foods such as nuts, insects and hard-shelled fruits.

This third major grade shift marked the evolution of the hominoid lineage (leading to apes and humans). This grade shift entailed further encephalization, as revealed by a yet greater intercept of log brain size regressed on log body size and superior performance on most tasks reflecting higher intelligence (Byrne 1995 b; Byrne 1997 b; Parker and McKinney 1999). The divergence of the hominid line, and particularly the evolution of genus *Homo*, defines the fourth major grade shift. The brain size and life span of modern humans are very extreme values among mammals, and even primates. Although the record is incomplete, it appears that brain enlargement and life history shifts co-occurred. Early *Home ergaster* shows both significant brain expansion and a lengthened developmental period (Smith 1993), but much less to than modern humans. Neanderthals display both brain sizes and dental development that are in the same range as modern humans. Modern humans have a brain size about three times that of female gorillas of similar weight, and about double the maximum life span.

The proposal here is that both shifts in mortality risks and in the benefits of information storage and processing due to changes in feeding niche underlie these directional changes in the primate lineage through time. However, in addition to these large-scale shifts, a great deal of adaptive variation exists

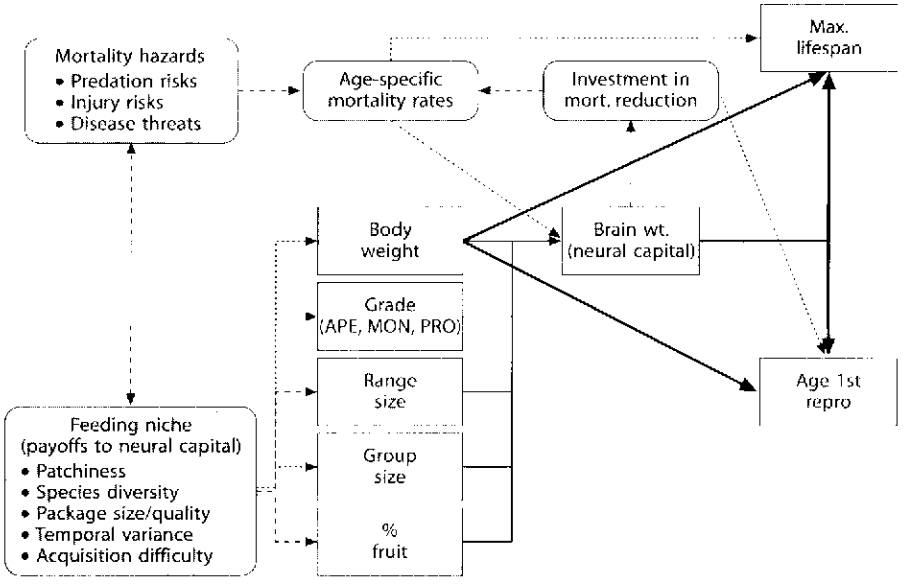

Fig. 5. A theoretical and empirical path model of primate brain evolution. MON, monkey; PRO, prosimian

among primates. Species of all four grades continue to co-exist, often sympatrically (especially monkeys, apes and humans). Moreover, not all evolutionary change has been in the direction of larger brains and longer lives. For example smaller-brained monkeys appear to have replaced apes in some niches at the end of the Miocene (Benefit 2000; Fleagle 1999). If changes in mortality risks and the learning intensiveness of the feeding niche explain the grade shifts, the same factors might also explain variation within grades.

Figure 5 illustrates the theory and the empirical model that it generates, given the available data. On the left, the two rounded boxes represent exogenous ecological variables.[1] Some features of the feeding niche that are likely to affect the payoffs to information acquisition and processing (and hence, brain size) are listed in the lower box. Resource patchiness tends to be associated with larger home ranges and potentially greater demands on spatial memory. The number of different species consumed potentially adds to demands for spatial memory, learned motor patterns, processing of resource characteristics, and temporal associations (Jerison 1973). Large, nutrient-dense, packages (such as big, ripe fruits) tend to be patchily distributed in space and often with very short windows of availability (Clutton-Brock and Harvey 1980; Milton 1981, 1993). Year-to-year abundance and location of high-quality packages also

[1] Although feeding niche is subject to selection, the suites of foods eaten are treated as givens in order to model how selection molds life history traits, brain size and other features of phenotype in response to niche conditions.

appear to vary. Hence, diets with a greater relative importance of large, high-quality packages are probably associated with increased brain size through several routes: by increasing the number of species exploited, by increasing the size of the home range, and by increasing the importance of predicting the timing and location of availability. In addition, some high quality foods, such as hard-shelled fruits, nuts, insects, and honey, must be extracted from protective casings and their exploitation often requires learned strategies and tools.

Features of the environmental/behavioral niche of the organism that are likely to affect mortality rates and the payoffs of investments in mortality reduction are listed in the upper, left box. Life in or near trees probably increases injury risk but decreases predation risk to overall lower mortality risks. Lowered risk of mortality due to predation is expected to increase investment in combating disease and, hence, decrease disease risks as well (though these have received little attention in primate studies to date). Lower mortality rates increase the probability of reaching older ages and therefore affect the payoffs to larger brains, holding feeding niche constant.

The co-evolution of brain size and mortality patterns is shown in the path diagram (dashed arrows depict effects of unmeasured conceptual or latent variables). Both features of the feeding niche and mortality risks affect optimal brain size. Brain size is expected to have both direct and indirect effects on life span and age of first reproduction. Larger brains may confer direct survival advantages through increased physiological efficiency and through learned predator avoidance (Allman et al. 1993; Armstrong 1982; Hakeem et al. 1996; Jerison 1973; Rose and Mueller 1998). In addition, since larger brains are associated with greater relative productivity at older ages, brain size is expect to be associated with investment in mortality reduction. Similarly, the energetic costs of the brain reduce energy available for growth, and learning-based feeding niches may lower productivity during the juvenile period. This would produce slower growth rates and a later age of first reproduction, holding body size constant. The greater allocations to mortality reduction (e.g., increased immune function, reduced foraging time) would also slow the growth rates.

The rectangular boxes depict measured variables for which comparative data are available, and the solid arrows depict associations that can be tested empirically. The thinner lines represent the first stage in the model, predicting brain weight. Measures of feeding niche are captured by grade (ape, monkey vs. prosimian), range size, and percentage of fruit in the diet. We also include body and group size in this first stage. In addition to directly affecting brain size, body size is likely to be associated with dietary niche. For example, larger home ranges probably favor larger bodies, because of their greater locomotor efficiency. Larger home ranges may also be associated with larger groups, because holding resource abundance constant, a patchy environment will tend to produce both larger home ranges and a larger number of individuals feeding at each resource patch (Wrangham 1979). Because the social intelligence hypothesis has figured so prominently in the literature (Barton and Dunbar 1997; Byrne 1995 b; Dunbar 1998), the path between group size and brain size is also

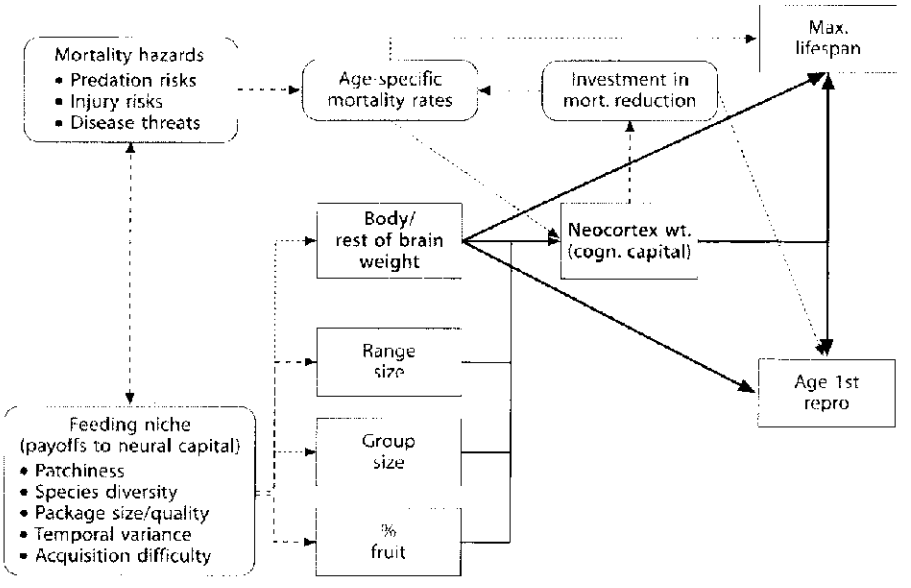

Fig. 6. A theoretical and empirical path model of neocortex evolution

included. In addition, if social intelligence takes time to acquire and its benefits are weighted towards older ages (as may well be the case), embodied capital theory does predict that selection on social intelligence will co-evolve with longevity and mortality rates. For example, social intelligence might allow alpha males to retain their high status to older ages and it might confer greater benefits to females when they have many descendants (in the case of ranked matrilines). Such effects would also be consistent with the model. The second stage, shown with bold arrows, examines the effects of brain size and body size on age of first reproduction and maximum recorded life span, respectively.

A second model will also be tested (see Fig. 6). The logic of the embodied capital model suggests that the brain functions that are most involved in transforming present experience into future performance should have the greatest impact on the payoffs to living longer and allocating effort to mortality reduction. In addition, it has been argued that the association of brain size with life span in primates, after controlling for body size, is spurious and due to greater measurement error in body size than in brain size [Dunbar 1998; Economos 1980, although Allman et al. (1993) have shown that brain size is a better predictor of life span than the size of other organs]. To address these issues, the size of the neocortex will be disaggregated from the rest of the brain. The neocortex should better reflect the learning intensiveness of the feeding niche and social system than the rest of the brain. In the second model, neocortex weight replaces brain weight, and the weight of the rest of the brain replaces body weight, as an instrument (since measurement error for neocortex and rest of brain weight, respectively, should be similar).

The Primate Sample

Data are available on the total adult brain weights (in grams) for 124 species, compiled from secondary sources (Barton 1999; Harvey et al. 1987). From this sample, there are 95 species for which data are available on mean adult body weight (in grams), group size, age at first breeding for females (in months), maximum life span (in years), maximum home range (in hectares), and percent frugivory. Much of the data came from secondary sources (Barton 1996, 1999; Dunbar 1992; Harvey et al. 1987; Ross 1992). These data differ, however, from previous analyses in a heavier reliance on primary field data for female age at first breeding, maximum home range, and percent frugivory (see Kaplan et al. 2001 for details). They may thus more accurately represent the selection pressures faced by wild individuals, which are assumed to be living in conditions much more representative of the context in which these features co-evolved.

Results

A two-stage least squares regression analysis was performed to test the models. For the model in Figure 5, the first stage was conducted hierarchically. First, the natural logarithm of brain weight was regressed on the natural logarithms of body weight, range size, and group size, and on percent age of fruit in the diet. Then to capture other aspects of niche differentiation, grade (ape and monkey, compared to a prosimian baseline) was added as a fixed effect to determine if it significantly improved the model. The results are presented in Table 1. In the simple model without grade, body weight, range size and percent age of fruit in the diet are each positively related to brain weight, accounting for 94% of the variance. Group size was not significant. Grade significantly improved the model fit ($p < .0001$), with the model now accounting for 97% of the variance. In this model, percent age of fruit is no longer significant, but group size is. The predicted values of log brain size from this full model are then used in the second stage of the analysis.[2]

Part B of Table 1 shows the results of the second stage, in which the natural logarithms of female age at first reproduction and maximum reported life span, respectively, are regressed on the logs of predicted brain weight and body weight. In both cases, brain weight explains most of the variance and the effect of body weight is now negative. When brain weight is not in the model, the association between body weight and both life span and age of first reproduction is, of course, strongly positive. It may be that, after controlling for brain weight, larger bodied species eat lower quality diets (Aiello and Wheeler 1995; Milton 1981, 1987, 1988, 1993; Milton and Demment 1988), and this is associated with a relatively shorter life span and earlier age at first reproduction.

[2] Since brain size is endogenous, the problem of simultaneity can be addressed by using predicted brain size in this second regression. Similar results are obtained, however, when measured values are used instead of predicted values.

Table 1. Two-stage model of brain size and life history

A. Stage I, brain weight

	$R^2 = 0.94$, N = 95				$R^2 = 0.97$, N = 97			
Parameter	B	Std. Error	t	Sig.	B	Std. Error	t	Sig.
Intercept	−1.74	0.16	−11.22	0.0000	−1.74	0.16	−11.22	0.0000
Body weight	0.68	0.03	23.01	0.0000	0.59	0.02	24.83	0.0000
Range size	0.05	0.03	1.95	0.0550	0.05	0.02	2.54	0.0130
Group size	0.07	0.04	1.64	0.1040	0.07	0.04	2.02	0.0468
Percentage fruit	0.00	0.00	2.65	0.0100	0.00	0.00	1.46	0.1484
Ape	–	–	–	–	0.87	0.10	8.73	0.0000
Monkey	–	–	–	–	0.45	0.07	6.42	0.0000
Prosimian					0.00	–	–	–

B. Stage II

	Maximum life span, $R^2 = 0.52$, N = 80				Age of first reproduction, $R^2 = 0.74$, N = 79			
Parameter	B	Std. Error	t	Sig.	B	Std. Error	t	Sig.
Intercept	3.14	0.34	9.32	0.0000	2.78	0.37	7.58	0.0000
Body weight	−0.24	0.10	−2.36	0.0208	−0.21	0.11	−1.99	0.0503
Brain weight	0.53	0.13	4.12	0.0001	0.71	0.14	5.19	0.0000

The results of decomposing brain weight into the neocortex and the rest of the brain (Fig. 6) are shown in Tables 2 and 3. Using the same set of regressors as in the full model of brain size, the natural logarithms of the weights of rest of the brain and of the neocortex (shown on the left and right sides of Table 2, respectively) are each treated as dependent variables. Body weight and grade are the only variable that significantly affects the weight of non-neocortical brain tissue, and the effect of grade is rather small. With respect to neocortex

Table 2. Neocortex and rest of brain weight

	Rest of brain weight (Brain weight−Neocortex weight) $R^2 = 0.94$, N = 32				Neocortex weight, $R^2 = 0.98$, N = 32			
Parameter	B	Std. Error	t	Sig.	B	Std. Error	t	Sig.
Intercept	−2.03	0.30	−6.66	0.0000	−2.46	0.25	−9.68	0.0000
Body weight	0.55	0.05	10.42	0.0000	0.57	0.04	13.02	0.0000
Range size	0.08	0.04	1.79	0.0860	0.12	0.04	3.41	0.0020
Group size	−0.05	0.07	−0.68	0.5010	0.02	0.06	0.28	0.7800
Percentage fruit	0.00	0.00	0.42	0.6810	0.00	0.00	0.77	0.4470
Ape	0.51	0.23	2.25	0.0330	0.89	0.19	4.64	0.0000
Monkey	0.27	0.16	1.73	0.0950	0.71	0.13	5.51	0.0000
Prosimian	0.00	–	–	–	0.00	–	–	–

Table 3. Two stage model of neocortex size and life history

Stage I				
	Neocortex size, $R^2 = 0.996$, N = 32			
Parameter	B	Std. Error	t	Sig.
Intercept	−0.26	0.05	−5.03	0.0000
Rest of brain weight	1.10	0.03	31.83	0.0000
Range size	0.01	0.02	0.76	0.4556
Group size	0.06	0.03	2.35	0.0270
Percentage fruit	0.00	0.00	0.88	0.3871
Ape	0.52	0.09	5.99	0.0000
Monkey	0.47	0.06	8.29	0.0000
Prosimian	0.00	–	–	–
Stage II				
	Maximum life span, $R^2 = 0.70$, N = 32			
Parameter	B	Std. Error	t	Sig.
Intercept	2.66	0.11	25.21	0.0000
Rest of brain weight	−0.20	0.24	−0.83	0.4122
Neocortex weight	0.38	0.19	2.05	0.0506
	Age of first reproduction, $R^2 = 0.79$, N = 32			
Intercept	2.49	0.14	17.42	0.0000
Rest of brain weight	−0.30	0.31	−0.99	0.3304
Neocortex weight	0.64	0.24	2.69	0.0119

weight, however, both range size and grade have large effects. Thus, consistent with the above logic, feeding niche has a larger effect on neocortex weight than on the rest of the brain, which appears to be more a function of body weight.

In Table 3, the weight of the rest of the brain is used as an instrument for body weight in stage 1 of the model. This model shows that neocortical weight increases more than proportionally with the rest of the brain (b = 1.1), and that both grade and group size have large significant effects. In the second stage, predicted neocortical weight is positively associated with both age at first reproduction and maximum life span, whereas the rest of the brain is not significantly associated with the life history variables. This finding is also consistent with the model. These results should be treated with some caution, however, because the two measures of brain weight are highly collinear.

The Evolution of *Homo*: Chimpanzees and Modern Humans Compared

The same principles may explain the very long lives and the very large brains characteristic of the genus *Homo* and particularly of modern *Homo sapiens*. *Homo* has existed for about 2 million years. Figure 7 shows that human ances-

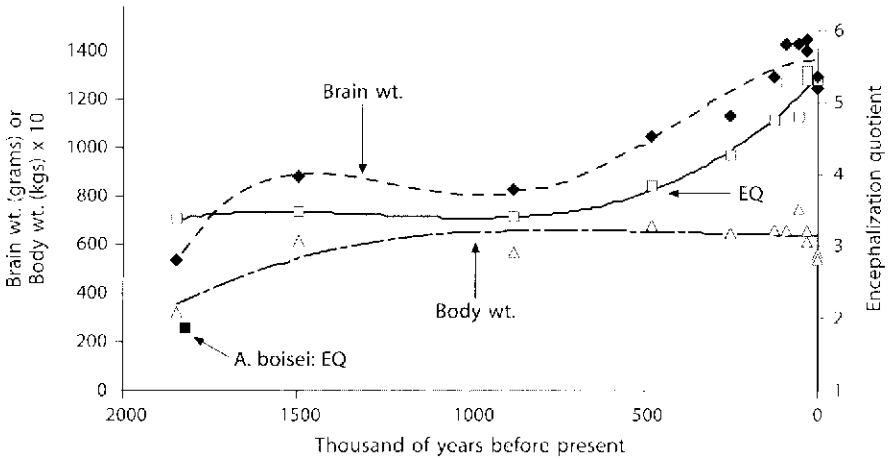

Fig. 7. Hominid brain size and body weight. EQ, encephalization quotient

tors experienced a dramatic increase in brain size but a much less marked increase in body size, especially during the second half of this period. Using Martin's (Martin 1981) measure of "Encephalization Quotient *(EQ)*" (i.e., brain weight corrected allometrically for body weight, with $EQ = \frac{(brain\ wt)}{11.22^{*}\ (body\ wt.)^{.76}}$; one is the average value for a mammal), the large increases in brain size relative to body size are shown with the bold line. Australopithecus, the presumed evolutionary ancestor of *Homo*, coexisted with early *Homo*. *A. boisei*, in particular, had an *EQ* of just over two, which compares to about 3.5 for early *Homo*. Life spans of extinct species are not directly observable, of course, but indirect evidence suggests the life span of australopithecines was much less than that of modern humans and comparable to that of chimpanzees (Smith 1991), with early species in the genus *Homo* having life spans (Smith 1993) that are intermediate between chimpanzees and modern humans.

Hominids have subsisted on hunting and gathering, perhaps supplemented by scavenging, for all but the last 10,000 years of our evolutionary history. Our proposal (see Kaplan + Robson 2002; Kaplan 1997; Kaplan et al. 2000) is the hunting and gathering lifestyle is responsible for the evolution of these extreme values with respect to brain size and longevity. Our proposal is that large brains and long lives are co-evolved responses to an equally extreme commitment to learning-intensive foraging strategies and a dietary shift towards high-quality, nutrient-dense, and difficult-to-acquire food resources. The following logic underlies our proposal. First, high levels of knowledge, skill, coordination and strength are required to exploit the suite of high-quality, difficult-to-acquire resources humans consume. The attainment of those abilities requires time and a significant commitment to development. This extended learning phase during which productivity is low is compensated for by higher productivity during the

adult period, with an intergenerational flow of food from old to young. Since productivity increases with age, the time investment in skill acquisition and knowledge leads to selection for lowered mortality rates and greater longevity, because the returns on the investments in development occur at older ages.

Second, we believe that the feeding niche specializing on large, valuable food packages, and particularly hunting, promotes cooperation between men and women and high levels of male parental investment, because it favors sex-specific specialization in embodied capital investments and generates a complementarity between male and female inputs. The economic and reproductive cooperation between men and women facilitates provisioning of juveniles, which both bankrolls their embodied capital investments and acts to lower mortality during the juvenile and early adult periods. Cooperation between males and females also allows women to allocate more time to childcare and increases nutritional status, increasing both survival and reproductive rates. Finally, large packages also appear to promote inter-familial food sharing. Food sharing assists recovery in times of illness and reduces risk of food shortfalls due to both the vagaries of foraging luck and to variance in family size due to stochastic mortality and fertility. These buffers against mortality also favor a longer juvenile period and higher investment in other mechanisms to increase life span.

Thus, the proposal is that the long human life span co-evolved with the lengthening of the juvenile period, increased brain capacities for information processing and storage, and intergenerational resource flows – all as a result of an important dietary shift. Humans are specialists in that they only consume the highest quality plant and animal resources in their local ecology and rely on creative, skill-intensive techniques to exploit them. Yet, the capacity to develop new techniques for extractive foraging and hunting allows them to exploit a wide variety of different foods and to colonize all of the Earth's terrestrial and coastal ecosystems.

The best available evidence for evaluating this theory is to compare wild living chimpanzees, human's closest living relatives, with contemporary hunter-gatherers who still subsist on foraging for subsistence and who have little or no access to Western medicine. Both chimpanzees and contemporary foragers have been affected by current global trends, such as deforestation, population movements, and other effects of modern economies. They cannot be treated as replicas of the evolutionary past. Nevertheless, the differences in the diets, survival rates, and age profiles of productivity between chimpanzees and contemporary hunter-gatherers are striking and consistent with the theory.

Diet, Survival and Age Profiles of Productivity among chimpanzees and contemporary hunter-gatherers

Diet

There are ten foraging societies[3] and five chimpanzee communities for which caloric production or time spent feeding were monitored systematically (Kaplan et al. 2000). Modern foragers all differ considerably in diet from chimpanzees. Measured in calories, the major component of forager diets is vertebrate meat, which ranges from about 30 % to around 80 % of the diet in the sampled societies, with most diets consisting of more than 50 % vertebrate meat (equally weighted mean = 60 %), whereas chimpanzees obtain about 2 % of their food energy from hunted foods.

The next most important food category in the forager sample is extracted resources, such as roots, nuts, seeds, most invertebrate products, and difficult-to-extract plant parts, such as palm fiber or growing shoots. They may be defined as non-mobile resources that are embedded in a protective context such as underground, in hard shells or bearing toxins that must be removed before they can be consumed. In the ten-forager sample, extracted foods accounted for about 32 % of the diet, as opposed to 3 % among chimpanzees.

In contrast to hunted and extracted resources, which are difficult to acquire, collected resources form the bulk of the chimpanzee diet. Collected resources, such as fruits, leaves, flowers, and other easily accessible plant parts, are simply gathered and consumed. They account for 95 % of the chimpanzee diet, on average, and only 8 % of the forager diet.

The data suggest that humans specialize in rare but nutrient-dense resource packages or patches (meat, roots, nuts) whereas chimpanzees specialize in ripe fruit and low-nutrient-density plant parts. These differences in nutrient density of foods ingested are also reflected in human and chimpanzee gut morphology and food passage time, with chimpanzees specialized for rapid processing of large quantities and low nutrient, bulky, fibrous meals (Milton 1999).

The Age Profile of Acquisition for Collected, Extracted, and Hunted Resources

In most environments, fruits are the easiest resources that people acquire. Daily production data among Ache foragers show that both males and females reach their peak daily fruit production by their mid to late teens. Some fruits that are simply picked from the ground are collected by two- to three-year-olds at 30 % of the adult maximum rate. Ache children acquire five times as many calories

[3] The hunter-gatherer data come from studies on populations during periods when they were almost completely dependent on wild foods, with little modern technology (and no firearms), no significant outside interference in interpersonal violence or fertility rates, and no significant access to modern medicine (see Kaplan et al. 2000 for details).

per day during the fruit season as during other seasons of the year (Kaplan 1997). Similarly, among the Hadza, teen girls acquired 1650 calories per day during the wet season, when fruits were available, and only 610 calories per day during the dry season, when fruits were not. If we weight the wet and dry season data equally, Hadza teen girls acquire 53% of their calories from fruits, compared to 37% and 19% for reproductive-aged and post-reproductive women, respectively (Hawkes et al. 1989).

In contrast to fruits, the acquisition rate of extracted resources often increases through early adulthood as foragers acquire necessary skills. Data on Hiwi women show that root acquisition rates do not asymptote until about age 35–45 (Kaplan et al. 2000) and the rate of 10-year-old girls is only 15% of the adult maximum. Hadza women appear to obtain maximum root digging rates by early adulthood (Hawkes et al. 1989). Hiwi honey extraction rates by males peak at about age 25. Again the extraction rate of 10-year-olds is less than 10% of the adult maximum. Experiments done with Ache women and girls clearly show that young adult girls are not capable of extracting palm products at the rate obtained by older Ache women (Kaplan et al. 2000). Ache women do not reach peak return rates until their early 20s.! Kung (Ju/'hoansi) children crack mongongo nuts at a much slower rate than adults (Blurton Jones et al. 1994), and Bock (1995) has shown that nut-cracking rates among the neighboring Hambukushu do not peak until about age 35. Finally, chimpanzee juveniles also focus on more easily acquired resources than adult chimpanzees. Difficult-to-extract activities such as termite and ant fishing or nut cracking are practiced less by chimpanzee juveniles than by adults (Boesch and Boesch 1999; Hiraiwa-Hasegawa 1990; Silk 1978).

Human hunting differs qualitatively from hunting by other animals and is the most skill-intensive foraging activity. Unlike most animals that either sit and wait to ambush prey or use stealth and pursuit techniques, human hunters use a wealth of information to make context-specific decisions, both during the search phase of hunting and then after prey are encountered. Specifically, information on ecology, seasonality, current weather, expected animal behavior and fresh animal signs is all integrated to form multivariate mental models of encounter probabilities that guide the search and are continually updated as conditions change (Leibenberg 1990). Various alternative courses of action are constantly compared and referenced to spatial and temporal mental maps of resource availability (ibid). This information is collected, memorized and processed over much larger spatial areas than chimpanzees ever cover. For example, interviews with Ache men show that fully adult men (aged 35+) had hunted in an area of nearly 12,000 km^2 of tropical forest in their lifetimes. Almost all foragers surveyed use more than 200 km^2 in a single year, and many cover more than 1000 km^2 in a year (Kelly 1995; Table 4.1). Male chimpanzees, on the other hand, cover only about 10 km^2 in a lifetime (Wrangham and Smuts 1980; Wrangham 1975).

In addition, humans employ a wide variety of techniques to capture and kill prey, using astounding creativity (Kaplan et al. 2000). Those kill techniques are

tailored to many different prey under a wide variety of conditions. For example, from 1980 to 1996 our sample of weighed prey among the Ache includes a minimum of 78 different mammal species, at least 21 species of reptiles and amphibians, probably over 150 species of birds (more than we have been able to identify) and over 14 species of fish. Finally, human hunters tend to select prey that is in prime condition from the perspective of human nutritional needs rather than prey made vulnerable by youth, old age or disease as do so many carnivorous animals (Alvard 1995; Stiner 1991).

The skill-intensive nature of human hunting and the long learning process involved are demonstrated dramatically by data on hunting return rates by age. Hunting return rates among the Hiwi do not peak until age 30–35, with the acquisition rates of 10-year-old and 20-year-old boys reaching only 16 % and 50 % of the adult maximum, respectively. The hourly return rate for Ache men peaks in the mid 30s. The return rate of 10-year-old boys is about 1 % of the adult maximum, and the return rate of 20-year-old juvenile males is still only 25 % of the adult maximum. Marlowe (unpublished data) obtained similar results for the Hadza. Also, boys switch from easier tasks, such as fruit collection, shallow tuber extraction and baobab processing, to honey extraction and hunting in their mid to late teens among the Hadza, Ache and Hiwi (Blurton Jones et al. 1989, 1997; Kaplan et al. 2000). Even among chimpanzees, hunting is strictly an adult or subadult activity (Boesch and Boesch 1999; Stanford 1998; Teleki 1973).

Survival and Net Food Production

Figure 8 (Kaplan et al. 2000) shows the probabilities of survival and net production (i.e., food acquired minus food consumed) by age. The chimpanzee net production curve shows three distinct phases. The first phase, to about age five, is the period of complete to partial dependence upon mother's milk and of negative net production. The second phase is independent juvenile growth, lasting until adulthood, during which net production is zero. The third phase is reproductive, during which females, but not males, produce a surplus of calories that they allocate to nursing.

Humans, in contrast, produce less than they consume for about 20 years. Net production becomes increasing by negative until about age 14 and then begins to climb. Net production in adult humans is much higher than in chimpanzees and peaks at a much older age, reflecting the payoff of long dependency. More precisely, human peak net production is about 1750 calories per day, reached at about age 45. Among chimpanzee females, peak net production is only about 250 calories per day and, since fertility decreases with age, net productivity probably decreases throughout adulthood.

The survival curves, using the scale on the right-hand y-axis, reveal why the human age profile of productivity requires a long adult life span. Only about 30 % of chimpanzees ever born reach 20, the age when humans produce as

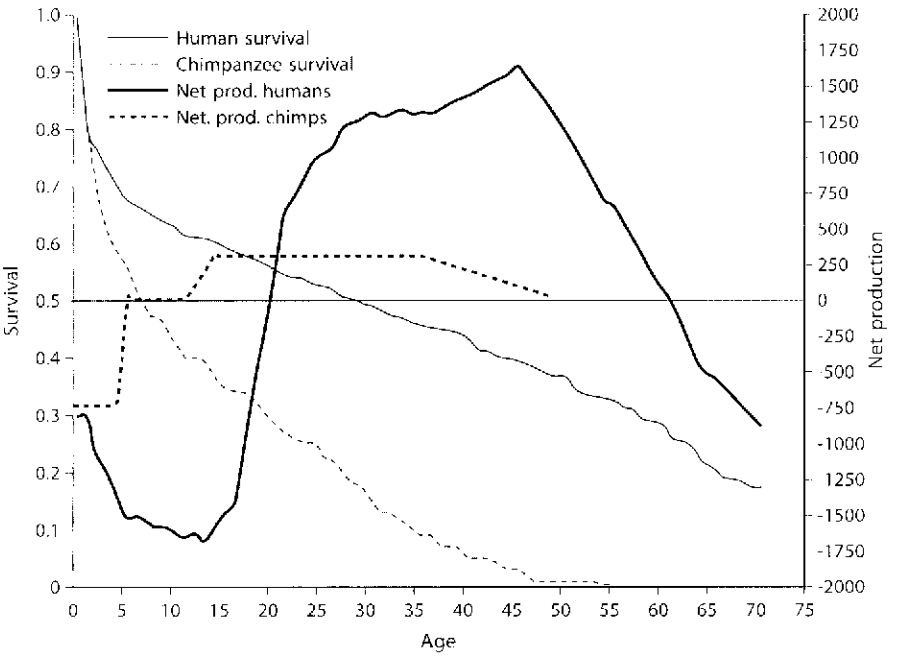

Fig. 8. Survival and net food production: human foragers and chimpanzees. On the left vertical axis is the probability of survival and on the right is net production in calories per day. The data on chimpanzee survival are derived from averaging age-specific mortality rates from all five study sites where systematic data on births and deaths are recorded (Hill et al. 2001); data on chimpanzee food consumption and production are from Gombe (Goodall 1986). Human survival rates are averaged from Ache (Hill and Hurtado 1996), Hiwi (Kaplan et al. 2000), and Hadza (Blurton Jones et al. 2002). Net production data are from the same groups (details on all sources and estimation procedures for both human and chimpanzee production and consumption data are in Kaplan et al. 2000

much as they consume. Less than 5 % of chimpanzees reach 45, when human net production peaks, but more than 15 % of hunter-gatherers survive to age 70. By age 15, chimpanzees have consumed 43 % and produced 40 % of their expected lifetime calories, respectively; in contrast, humans have consumed 22 % and produced only 4 % of their expected lifetime calories!

The relationship between survival rates and age profiles of production is made even clearer in Figure 9. The thin solid line plots net production by age for foragers (as in Fig. 8). The bold line shows expected net production for foragers, which is net production at each age multiplied by the probability of being alive at each age. The area of the "deficit" period, prior to age 20, is about the same size as the surplus after age 20. The dashed line shows the hypothetical "contrary to fact" expected net production profile of a human forager with a chimpanzee survival function. The area of the deficit is now much larger than the area of the surplus, since very few individuals survive to the highly productive ages. This shows that the human production profile would not be viable with chimpanzee survival rates, because expected lifetime net production would be negative.

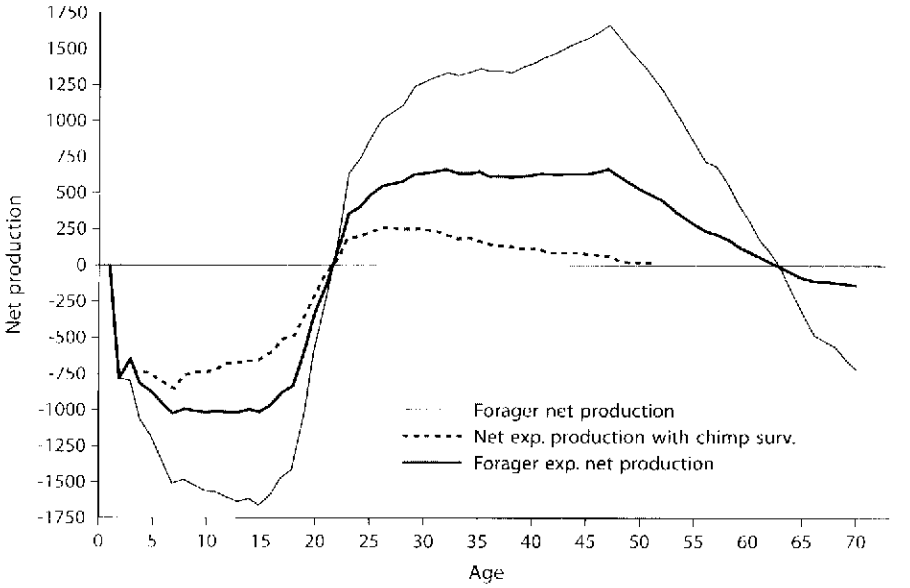

Fig. 9. Expected net production

Development and Cognitive Function Among Monkeys, Apes and Humans

Although human intelligence has long been recognized as our most distinctive specialization as a species, it is now becoming increasing clear that our larger brains and greater intellectual capacities depend upon the elongation or stretching out of development at every stage. The production of cortical neurons in mammals is limited to early fetal development and, compared to monkeys and apes, human embryos spend an additional 25 days in this phase (Deacon 1997; Parker and McKinney 1999). The greater original proliferation of neurons in early fetal development has cascading effects in greatly extending other phases of brain development, ultimately resulting in a larger, more complex and effective brain. For example, in monkeys, such as macaques, myelination of the brain begins prenatally and is largely complete in three month, but in humans continues to at least 12 years of age (Gibson 1986). Dendritic development is similarly extended to age 20 or greater in humans.

The timing of cognitive development is extended in chimpanzees relative to monkeys and in humans relative to apes (Parker and McKinney 1999). In terms of Piagetian stages, frequently used by comparative cognitive psychologists, macaque monkeys traverse only two sub-periods of cognitive development regarding physical phenomena by about six months of age and peak in their logical abilities by about three years of age; however, they fail to be able to represent objects symbolically, to classify objects hierarchically or to recognize themselves in a mirror. Chimpanzees traverse three to four subperiods of cogni-

tive development by about eight years of age.[4] They can recognize themselves in a mirror and are much better at classification than macaques but are not capable of constructing reversible hierarchical classes and abstract, logical reasoning. Human children traverse eight subperiods of cognitive development over 18–20 years.

It is interesting to note that even though humans take about 2.5 times as long to complete cognitive development as do chimpanzees, humans actually learn faster than chimpanzees. In most cognitive spheres, especially language, a two-year-old child has the abilities of a four-year-old chimpanzee, even with intensive training. Humans appear to have much more to learn and their brains require more environmental input to complete development. Formal abstract logical reasoning does not emerge until age 16 to 18. This is the age when productivity begins to increase dramatically among modern hunter-gatherers (see below). The ability to construct abstract scenarios and deduce logical relationships appears to allow for the growth in knowledge that results in peak productivity in the mid thirties.

Elongated development in humans is also associated with slowed aging of the brain. Macaques exhibit physiological signs of cognitive impairment, as evidenced by the appearance of Alzheimer-like neuropathology (senile plaques, neurocytoskeletal abnormalities) and cerebral atrophy by age 22–25, and chimpanzees by age 30, in contrast to humans for whom such changes are rare until age 60 ($< 1\,\%$) and only common ($> 30\,\%$) by age 80s (Finch 2002).

Mechanisms Underlying Brain-Longevity Co-Evolution

There are several possible pathways by which, at the species level, brain size and function might co-evolve with life span and the process of aging. One possibility is simply through the additive phenotypic effects of genes contributing to the selective environment of other genes. For example, the phenotypic effects of genes affecting brain development and function increase foraging returns for high-quality, nutrient-dense foods during adulthood. The ensuing diet and age profile of production then constitute the selective environment for genes affecting dietary physiology (e.g., the size of the large intestine) and rates of aging (e.g., accumulation of plaque and free radicals). At the same time, those phenotypic effects of those latter genes affect the selective environment for genes affecting brain tissue and brain development. This could result in a "ratcheting" process, in which both sets of genes change over time, resulting in nonrandom associations of brains and longevity at the species level.

Another possibility is that such associations could be due to pleitropy (i.e., single genes influencing more than one trait) and/or linkage disequilibrium (sets of genes jointly assorting during meiosis). Research into brain aging and

[4] The fourth subperiod, such as conservation of quantities of liquids under container transformations, seem to require tutelage and symbolic training.

longevity suggests that some genes may have such pleiotropic effects. The apolipoprotein (apoE) allele system is a good example since this seems to affect neurite growth and the aging of both the brain and the cardiovascular system. (The discussion here is based on Finch and Sapolsky 1999, which gives the original sources.) Brain aging, as in the symptoms of Alzheimer's disease, is common in long-lived mammals. These signs of brain aging are delayed in humans relative to apes and in apes relative to monkeys. In humans, apoE has at least three variants (apoE ε2, ε3 and ε4) whereas all nonhuman primates that have been studied have the same variant, most similar to human apoE ε4. Interestingly, this variant is a risk factor for both Alzheimer's disease and coronary artery disease, suggesting that the apoE ε2 and ε3 variants may have evolved to slow down both brain and cardiovascular aging. These other variants also promote neurite growth in cultured neurons, suggesting they also stimulate greater brain development and complexity.

Pleitropic effects of this nature could evolve by a similar ratcheting process. The sensitivity of one tissue type (e.g., neurons) to a gene product could affect selection on sensitivity of other tissue types (e.g., vascular tissue) to that same gene product, and vice versa. To the extent that associations between brains and longevity are due to pleitropic effects, this would generate correlations at the individual level as well as at the species level. Given the growing body of data suggesting that such individual-level associations exist among humans, pleitropy deserves careful consideration.

Another possible pathway underlying associations at the individual level, especially among humans, is through adaptive behavioral flexibility. Human life histories, including the life span, show evidence of systematic variation in response to environmental variation. Those effects appear to be the result of the interaction between changes in environmental conditions and human physiology and behavior. Perhaps the most dramatic example of that interaction is the pattern of changes accompanying modernization, the secular trend. Increased nutrition and decreased disease loads have systematic effects on human developmental physiology. Physical growth rates increase and maturation begins earlier, resulting in greater stature, higher body weight and earlier age of menarche in girls (Eveleth 1986; Lancaster 1986; Worthman 1999). This response is very likely the result of selection on adaptive flexibility in growth and maturation due to environmental variation in food supply and disease assault rates experienced during human evolutionary history.

In contrast to this increase in physical developmental rate, aging may be slowed in response to better nutrition and decreased disease loads. Although it is possible that humans would also show slowed aging in response to radical reductions in caloric intake (Shanley and Kirkwood 2000), it is also possible that under the more common range of variation, rates of aging are slowed and life spans are longer when nutrition is better and disease loads are lower (Fogel and Costa 1997), and this too is adaptive. The changing mortality rates among older people accompanying modernization and the fact that many chronic diseases occurred at earlier ages in the 19th century U.S. than today (Costa 2000) are consistent with such a possibility.

One countervailing force may also be the product of adaptive flexibility to past environmental variation. Increased risk of heart disease, diabetes and cancer due to overweight and lack of exercise may also be the result of evolved responses. Given the common activity regimes in our past and the variability in food supply, human appetites and nutritional biochemistry may be designed to store fat and increase blood lipid levels when food is abundant. Those adaptations may be acting to shorten the life span in the context of modern activity regimes and food access.

In addition to these physiological adaptations, there are also behavioral responses to modernization. The models outlined in Figures 2–4 are equally applicable to short-term behavioral variation as they are to long-term life history adaptations. Two dramatic effects of modernization are increased economic payoffs to educational capital and decreased mortality due to improvements in public health. Those models predict reinforcing endogenous behavioral responses to such changes. Increased payoffs to education should promote both increased investment in educational capital and in staying alive. Improvements in public health should also promote reinforcing increases in capital investment and staying alive. The fact that income grows more with age with increasing education suggests that there will be similar differentiation across educational groups. Thus, individuals with high levels of education and intelligence may expect their economic status to grow as they age, and therefore engage in behavior that increases their likelihood of reaching older ages; conversely, those whose future prospects seem poor or not likely to improve with age might be less inclined to invest in health. Similarly, variation in exposure to risks of mortality (associate with violence, AIDS, or other hazards) should affect willingness to invest in education and future earnings.

Discussions and Conclusions

The analyses in this paper have applied embodied capital theory to understanding primate radiations in brain size and longevity and the evolution of the human life course. Embodied capital theory organizes the relationships of ecology, brain size and longevity among primates, which existing debates about primate brain size evolution have failed to do. Most studies of brain evolution have ignored longevity and focused either on the benefits or on the costs of brains, but not both. The liveliest current debate concerns whether the benefit of a large brain is to solve ecological or social problems (Allman et al. 1993; Barton and Dunbar 1997; Byrne and Whiten 1988; Clutton-Brock and Harvey 1980; Dunbar 1998; Milton 1993). On the cost side, another debate concerns, for example, whether larger brains require smaller guts or lower metabolic rates (Aiello and Wheeler 1995; Barton 1999; Foley and Lee 1991; Martin 1996).

Studies examining the relationship between the brain and longevity fail to model simultaneous selection. One focus has been on whether the relationship between brain size and longevity is real or a statistical artifact (Allman et al.

1993; Barton 1999; Economos 1980; Foley and Lee 1991; Martin 1996). Another has been on the metabolic costs of growing large brains (Foley and Lee 1991; Martin 1996) and its indirect relationship to life span through body size. Others have focused either on the direct impacts of the brain on life span or on the benefits of a longer life span. For example, Sacher (1975) offers two proposals: 1) brains directly increase life span by ensuring more precise homeostasis of bodily functions; and 2) brains delay maturation and lower the reproductive rate, therefore requiring an extension of the life span. Other hypotheses are: 1) larger brains are beneficial to longer-lived animals because they are likelier to experience food shortages when knowledge of the habitat would facilitate survival (Allman et al. 1993); 2) larger brains decrease ecological vulnerability to environmental risks and select for increased longevity (Rose and Mueller 1998); and 3) larger brains help maintain tissue differentiation and slow the process of entropy leading to senescence (Hofman 1983).

The embodied capital theory shows how features of ecology, including both mortality risks and information processing demands, interact in determining optimal allocations to the brain and survival. It also suggests an alternative interpretation of primate social intelligence. Co-evolutionary selection on brains and longevity due to the complexity and the navigational demands of the primate diet may have produced pre-adaptations for the evolution of social intelligence. Given that primates live long lives with enduring social relationship and given that many species of primates eat foods whose distribution generates within-group competition, there would be selection for the application of existing enhancements in memory and information processing abilities to the management of social interaction. Many animals live in social groups, but primates are notable in terms of the complexity of their social arrangements. Perhaps social pressures alone are not sufficient to select for markedly increased brain size, but they might select for the extension of existing abilities to social problems. This may be why apes display remarkable social intelligence, even though group size is not particularly large (Byrne 1995 a, 1997 a). Orangutans, for example, are mostly solitary, but it takes about seven years for a young orangutan to become independent of its mother (presumably because of the learning-intensive nature of the diet). If this view is correct, it also suggests that the assumption of extreme domain specificity in intelligence may be unwarranted.

There is growing interest in the evolution of human life histories, especially longevity. One model, recently proposed by Hawkes and colleagues (Hawkes et al. 1998), often referred to as the "Grandmother Hypothesis", proposes that humans have a long life span because of the assistance that older, post-reproductive women contribute to descendant kin through the provisioning of difficult-to-acquire plant foods. Women, therefore, are selected to invest in maintaining their bodies longer than chimpanzee females. This model offers no explanation why men live so long. In contrast to this female-centered view, Marlowe (2000) proposes that reproduction by males late in life selects for the lengthening of the human life course, with effects on females being incidental.

The embodied capital theory explains why both men and women live long lives. Both men and women exploit high quality, difficult-to-acquire foods (females extracting plant foods and males hunting animal foods), sacrificing early productivity for later productivity, with a life history characterized by an extended juvenile period where growth is slow and much is learned, and a high investment in mortality reduction to reap the rewards of those investments.

The human adaptation is broad and flexible, in one sense, and very narrow and specialized, in another sense. It is broad in the sense that, as hunter-gatherers, humans have existed successfully in virtually all of the world's major habitats. This has entailed eating a very wide variety of foods, both plant and animal, and a great deal of flexibility in the contributions of different age- and sex-classes of individuals. The human adaptation is narrow and specialized in that it is based on extremely high investments in brain tissue and learning. In every environment, human foragers consume the largest, highest quality, and most difficult-to-acquire foods, using techniques that often take years to learn. It is this legacy that modern humans bring to the complex economies existing today, where education-based embodied capital determines income and the economy is a complex web of specialization and cooperation between spouses, families and larger social units. We are only beginning to explore the implications of this legacy for understanding modern behavior.

References

Aiello L, Wheeler P (1995) The expensive-tissue hypothesis: The brain and the digestive system in human and primate evolution. Curr Anthropol 36: 199–221

Allman J, McLaughlin T, Hakeem A (1993) Brain weight and life-span in primate species. Proc Nat Acad Sci pp. 118–122

Alvard M (1993) Intraspecific prey choice by Amazonian hunters. Curr Anthropol 36: 789–818

Armstrong E (1982) A look at relative brain size in mammals. Neurosci Lett 34: 101–104

Armstrong E, Falk D (eds) (1982) Primate brain evolution. New York: Plenum Press

Austad SN, Fischer KE (1991) Mammalian aging, metabolism, and ecology: evidence from the bats and marsupials. J Gerontol 46: 47–51

Austad SN, Fischer KE (1992) Primate longevity: Its place in the mammalian scheme. Am J Primatol 28: 251–261

Barton RA (1996) Neocortex size and behavioral ecology in primates. Proc Roy Acad (Biol Sci) 263: 173–177

Barton RA (1999) The evolutionary ecology of the primate brain. In: Lee PC (ed) Comparative primate socioecology. Cambridge: Cambridge University Press, pp. 167–203

Barton RA, Dunbar RIM (1997) Evolution of the social brain. In: Whiten A, Byrne RW (eds) Machiavellian Intelligence II. Cambridge: Cambridge University Press, pp. 240–263

Benefit BR (2000) Old World monkey origins and diversification: An evolutionary study of diet and dentition. In: Whitehead P, Jolly CJ (eds) Old World monkeys. Cambridge: Cambridge University Press, pp. 133–179

Blurton Jones N, Hawkes K, O'Connell J (1989) Modeling and measuring the costs of children in two foraging societies. In: Standen V, Foley RA (eds) Comparative socioecology of humans and other mammals. London: Basil Blackwell, pp. 367–390

Blurton Jones NG, Hawkes K, Draper P (1994) Foraging returns of !Kung adults and children: Why didn't !Kung children forage? J Anthropol Res 50: 217–248

Blurton Jones NG, Hawkes K, O'Connell J (1997) Why do Hadza children forage? In: Segal NL, Weisfeld GE, Weisfield CC (eds) Uniting psychology and biology: integrative perspectives on human development. New York: Am Psychol Assoc pp. 297–331

Blurton Jones NB, Hawkes K, O'Connell J (2002) The antiquity of post-reproductive life: Are there modern impacts on hunter-gatherer post-reproductive lifespans? Human Biol 14: 184–205

Bock JA (1995) The determinants of variation in children's activities in a Southern African Community. Ph. D. Dissertation, University of New Mexico, Albuquerque

Boesch C, Boesch H (1999) The chimpanzees of the Tai forest: behavioral ecology and evolution. Oxford: Oxford University Press

Byrne RW (1995 a) The smart gorilla's recipe book. Nat Hist Oct: 12–15

Byrne RW (1995 b) The thinking ape: the evolutionary origins of intelligence. Oxford: Oxford University Press

Byrne RW (1997 a) Machiavellian intelligence. In: Whiten A, Byrne RW (eds) Machiavellian intelligence II: extensions and evaluations. Cambridge: Cambridge University Press, pp. 1–23

Byrne RW (1997 b) The technical intelligence hypothesis: an additional evolutionary stimulus to intelligence? In: Whiten A, Byrne RW (eds): Machiavellian intelligence II: extensions and evaluations. Cambridge: Cambridge University Press, pp. 289–311

Byrne RW, Whiten A (eds) (1988) Machiavellian intelligence. Oxford: Clarendon Press

Charnov EL (1993) Life history invariants: some explanations of symmetry in evolutionary ecology. Oxford: Oxford University Press

Clutton-Brock TH, Harvey PH (1980) Primates, brains and ecology. J Zool London 109: 309–323

Cole LC (1954) The population consequences of life history phenomena. Quart Rev Biol 29: 103–137

Costa DL (2000) Understanding the twentieth century decline in chronic conditions among older men. Demography 37: 53–72

Deacon TW (1997) The symbolic species. New York: W. W. Norton & Co

Dunbar RIM (1992) Neocortex size as a constraint on group size in primates. J Human Evol 20: 469–493

Dunbar RIM (1998) The social brain hypothesis. Evol Anthropol 6: 178–190

Economos AC (1980) Brain-life span conjecture: A re-evaluation of the evidence. Gerontology 26: 82–89

Eveleth PB (1986) Timing of menarche: Secular trend and population differences. In: Lancaster JB, Hamburg BA (eds) School age pregnancy and parenthood. Hawthorne, NT: Aldine de Gruyter, pp. 39–53

Finch CE (2002) Evolution and the plasticity of aging in the reproductive schedules in long lived animals: The importance of genetic variation in neuroendocrine mechanisms. In: Pfaff D, Arnold A, Etgen A, Fahrback S, Rubin R (eds) Hormones, brain and behavior. San Diego: Academic Press

Finch CE, Sapolsky RM (1999) The evolution of Alzheimer disease, the reproductive schedule and the apoE isoforms. Neurobiol Aging 20: 407–428

Fleagle JG (1999) Primate adaptation and evolution. New York: Academic Press

Fogel RW, Costa DL (1997) A theory of techniophysio evolution, with some implications for forecasting population, health care costs and pension costs. Demography 34: 49–66

Foley RA, Lee PC (1991) Ecology and energetics of encephalization in human evolution. Phil Trans Roy Soc London B 334: 63–72

Gadgil M, Bossert WH (1970) Life historical consequences of natural selection. Am Naturalist 104: 1–24

Gibson KR (1986) Cognition, brain size and the extraction of embedded food resources. In: Else JG, Lee PC (eds) Primate ontogeny, cognition, and social behavior. Cambridge: Cambridge University Press, pp. 93–105

Goodall J (1986) The chimpanzees of the Gombe: patterns of behavior. Cambridge: Cambridge University Press

Hakeem A, Sandoval GR, Jones M, Allman J (1996) Brain and life span in primates. In: Abeles RP, Catz M, Salthouse TT (eds) Handbook of the psychology of aging. San Diego: Academic Press, pp. 78–104

Harvey PH, Martin RD, Clutton-Brock TH (1987) Life histories in comparative perspective. In: Smuts BB, Cheney DL, Seyfarth RM, Wrangham RW, Struthsaker TT (eds) Primate societies. Chicago: University of Chicago

Hawkes K, O'Connell JF, Blurton Jones N (1989) Hardworking Hadza grandmothers. In: Standen V, Foley RA (eds) Comparative socioecology of humans and other mammals. London: Basil Blackwell, pp. 341–366

Hawkes K, O'Connell JF, Blurton Jones NG, Alvarez H, Charnov EL (1998) Grandmothering, menopause, and the evolution of human life histories. Proc Nat Acad Sci USA 95: 1336–1339

Hill K, Hurtado AM (1996) Ache life history: the ecology and demography of a foraging people. Hawthorne, NY: Aldine

Hill K, Boesch C, Goodall J, Pusey A, Williams J, Wrangham R (2001) Mortality rates among wild chimpanzees. J Human Evol 39: 1–14

Hiraiwa Hasegawa M (1990) The role of food sharing between mother and infant in the ontogeny of feeding behavior. In: Nishida T (ed) The chimpanzees of the Mahale Mountains: sexual and life history strategies. Tokyo: Tokyo University Press, pp. 267–276

Hofman MA (1983) Energy metabolism, brain size and longevity in mammals. Quart Rev Biol 58: 495–512

Holliday MA (1978) Body composition and energy needs during growth. In: Falker F, Tanner JM (eds) Human growth. New York: Plenum Press, pp. 117–139

Jerison H (1973) Evolution of the brain and intelligence. New York: Academic Press

Jerison HJ (1976) Paleoneurology and the evolution of mind. Sci Am 234: 90–101

Kaplan HS (1997) The evolution of the human life course. In: Wachter K, Finch C (eds) Between Zeus and salmon: the biodemography of aging. Washington, D.C.: Natl Acad Sci, pp. 175–211

Kaplan H, Robson A (2001 a) The coevolution of intelligence and life expectancy in hunter-gatherer economies. London, Ontario: Department of Economics, University of Western Ontario

Kaplan H, Robson A (2001 b) The co-evolution of intelligence and lifespan and the emergence of humans. Albuquerque, NM: Department of Anthropology, University of New Mexico

Kaplan H, Robson A. in press. "The evolution of life expectancy and intelligence in hunter-gatherer economies." American Economic Review.

Kaplan H.S, A. Robson. 2002. "The emergence of humans: The coevolution of intelligence and longevity with intergenerational transfers." Proceedings of the National Academy of Sciences 99: 10221–10226.

Kaplan H, Hill K, Hurtado A.M, Lancaster J.B, Robson A. in press. "Embodied capital and the evolutionary economics of the human lifespan." Population and Development Review, Supplement.

Kaplan HS, Hill K, Lancaster JB, Hurtado AM (2000) A theory of human life history evolution: Diet, intelligence, and longevity. Evol Anthropol 9: 156–185

Kaplan HS, Gangestad S, Mueller TC, Lancaster JB (2001) The evolution of primate brains and life histories: A new model and empirical analysis. Albuquerque, NM: University of New Mexico

Kelly RL (1995) The foraging spectrum: Diversity in hunter-gatherer lifeways. Washington, D.C.: Smithsonian Institution Press

Kozlowski J, Wiegert RG (1986) Optimal allocation to growth and reproduction. Theoret Pop 29: 16–37

Lack D (1954) The natural regulation of animal numbers. Oxford: Oxford University Press

Lancaster JB (1986) Human adolescence and reproduction: An evolutionary perspective. In: Lancaster JB, Hamburg BA (eds) School-age pregnancy and parenthood. Hawthorne, NY: Aldine de Gruyter, pp. 17–39

Leibenberg L (1990) The art of tracking: the origin of science. Cape Town: David Phillip

Lloyd DG (1987) Selection of offspring size at independence and other size-versus-number strategies. Am Naturalist 129: 800–817

Marlowe F (2000) The patriarch hypothesis: An alternative explanation of menopause. Human Nat 11, pp. 27–42

Martin RD (1981) Relative brain size and basal metabolic rate in terrestrial vertebrates. Nature 293: 57–60

Martin RD (1996) Scaling of the mammalian brain: The maternal energy hypothesis. News Physiol Sci 11: 149–156

Milton K (1981) Distribution patterns of tropical plant foods as an evolutionary stimulus to primate mental development. Am Anthropol 83: 534–548

Milton K (1987) Primate diet and gut morphology: implications for human evolution. In: Harris M, Ross EB (eds) Food and evolution: toward a theory of human food habits. Philadelphia: Temple University Press, pp. 93–116

Milton K (1988) Foraging behaviour and the evolution of primate intelligence. In: Byrne RW, Whiten A (eds) Machiavellian intelligence. Oxford: Clarendon Press, pp. 285–305

Milton K (1993) Diet and primate evolution. Sci Am 269: 70–77

Milton K (1999) A hypothesis to explain the role of meat-eating in human evolution. Evol Anthropol 8: 11–21

Milton K, Demment M (1988) Digestive and passage kinetics of chimpanzees fed high and low fiber diets and comparison with human data. J Nutrit 118: 107

Parker ST, McKinney ML (1999) Origins of intelligence: the evolution of cognitive development in monkeys, apes and humans. Baltimore: Johns Hopkins Press

Partridge L, Harvey P (1985) Costs of reproduction. Nature 316: 20–21

Promislow DEL (1991) Senescence in natural populations of mammals: A comparative study. Evolution 45: 1869–1887

Rose MR, Mueller LD (1998) Evolution of the human lifespan: past, future and present. Am J Human Biol 10: 409–420

Ross C (1992) Basal metabolic rate, body weight and diet in primates: An evaluation of the evidence. Folia Primatol 58: 7–23

Sacher GA (1975) Maturation and longevity in relation to cranial capacity in hominid evolution. In: Tuttle R (ed) Primate functional morphology and evolution. The Hague: Mouton, pp. 417–441

Shanley DP, Kirkwood TBL (2000) Calorie restriction and aging: a life-history analysis. Evolution 54: 740–750

Silk JB (1978) Patterns of food sharing among mother and infant chimpanzees at Gombe National Park, Tanzania. Folia Primatol 29: 129–141

Smith BH (1991) Dental development and the evolution of life history in Hominidae. Am J Physical Anthropol 86: 157–174

Smith BH (1993) The physiological age of KNM-WT 15 000. In: Walker A, Leakey R (eds) The Nariokotome Homo erectus skeleton. Cambridge: Harvard University Press, pp. 196–220

Smith CC, Fretwell SD (1974) The optimal balance between size and number of offspring. Am Naturalist 108: 499–506

Stanford CB (1998) Chimpanzee and Red Colobus: the ecology of predator and prey. Cambridge, MA: Harvard University Press

Stiner M (1991) An interspecific perspective on the emergence of the modern human predatory niche. In: Stiner M (ed) Human predators and prey mortality. Boulder: Westview Press, pp. 149–185

Teleki G (1973) The predatory behavior of wild chimpanzees. Lewisburg, PA: Bucknell University Press

Worthman CM (1999) Evolutionary perspectives on the onset of puberty. In: Trevathan WR, Smith EO, McKenna JJ (eds) Evolutionary medicine. Oxford: Oxford University Press, pp. 135–163

Wrangham RW (1979) On the evolution of ape social systems. Soc Sci Info 18: 335–368

Wrangham RW, Smuts B (1980) Sex differences in behavioral ecology of chimpanzees in Gombe National Park, Tanzania. J Reprod Fertil (Suppl) 28: 13–31

Wrangham W (1975) The behavioral ecology of chimpanzees in Gombe National Park, Tanzania. Ph.D. dissertation, Cambridge University, Cambridge