

The Evolution of Human Life History

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Slow Life Histories and Human Evolution

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SUMMARY

Compared with other mammals, including other primates, humans take a long time to reach adulthood. Our very late maturity is often attributed to developmental and learning requirements that evolved with ancestral reliance on hunting. But other determinants may explain it. Across the mammals, age at first birth is correlated with a set of life history variables along a fast-slow continuum. I review data and theory showing that adult mortality rates are likely determinants of both age at maturity and maternal investment in individual offspring. Noting the riddle posed by increasingly expensive babies as life histories slow, I speculate that mechanisms of aging might explain this mammalian pattern. Perhaps when selection favors greater allocation to somatic maintenance and repair to slow aging, this lowers juvenile mortality as well, raising the marginal survival gains for additional investment in individual offspring. Whatever the merits of this suggestion, the relationships among adult lifespan, age at maturity, and rate of offspring production documented for mammals form the foundation for a previously proposed hypothesis that our very slow life histories are due to a novel productive role for ancestral grandmothers. I conclude by comparing and contrasting that hypothesis with

a recent version of the more venerable alternative that attributes our late maturity and expensive juveniles to ancestral reliance on hunting.

Slow human maturation has been a topic of interest in Western scholarship since before Darwin (Gould 1977). By mid-twentieth century, Adolph Schultz (1950, 1960) had shown variation in maturation rates across primates, noting that maturity seemed to be successively later in taxa more closely related to humans. A growing body of knowledge about primate social behavior led Sherwood Washburn and David Hamburg (1965:613) to surmise that “the adaptive function of prolonged biological youth is that it gives the animal time to learn.” Washburn incorporated this idea about slow maturation in his influential elaboration of the Hunting hypothesis to explain the evolution of many distinctively human features. He proposed that human maturity was delayed by the learning required for successful hunting. Building on Raymond Dart’s (1949) argument that the importance of hunting in savanna environments favored larger brains and bipedalism among human ancestors, Washburn argued that the shape of the pelvis in bipedal mothers, combined with the expanding brains of offspring, would create an obstetrical dilemma. Babies would have to be born at an “earlier stage of development” (Washburn 1960) and therefore need more care as infants, as well as a longer period of maternal dependence during which to grow and furnish those larger brains. This expanded offspring dependence would conflict with maternal hunting, leading mothers to pair with hunting mates. According to this hypothesis, nuclear families and sexual divisions of labor resulted from ancestral reliance on hunting because the larger brains favored in hunters necessitated the slow development and the extreme maternal dependence of human children.

Subsequent research showed the appearance of bipedalism, expanding brains, and archaeological evidence of big game hunting to be separated by millions of years (Johanson and White 1979; Klein 1999; Stiner 2000). Although the actual temporal record contradicted Washburn’s version of the Hunting hypothesis, elements of the hypothesis still remain compelling to some (for example, Kaplan et al. 2000; Kaplan and Robson 2002; Robson and Kaplan 2003). The idea that we take so long to mature because of the time required to grow and out-

fit our big brains is especially persuasive to many. I will return to the Hunting hypothesis after a lengthier review of the background and content of an alternative to this persistent idea.

Instead of constraints of juvenile requirements shaping ontogeny, selection may adjust juvenile development as a consequence of changes in adult mortality rates. The assumption that selection adjusts maturation rates in the face of adult mortality constraints is commonly used in evolutionary life history theory, an approach that derives from a combination of demographic theory and theory about natural selection shaping phenotypic trade-offs (see chapter 3). In the evolutionary model that is most successful at explaining cross-species regularities in life history variation, adult mortality rates determine age at maturity. I review the background, evidence, and elements of this model, focusing on cross-species regularities in life history variation and the likely role of adult mortality rates as the pacemaker of life histories. I then draw special attention to implications for links between aging rates and benefits for investment in offspring. In conclusion, I discuss implications of these regularities for human evolution, reviewing the Grandmother hypothesis and contrasting it with recent versions of the Hunting hypothesis.

EVOLUTIONARY LIFE HISTORY THEORY

Evolutionary life history theory (Fisher 1930; Lack 1947; Williams 1957, 1966a, 1966b; Hamilton 1966; Stearns 1992; Charlesworth 1994) regularly employs both mathematical demography (Lotka 1922; Keyfitz 1977) and optimality models (Maynard Smith 1978; Seger and Stubblefield 1996) to explain the wide diversity of life cycles in living things. Stable population theory from demography explains the interdependence of population vital rates. Whenever age-specific birth and death rates remain constant for a few generations (and migration is negligible), populations reach stable age distributions. The proportion of individuals in each age class does not change, whether or not the population is growing. The relative lifetime fitness effect of any change in fertility or mortality at a particular age can be calculated by using stable population models. Modelers sometimes assume that populations are not only stable but also stationary (nongrowing), a simplification justified by the geometric effects of population growth rates. Even

small departures from zero growth quickly lead to either extinction or unsustainably large populations, so growth rates must be near zero most of the time.

In addition to stable population models, evolutionary life history theorists use both standard and frequency-dependent optimality (or Evolutionarily Stable Strategy [ESS]) models to investigate the trade-offs imposed by a finite world. Because time and energy are limited, more allocated to one thing leaves less for something else. More into maintenance means less for current reproduction; more into individual offspring means fewer produced. The guiding premise is that natural selection favors tendencies and capacities to trade off these fitness components so as to enhance overall lifetime fitness. Because costs and benefits from various components depend both on an organism's own characteristics and on local circumstances, optimal allocations differ for individuals over time, among individuals within populations, and among populations of the same species. Capacities for adjustment to immediate circumstances might result in wide phenotypic variation. Yet, the strong regularities within taxonomic groups suggest that a limited number of fundamental trade-offs usually predominate (Charnov 1993; see Robson, van Schaik, and Hawkes, chapter 2, this volume).

Given general expectations about allocation trade-offs, some of the regular interrelationships among life history variables are initially puzzling. Paul Harvey and Timothy Clutton-Brock (1985) noted that in mammals generally and primates in particular, one might expect "compensation." Longer gestation length might mean shorter time to weaning, or later weaning might mean less time to maturity. Instead of negative correlations among these timing variables across primate species, though, they found just the reverse: "In particular, relative neonatal weight is positively correlated with relative gestational length, relative weaning age and relative age at maturity" (Harvey and Clutton-Brock 1985:578). These positive correlations multiply differences in potential reproductive rates, expanding the range of slow-fast variation.

Variation in adult body size is correlated with the slow-fast differences (Blueweiss et al. 1978; Western 1979; J. Eisenberg 1981; Western and Ssemakula 1982). Andrew Purvis and Paul Harvey (1995) compiled a mammalian life history data set that contains sixty-four species,

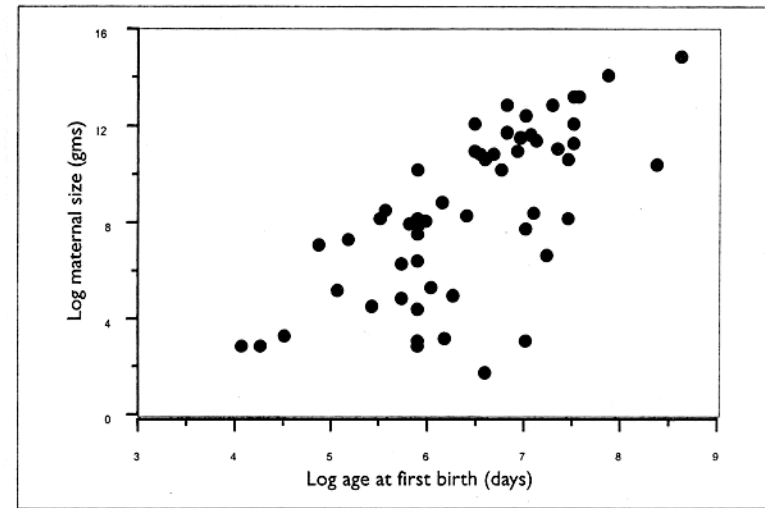
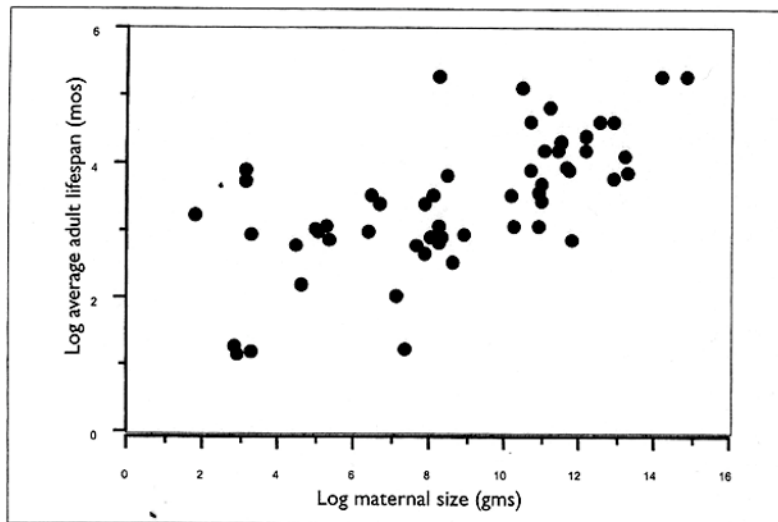


FIGURE 4.1

Natural log/log plot of adult body size by age at first birth across species of mammals. Data from Purvis and Harvey (1995) includes sixty-four species, ranging from rodents to elephants. This data set includes only two species of primates. Least squares regression $r = 0.684$.

ranging from rodents to elephants. Log/log plots of age at maturity and adult body size for these species (figure 4.1) confirm that larger-bodied species are slower to mature.

Because it takes longer to grow bigger, a correlation between age at maturity and body size is not surprising. But the magnitude of associated differences in reproductive rates can far outpace the differences of size: "For example, consider the smallest and the largest species of primate. A female mouse lemur (*Microcebus murinus*) born at the same time as a female gorilla (*Gorilla gorilla*) could leave 10 million descendants before the gorilla became sexually mature" (Harvey, Read, and Promislow 1989:14). After some feasible threshold, the time spent growing larger means reproduction foregone. There must be benefits that compensate this cost. Possible advantages for growing larger include predator defense, mobility over larger foraging ranges, and tolerance of lower diet quality or local resource fluctuations, all of which would reduce adult mortality risk (McNab 1963, 1980; Western 1979; Western and Ssemakula 1982; Clutton-Brock and Harvey 1983;

**FIGURE 4.2**

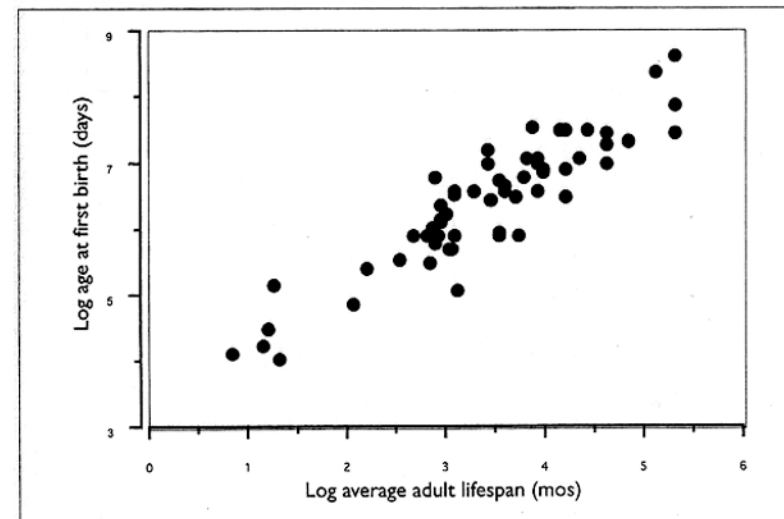
Natural log/log plot of average adult lifespan by adult body size. Data from Purvis and Harvey (1995); $n = 63$; $r = 0.669$ (missing data on one of the sixty-four species in this data set).

Pagel and Harvey 1993; Kozlowski and Weiner 1997). Growing larger might lower adult mortality.

Species with larger adult body sizes do have longer adult lifespans (figure 4.2), but the direction of causality could also run the other way. Larger body sizes could be a consequence of lower adult mortality risk. When this risk is lower, juveniles can afford a longer growth period and get larger before maturing (Kozlowski and Weigert 1987; Charnov 1990).

The positive correlation between adult lifespan and age at maturity is consistent with either direction of causality. Yet, the relationship between those timing variables remains when body size is removed (Harvey and Zammuto 1985; Sutherland, Grafen, and Harvey 1986; Read and Harvey 1989; Promislow and Harvey 1990). Average adult lifespan and age at maturity are more closely correlated with each other than either is with body size (figure 4.3; compare with figures 4.1 and 4.2).

Using order means as data points, Andrew Read, Paul Harvey, and Daniel Promislow (Read and Harvey 1989; Harvey, Read, and Promislow 1989) found that the relationship between adult lifespan and age at maturity remains but that orders change position relative to

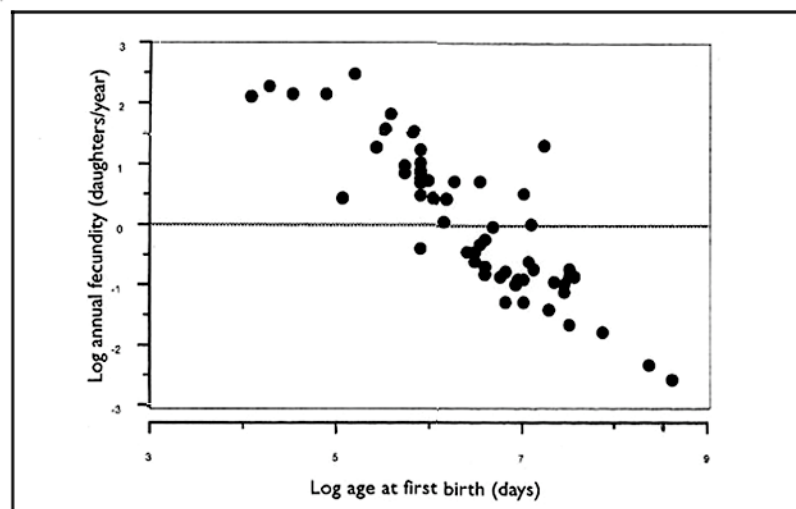
**FIGURE 4.3**

Natural log/log plot of age at first birth by average adult lifespan. Data from Purvis and Harvey (1995); $n = 63$; $r = 0.913$ (missing data on one of the sixty-four species in this data set).

each other on these fast-slow dimensions when body size is removed: "Species with high mortality rates for their body size had the suite of life history characteristics associated with the fast end of the fast-slow continuum: short gestation lengths, early ages at weaning and maturity, short periods from weaning to maturity, and large litters" (Harvey, Read, and Promislow 1989:23).

This could imply that adult mortality risk determines the other traits, with selection favoring delayed maturity when the mortality risk of waiting declines. Reproductive efficiency might, for example, improve with age (Harvey, Read, and Promislow 1989). Across the fast-slow continuum, the rate of offspring production is correlated with age at maturity, but the direction of the correlation is negative. Species that wait longer have babies at a slower rate (figure 4.4).

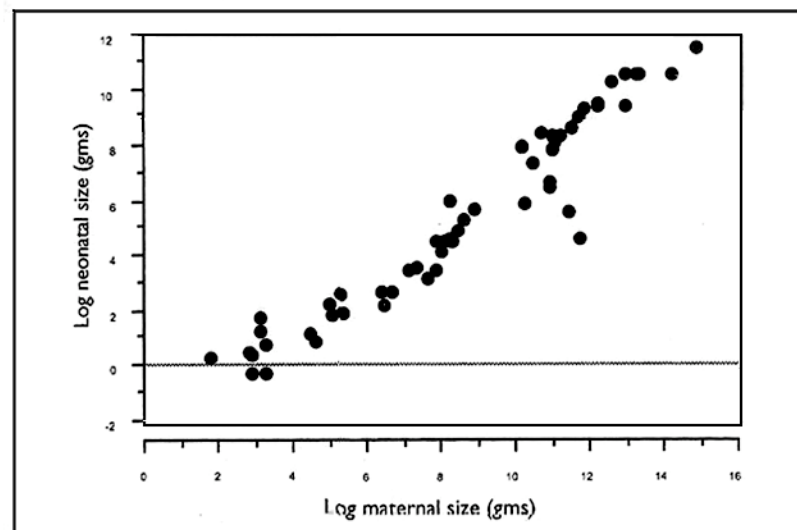
This is a central feature of slow mammalian life histories. Harvey, Read, and Promislow (1989:16–17) posed the puzzle this way: "[While] it is plausible that an upper limit on neonate size is sometimes set by the size of the birth canal or the ability of the placenta to sustain a large, growing fetus...it is by no means clear why elephants do not

**FIGURE 4.4**

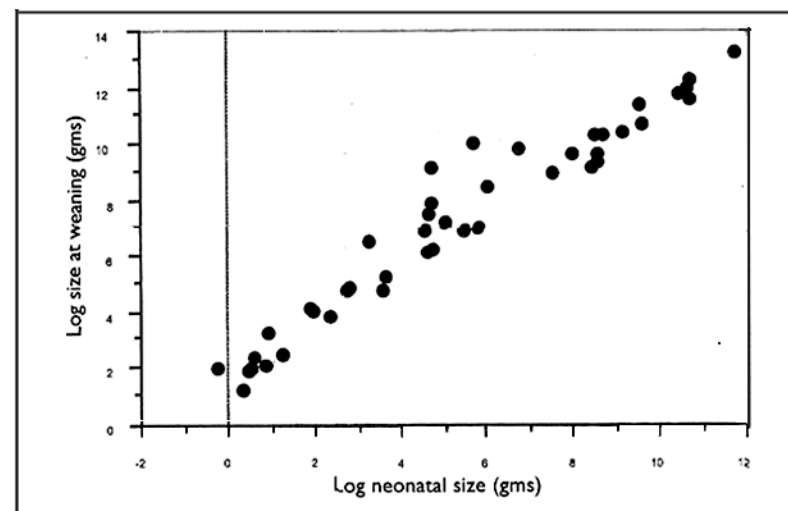
Natural log/log plot of annual fecundity (daughters/years) by age at first birth. Data from Purvis and Harvey (1995); $n = 59$; $r = 0.880$ (missing data on five of the sixty-four species in this data set).

produce two horse-sized or three hippopotamus-sized neonates, which together would weigh the same as an elephant at birth (and fit through the birth canal more easily)." In spite of the costs, baby size increases with the mother's size (figure 4.5). Greater postnatal investment in larger neonates then compounds the puzzle. Babies that are larger at birth are then larger at weaning (figure 4.6) (Millar 1977; Lee, Majluf, and Gordon 1991). Bigger mothers, instead of producing weanlings at a faster rate, produce bigger weanlings (figure 4.7).

This is part of the vast empirical evidence that selection does not simply maximize fertility. David Lack (1947, 1953) provided an explanation for the general pattern by pointing out the crucial trade-off between quantity and quality of offspring. Parents typically leave more *surviving* descendants when they divide their effort among fewer and allot more to each (Williams 1966b; Smith and Fretwell 1974). But why does greater investment in offspring quality regularly accompany delayed maturity, correlations that further reduce potential reproductive rates? Why do those who wait longer to mature then invest more in each baby, reducing their rates of offspring production even further?

**FIGURE 4.5**

Natural log/log plot of neonatal size by maternal size. Data from Purvis and Harvey (1995); $n = 60$; $r = 0.962$ (missing data on four of the sixty-four species in this data set).

**FIGURE 4.6**

Natural log/log plot of weanling size by neonatal size. Data from Purvis and Harvey (1995); $n = 43$; $r = 0.970$ (missing data on twenty-one of the sixty-four species in this data set).

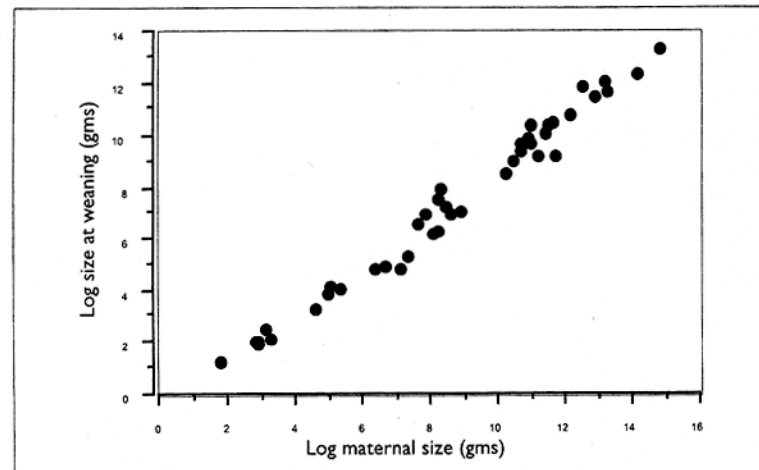


FIGURE 4.7
Natural log/log plot of weanling size by maternal size. Data from Purvis and Harvey (1995); $n = 42$; $r = 0.991$ (missing data on twenty-two of the sixty-four species in this data set).

CHARNOV'S 1991 MODEL

The description of these general regularities in mammals (and similar regularities in birds [Saether 1988]) and the evolutionary questions they pose were crucial developments in the late 1980s. Harvey and colleagues established the empirical associations among the life history variables and surmised that because the timing variables remain correlated when the effects of body size are statistically removed, body size might not be as important as previously assumed. Eric Charnov (1990) focused attention on indications that body size was nevertheless the scaffolding of the regularities. The timing variables all scale with body size in the same way. Consequently, the ratios of the variables (such as the ratio of age at maturity to average adult lifespan) remain the same across large changes in the individual variables themselves. Charnov underlined both the persistence of characteristic ratios across large changes of body size within taxa (for example, among mammals) and the differences in these ratios from one taxon to another (from fish to mammals to birds) (Charnov and Berrigan 1990b; Charnov 2002).

Charnov's (1991) model of mammalian life history evolution explained the invariance of these ratios across transformations of body

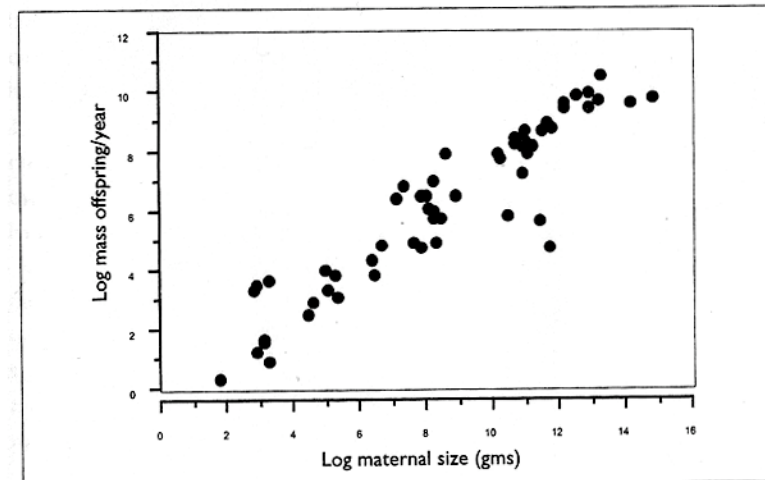


FIGURE 4.8
Natural log/log plot of offspring mass/year (neonatal weight times litter size times litters per year) by maternal size. Data from Purvis and Harvey (1995); $n = 56$; $r = 0.929$ (missing data on eight of the sixty-four species in this data set).

size. Building on his previous work and that of many others, he assumed a simple growth model in which a juvenile channels production into growing herself and at maturity redirects that production into offspring. Maternal body size is then the consequence of age at maturity. A simple exponential growth equation ($dW/dt = AW^{0.75}$) made growth an allometric function of body size (W) and a taxon-specific production function (A). As a juvenile grows bigger, she can mobilize more production for growing herself. The bigger she is at maturity, the more production she can put into growing offspring.

The simple growth model uses an average value (A) for the rate of production throughout juvenile life, ignoring the variation in growth trajectories at different ages. In exchange for subsuming those "evolutionarily labile" (Leigh and Park 1998) variables into rate averages, the model captures some broader patterns. Hill and Hurtado (1996) and Blurton Jones (chapter 8, this volume) show that it also accounts for general features of human growth. Charnov (1991) assumes that the benefit of growing larger is that larger mothers have more production available (mass/time) to put into babies (also a rate average). Figure 4.8 shows the broad mammalian relationship between maternal size

and the annual mass of offspring produced; the slope of the best-fit linear regression of this log/log plot (equivalent to the exponent in the growth equation) is 0.728.

In addition to the simple growth model, Charnov used a simplified mortality assumption: a high burst of early mortality (during which any density-dependent mortality occurs) dropping to a constant adult rate before some feasible age at maturity. Optimal age at maturity (α) then depends on the trade-off between the gains for continuing to grow larger and the risk of dying before reproducing. Later maturity is favored when that risk of dying declines.

Charnov's model accounts for the tighter correlations between timing variables themselves than between any of them and body size by recognizing the importance of (average) growth rate differences (Case 1978) and the covariation of these with the rate of offspring production. Both are represented by one variable, the production coefficient (A). Species characterized by a low ' A ' grow slowly and therefore are relatively small at a given age of maturity. They also have a low rate of offspring production at a given size (this variation contributes to the scatter in figures 4.1 and 4.2, compared with that in figure 4.3). Primates are smaller for a given age at maturity and produce babies at a slower rate than nonprimate mammals of a given size. Fitting data to the model, ' A ' averages about 0.4 in primates, compared with about 1.0 in mammals generally (Charnov and Berrigan 1993).

The puzzle of why rates of offspring production (across species) go down as age at maturity goes up is treated in Charnov's mammal model as follows. Productive capacity scales up allometrically with size at about 0.75 power (see figure 4.8, slope 0.728), whereas offspring size increases almost isometrically with maternal size. Babies are nearly a constant fraction of their mother's mass (the slope of the line in figure 4.7 is 0.955). Because baby size goes up faster than maternal productive rate, bigger mothers take longer to grow their offspring to weaning size and must turn them out at a slower rate.

The model is clear, simple, and consistent with the data. It explains the empirical relationships by treating age at maturity as one optimization (assuming a fixed offspring size), followed by a subsequent and independent second optimization that establishes a new parental quantity/quality equilibrium (ESS) (Charnov 1993, 2001b). Maternal size and fecundity are assumed to be positively related within a species

but negatively related across species because optimal investment per child changes among species. Across species, bigger mothers *do* make bigger babies (see figure 4.7), so they must make them at a slower rate. That is the empirical pattern. But the question raised by Harvey and colleagues above still has force: Why do bigger mothers make bigger, more expensive babies rather than use their greater productive capacity to make more babies?

Aspects of life histories not included in Charnov's initial mammal model might contribute to an explanation. Fast-slow life history variation entails not only differences in age at maturity and rates of offspring production but also varying rates of senescence. Both theory and data provide a basis for hypothesizing a link between aging and offspring investment.

AGING

Throughout an organism's life history, allocations to growth and reproduction compete with somatic maintenance (Williams 1957, 1966b; Gadgil and Bossert 1970). Kirkwood's (1977, 1981; Kirkwood and Rose 1991) disposable soma model focuses specifically on the question of varying allocation to maintenance. It is a version of Williams' (1957) antagonistic pleiotropy hypothesis about the evolution of aging, which explicitly focuses attention on the cost of maintenance and repair. If the processes of life result in inevitable damage to cells and tissues, accumulating impairment will increase vulnerability to mortality risks. The damage can be limited by buffering against it and can be reduced by repair, but allocation to somatic maintenance reduces allocation to growth and reproduction. From this perspective, accumulating damage is an intrinsic component of mortality risk that combines with extrinsic risks to determine the overall mortality rate (Cichon and Kozlowski 2000).

Trade-offs due to the cost of maintenance and repair imply a necessary qualitative relationship between extrinsic and intrinsic components of mortality risk. If extrinsic risk is high, then allocation to maintenance and repair is less beneficial because the chance of dying is high, no matter the repair. Conversely, if extrinsic risk is low, the chance of future benefit from repair is high. The present value of possible future payoffs may be considerable. Selection can favor trading off some growth and/or current reproduction for more maintenance

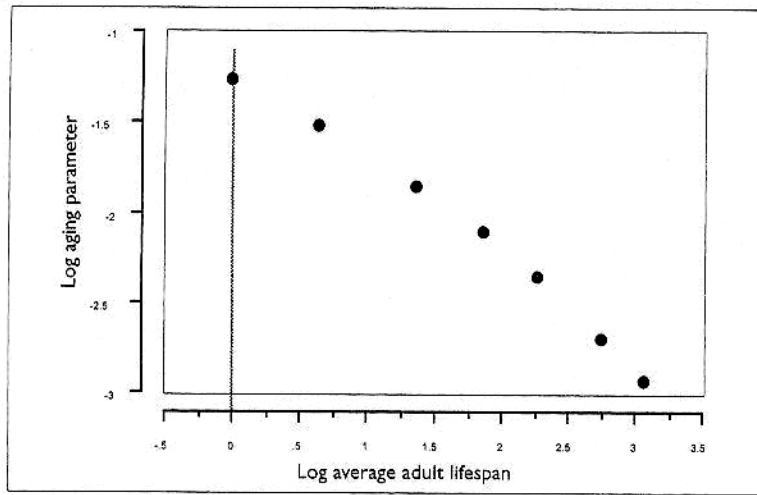


FIGURE 4.9

Natural log/log plot of rate of senescence (Ricklefs' aging parameter) by average adult lifespan. Estimates from Ricklefs (1998); modeling and data from eighteen bird species and twenty-seven mammal species.

and repair because an organism might live long enough to gain from persistent competence. As George Williams (1957:404) pointed out long ago, "low adult death rates should be associated with low rates of senescence, and high adult death rates with high rates of senescence...[so] we should be able to predict rates of senescence on the basis of adult mortality rates" (italics original).

Robert Ricklefs (1998) modeled and measured cross-species variation in intrinsic and extrinsic components of adult mortality in a sample of eighteen bird species and twenty-seven mammal species. He used the minimum mortality exhibited by an age class (the initial mortality rate) to estimate extrinsic mortality risks. Differences between the minimum rate and rates at older ages provided his estimate of intrinsic mortality, and a function fitted to changes in adult mortality with age indexed the rate of senescence.

Using these measures, Ricklefs showed two things. First, the rate of senescence increases with extrinsic mortality risk (figure 4.9). In species with high death rates, individuals age more quickly. Second, the fraction of adult mortality due to the intrinsic (senescent) component increases when adult lifespans lengthen (figure 4.10).

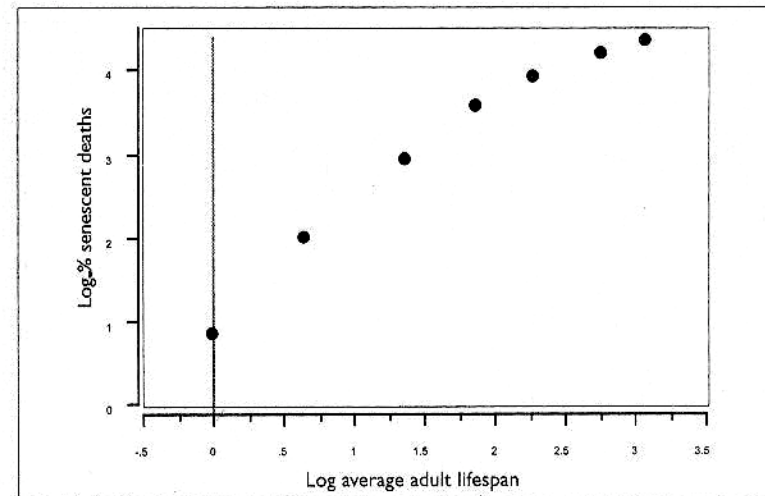


FIGURE 4.10

Natural log/log plot of the fraction of adult deaths due to senescence by average adult lifespan. The proportion of age-specific mortality above the initial mortality rate is death that would not have occurred if mortality rates remained constant with adult age. This death is assumed to reflect declining fitness-related performance with age. Estimates from Ricklefs (1998); modeling and data from eighteen bird species and twenty-seven mammal species.

High extrinsic mortality favors less repair and senescence is faster, but most individuals die before they have a chance to grow old. Conversely, when extrinsic mortality is low and more repair slows aging, the proportion of deaths due to senescence becomes remarkably high. The older the age structure, the more aging-related adult deaths.

Ricklefs' empirical demonstration is consistent with the theoretical expectation that benefits for maintenance and repair increase with the chance of future survival. As adult lifespans lengthen, aging rates slow. As life histories slow and populations have older age structures, the proportion of senescent deaths increases. Imperfect repair allows damage to accumulate, or, perhaps, as modeled by Cichon and Kozlowski (2000), allocation to repair varies with age. Either way, intrinsic mortality plays an increasingly dominant role in survival schedules when extrinsic mortality is low and populations have an older age structure.

There is another variable that should affect optimal allocation to somatic maintenance and repair. Williams (1957:406) predicted that

aging would be slower in organisms that continue to increase in fecundity after maturity: "[A]n increase in fecundity has the opposite effect from mortality." He was comparing the rapid senescence of determinant growers, such as mammals, with the slower senescence of indeterminant growers, such as many fish and invertebrates that continue to grow larger after maturity and produce more eggs as they get bigger. The point, however, is a general one. Charnov (1993) applied it to sex-changing fish. The same idea applies whenever individuals can have larger effects on their own fitness at older ages.

Greater allocation to somatic durability slows aging in adults. It might also shift life history allocations throughout development. More elaborate maintenance and repair might start early, perhaps from the initial stages of an individual's development, and affect the durability of juveniles similarly to that of adults. As more effective buffers against mortality are favored in adults, they will be reproduced in the offspring. Promislow and Harvey (1990) reported comparative results consistent with this reasoning. They found that "juvenile and adult mortality were significantly and positively correlated after removing the effects of body weight (which accounts for over 65% of the total variance in mortality). Species tend to have high or low mortality for their body weight throughout their lifetime" (Promislow and Harvey 1990:428).

The cross-species correlation between juvenile and adult mortality rates (figure 4.11) could result simply from adjustments in the timing of maturity as a consequence of adult mortality rates (Charnov 1991), as discussed above. If maturity is delayed when adult mortality is low, then the juvenile period includes more time at low mortality rates, reducing the average juvenile mortality rate. The correlation is also consistent with differences in somatic durability throughout life. Species with more durable adults may also have more durable juveniles.

Biodemographers use the minimum mortality rate exhibited by an age class (the initial mortality rate) to index extrinsic mortality (Finch 1990; and Ricklefs 1998, as discussed above). By the argument above, however, that rate is likely affected by intrinsic mortality buffers, not just extrinsic risk. Physiological processes of maintenance and repair should affect the vulnerability of juveniles just as they affect adults. Finch, Pike, and Whitten (1990) noted large differences in the initial mortality rates of various laboratory and captive populations.

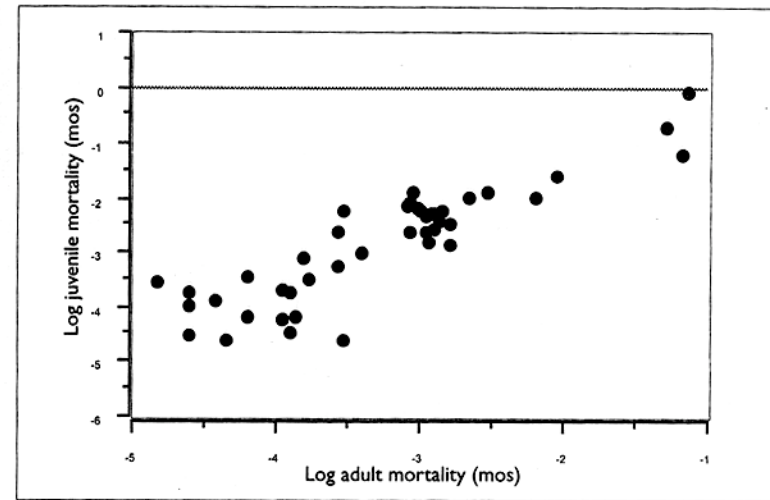


FIGURE 4.11

Natural log/log plot of juvenile mortality rate by adult mortality rate across mammals.

Data from Purvis and Harvey (1995); $n = 49$; $r = 0.891$ (missing data on twenty-five of the sixty-four species in this data set).

Husbandry conditions are surely implicated, but other differences may also play a role. Because extrinsic sources of mortality are substantially reduced in captivity, initial mortality rates may also depend on intrinsic processes of maintenance and repair in juveniles. Finch and colleagues suggested that evolution might adjust not only rates of aging but also initial mortality rates.

If selection for more durable adults results in more durable infants and juveniles, then this might systematically shift the mother's gain curve for investment in each offspring. Greater marginal gains for additional investment could help explain why longer-lived mammalian mothers invest more in each offspring. This line of reasoning prompts the hypothesis that cell and molecular processes associated with differential aging may link adult mortality rates with juvenile mortality rates and also link mammalian aging rates with the optimal quality/quantity trade-off for offspring investment.

WHAT ABOUT HUMAN EVOLUTION?

These general mammalian fast-slow regularities provide a framework for investigating the evolution of human life histories. Modern

humans are a very recent species, only one hundred thousand to two hundred thousand years old, whereas the ancestors we share with modern chimpanzees lived about six million years ago (King and Wilson 1975; Klein 1999; Glazko and Nei 2003). Over the past six million years, many different species evolved in our lineage (see Skinner and Wood, chapter 11, this volume). Genetic, morphological, and biochemical evidence from both living and ancient populations calibrates some of the similarities and differences among them. However, all past members of our lineage are known only from the fossil and archaeological record. It is impossible to observe their vital rates. This does not mean that the age structures of these populations could have been anything at all. Like the laws of physics and chemistry, demographic imperatives apply to persistent populations at any time and place, those in the past as well as the present. Ancient populations must have reproduced themselves at replacement rates; otherwise, they would have overrun the planet or disappeared. If we can infer some things about the life histories of individuals in past populations, this will dictate the probable range of other life history variables (Cole 1954; Keyfitz 1977; Charlesworth 1994; Charnov 1997).

The regularities of the fast-slow variation in mammalian life histories inspire even stronger presumptions. Life history variation reflects fundamental trade-offs for living primates, including the most unusual primate, modern humans. Therefore, those trade-offs likely applied to others in our hominin clade as well. Across the living primates, the fast-slow variation is broadly correlated with body size, for example, the mouse lemur/gorilla comparison. The data set compiled by Caroline Ross and Kate Jones (1999) shows that primates display the same relationships among life history variables and size found in the Purvis and Harvey (1995) data set for mammals as a whole. Primate species with longer adult lifespans mature later (figure 4.12). Species with later maturity are larger (figure 4.13). Larger species put more production into babies (figure 4.14). Bigger mothers have bigger newborns (figure 4.15), which they then nurse longer (figure 4.16). Having more expensive babies, mothers have them at a slower rate (figure 4.17). The later the age at maturity, the slower the rate of baby production (figure 4.18).

Body size and age at maturity are two variables that can be indexed in the fossil record (though not without difficulty; see, for example, Kappelman [1996]; R. Smith [1996]). The evolutionary life history

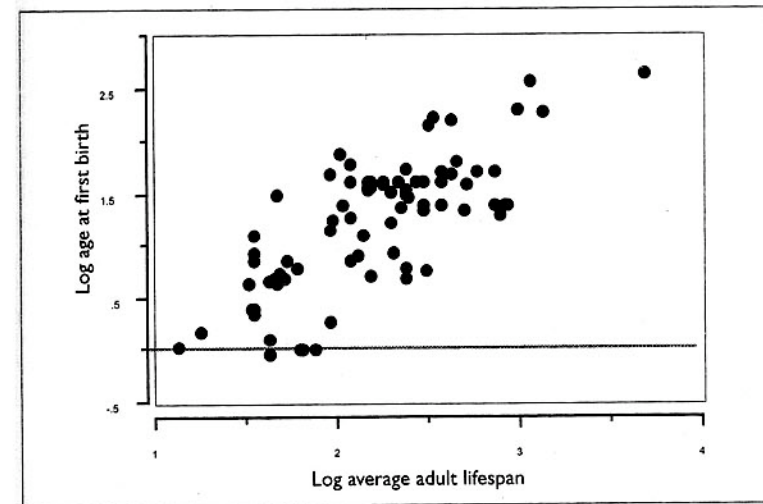


FIGURE 4.12

Natural log/log plot of age at first birth by average adult lifespan. Data in Ross and Jones (1999) includes one hundred primate species. Here $n = 78$; $r = 0.754$ (missing data on twenty-two of the one hundred species in this data set). Average adult lifespan is calculated from the maximum recorded longevity (Ross and Jones 1999:table 4.2) by Charnov's method (1993:104, fig. 5.6).

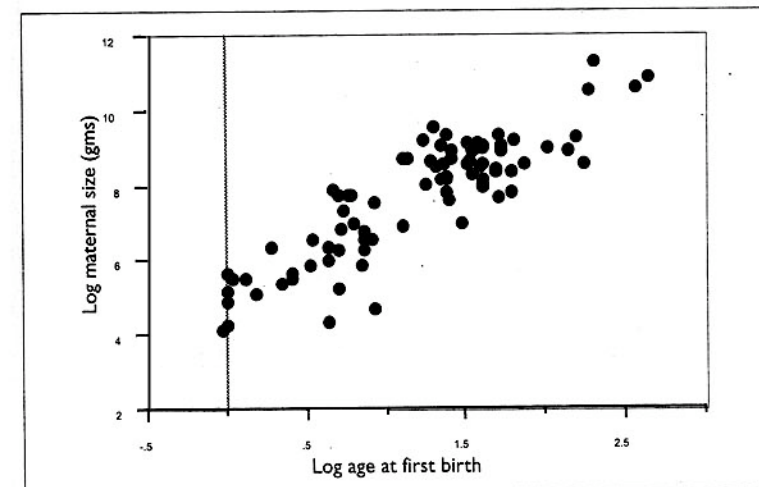
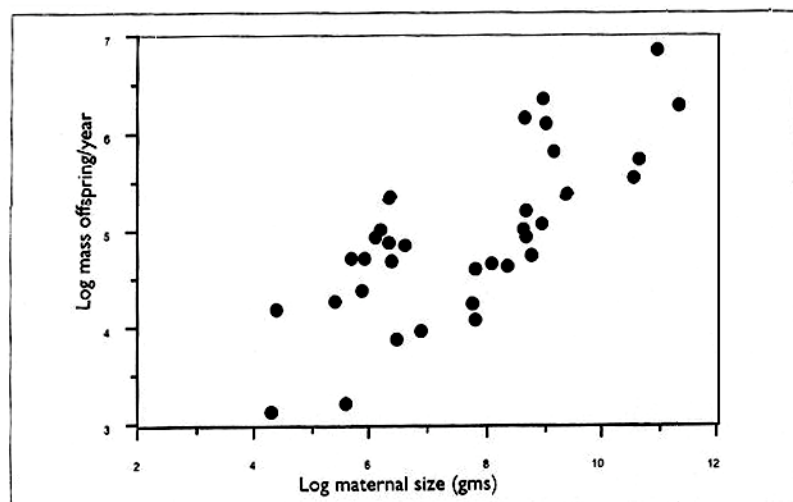
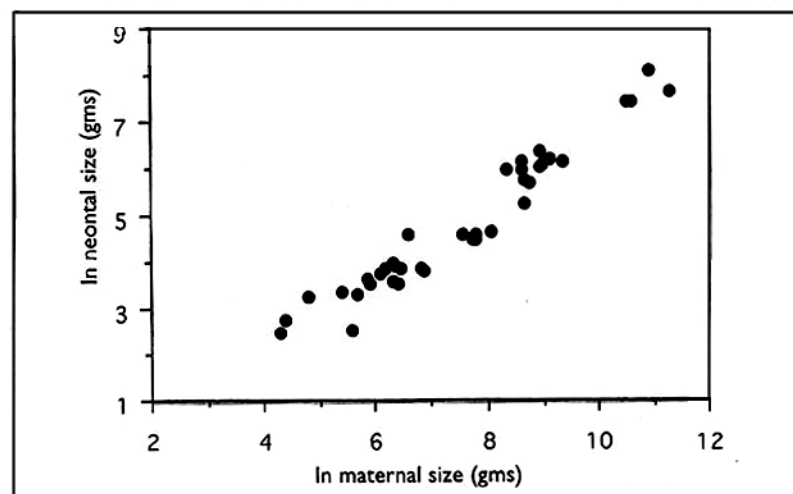


FIGURE 4.13

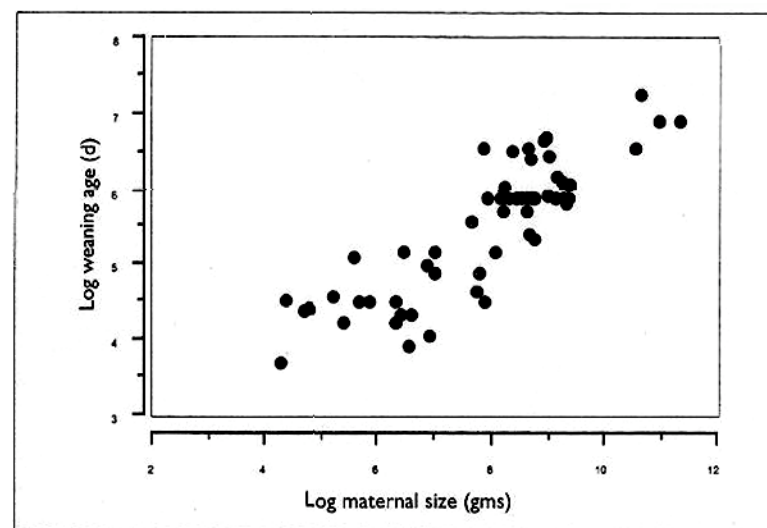
Natural log/log plot of adult body size by age at first birth. Data from Ross and Jones (1999); $n = 88$; $r = 0.871$ (missing data on twelve of the one hundred species in this data set).

**FIGURE 4.14**

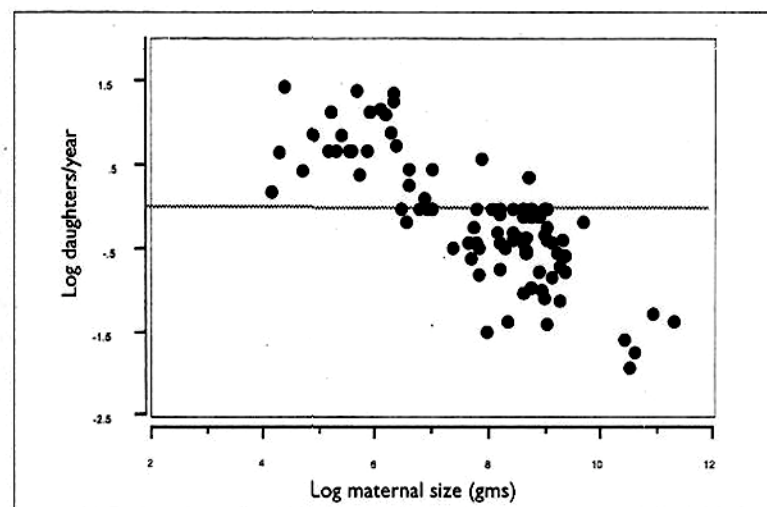
Natural log/log plot of offspring mass/year (neonatal size times litter size divided by interbirth interval) by maternal size. Maternal size from Ross and Jones (1999); neonatal weights, litter size, and interbirth interval from Chapman, Walker, and Lefebvre (1990); $n = 34$; $r = 0.755$.

**FIGURE 4.15**

Natural log/log plot of neonatal size by maternal size. Data from Ross and Jones (1999) and Chapman, Walker, and Lefebvre (1990); $n = 38$; $r = 0.970$.

**FIGURE 4.16**

Natural log/log plot of weaning age by maternal size. Data from Ross and Jones (1999); $n = 60$; $r = 0.852$ (missing data on forty of the one hundred species in this data set).

**FIGURE 4.17**

Natural log/log plot of annual fecundity (daughters per year) by maternal size. Data from Ross and Jones (1999); $n = 88$; $r \approx 0.800$ (missing data on twelve of the one hundred species in this data set).

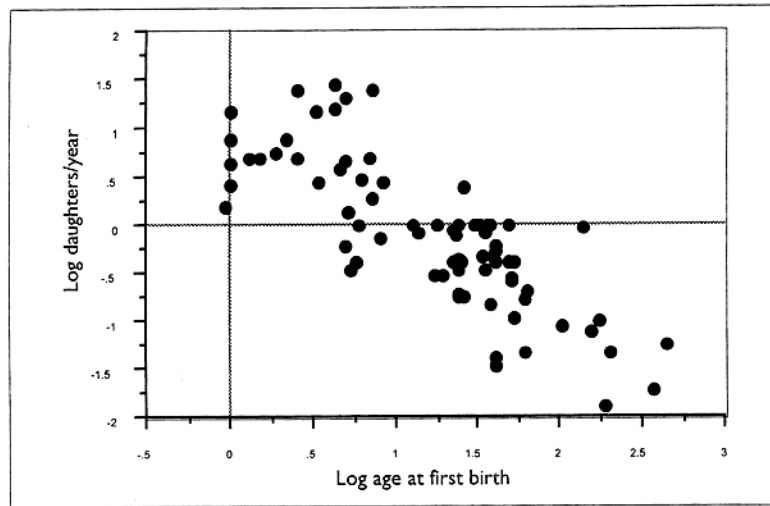


FIGURE 4.18

Natural log/log plot of annual fecundity (daughters/year) by age at first birth. Data from Ross and Jones (1999); $n = 78$; $r = 0.83$ (missing data on twenty-two of the one hundred species in this data set).

theory summarized above assigns the main causal role for the timing of maturation to adult mortality risk. From that perspective, maternal body size depends on the length of the juvenile period. When maturity is later, growth continues longer before it stops and production is redirected into offspring, so later maturity results in larger adult size (see Blurton Jones, chapter 8, this volume). Using this theory (and evidence from living species), adult body size and maturation age estimates imply likely ranges for other life history features.

Focus on these variables highlights a notable life history shift with the appearance of the first widely successful members of genus *Homo*. Earlier hominins, the australopiths (used in the broad sense to include *Australopithecus* and *Paranthropus*) are similar in body size and maturation to modern chimpanzees (Bromage and Dean 1985; B. Smith 1989a, 1991a; McHenry 1992, 1994; Smith and Tompkins 1995). The several taxa in this australopith radiation also have brains about the size of modern chimpanzees and have been characterized as “bipedal apes” (Klein 1999; see Skinner and Wood, chapter 11, this volume).

Brain size increases with the appearance of genus *Homo* around

two million years ago. Because body size also increases substantially, though, the *relative* brain size change is less dramatic (McHenry 1992, 1994; Aiello and Wheeler 1995; Wood and Collard 1999b). Collard and Wood (1999:324) go so far as to suggest that “although there are twofold differences in the mean absolute brain size of early hominids, these differences are almost certainly not significant when body mass is taken into account. A notable effect of body-mass correction is that the absolutely larger brain of *H. ergaster* is ‘cancelled out’ by its substantial estimated body mass.”

The larger body size of this taxon is associated with a later age at maturity than that which characterized the australopithecines (B. Smith 1991a, 1993). *H. ergaster/erectus* was similar to modern humans in body size and shape (Aiello and Key 2002), with maturation patterns perhaps in the range of modern humans (B. Smith 1994; Tardieu 1998; Clegg and Aiello 1999; Antón 2002a; S. Smith 2004; but see Dean et al. 2001; Skinner and Wood, chapter 11, this volume). The exceptionally large brains that distinguish modern humans, however, did not appear in our lineage until the radiation of “archaic” sapiens, which began at least a million years after the spread of the first widely successful members of our genus (see McHenry 1994; Ruff, Trinkaus, and Holliday 1997; Klein 1999; Skinner and Wood, chapter 11, this volume).

From the theoretical perspective outlined above, delay in maturity and growth to larger adult size are favored when lifespans lengthen. Otherwise, gains from growing larger are outweighed by the risk of dying before the benefits of larger size can be realized. Assuming that australopiths had life histories resembling those of modern chimpanzees (Smith and Tompkins 1995), they were at the slow end of the primate variation, with adult lifespans long enough to allow quite late maturity. Using Ricklefs’ findings of the fraction of senescent deaths associated with chimpanzeelike lifespans, most adult deaths (about 70 percent) would have been due to senescent decline. What could have slowed rates of aging even further among the first widely successful members of our genus?

A GRANDMOTHER HYPOTHESIS

Observations among Hadza hunter-gatherers in northern Tanzania (Woodburn 1968; Blurton Jones et al. 1992) suggest an answer (Hawkes et al. 1998; Blurton Jones, Hawkes, and O’Connell 1999; O’Connell,

Hawkes, and Blurton Jones 1999). Among these modern people, children are productive and energetic foragers (Blurton Jones, Hawkes, and O'Connell 1989) but cannot effectively handle one essential dietary staple, the large root of a plant that is deeply buried and requires strength to excavate (Hawkes, O'Connell, and Blurton Jones 1995). Women past menopause are especially active tuber diggers (Hawkes, O'Connell, and Blurton Jones 1989). In this population, a mother's foraging effort has a measurable effect on her children's nutritional welfare, except when she bears another baby and turns her efforts to nursing her newborn. Then it is the work of postmenopausal grandmothers that differentially affects the nutritional welfare of weaned children (Hawkes, O'Connell, and Blurton Jones 1997).

The Hadza patterns underline an important consequence of the ecological changes that coincided with the appearance of our genus (O'Connell, Hawkes, and Blurton Jones 1999). Drying environments in the late Pliocene (Cerling 1992; deMenocal 1995) probably constricted the availability of foods that young juveniles can handle. Intensified aridity and seasonality would likely have favored plants that cope well with dry seasons, for example, by holding nutrients in hard-cased seeds, nuts, and underground storage organs. Such resources can give high return rates to human foragers with the strength and skill to extract and process them, which young juveniles lack. To depend on these resources and succeed in these environments, mothers have to provision offspring who are still too young to extract and process the foods for themselves. The mother-offspring provisioning allows the occupation of otherwise uninhabitable environments. The fossil and archaeological evidence shows that *Homo ergaster/erectus* is the first hominin found outside Africa, as far east as Indonesia, and as far north as latitude 45 degrees (Swisher et al. 1994; Gabunia et al. 2000; Larick et al. 2001; Zhu et al. 2001).

Such a difference in feeding ecology is proposed to have the following consequence for life history evolution: maternal provisioning of weaned offspring creates a novel fitness opportunity for older females whose own fertility is declining. If the older females help feed their weanling grandchildren, the mothers of those weanlings can have shorter interbirth intervals without reductions in offspring survivorship. Thus, the more vigorous elders without nursing infants of their own can raise their daughters' reproductive success, increasing the

number of descendants who share their characteristics. By this pathway, selection would favor greater allocation to physiological processes that buffer against mortality risk, slowing the rate of senescence through enhanced somatic maintenance and repair.

More allocation to somatic maintenance means less to current reproduction. If the ancestors of our genus were close to evolutionary stability for that trade-off, more maintenance and repair (slower aging) could only be favored if there were a benefit that outweighed that cost in current reproduction during the childbearing years. In this Grandmother hypothesis, the cost is paid by the contribution the older females make to the fertility and survival of their younger kin.

TWO INVARIANTS, A FALSIFICATION, AND HUMAN AGE STRUCTURES

Hawkes and co-authors (1998) argued that differences between humans and other living apes on three life history variables—adult mortality (M), age at maturity (α), and annual fecundity (b)—are consistent with the Grandmother hypothesis and Charnov's (1991) mammal model. Alvarez (2000) compiled a sample of sixteen primate species to evaluate the similarities and differences statistically. If the entire adult lifespan in humans is spent contributing to the production of descendants, then age at maturity (α) should be adjusted to adult mortality (M). Alvarez used the foraging Aché (Hill and Hurtado 1996) to represent humans and confirmed that the late age of first birth in this population—similar to the age of first birth for the !Kung (Howell 1979) and the Hadza (Blurton Jones et al. 1992; Blurton Jones, Hawkes, and O'Connell 2002)—is the age of first birth (α) predicted by the mammalian invariant (αM). Ross and Jones' (1999) data set is larger and much noisier than the data used by Alvarez but shows the same thing. Humans are within the 95 percent confidence interval when age at maturity (α) is regressed on average adult lifespan (figure 4.12 shows the relationship between these variables).

Alvarez (2000) also found human annual fecundity to be higher than expected from the mammalian αb . The Aché birthrate (b , annual fecundity) is above the 95 percent confidence interval for the regression of (b) on age at maturity (α) in her sixteen-species sample. The Grandmother hypothesis predicts the human rate of baby production to be higher than that of a primate with our age at maturity and no

grandmothering, because our childbearing rate is elevated by grandmothers' help. Alvarez's (2000) statistical results are therefore consistent with the claim that Hawkes and colleagues (1998) made about high human fecundity based on comparisons among humans, chimpanzees, gorillas, and orangutans.

There is another implication of this argument to test from the same data, an implication highlighted by the previous discussion of connections between slowed aging and more expensive offspring. With humans' slow rates of aging, we should have very expensive babies. The Grandmother hypothesis predicts annual fecundity during the childbearing years to be high for our age at maturity, not because human mothers favor more quantity and less quality than expected but because grandmothers' production subsidizes the production of the childbearers. If all the adult women are counted as contributors, the rate per contributor should be consistent with the mammalian invariant. Averaged over both childbearing and grandmothering years, human fecundity should be lower than the average in the other living apes and consistent with the rate predicted for a primate with our late age at maturity.

That test has now been superseded by Alvarez's subsequent work, investigating the possibility that we (Hawkes et al. 1998; Alvarez 2000) may have mistaken features resulting from high growth rates in human populations for real life history characteristics. Because human populations are usually growing while many other primate populations, great apes in particular, are not, the life history differences we have inferred from using observed fecundities may be biased by the population growth differences. This is an important concern. Alvarez's (2004a) new analysis shows that we did indeed overestimate human fertility.

Charnov's mammal model assumes stationary populations. Adjusting observed values for both human and chimpanzee populations to make them stationary, Alvarez (2004a) then used the stationary models to estimate the variables just discussed. The stationary models retain the characteristic features of both species. Adult mortalities in humans are lower than in chimpanzees, so average adult lifespans are longer, with about a quarter of the adult women past childbearing age. The human fertilities are substantially lower than

chimpanzee fertilities in the stationary populations. Whether calculated over the childbearing years only or over all the adult years, the human fertilities are within the confidence interval for fecundity regressed on age at maturity in Alvarez's sixteen-species sample.

Because average adult mortality rates are lower in the human than in the chimpanzee stationary models (as they are in observed populations), the rate at which juveniles become adults in human populations must also be slower than in chimpanzees. As in the observed populations, the fraction of adults past the childbearing age is large in the stationary human populations but negligible in the chimpanzees. Necessarily, then, the human rate of baby production is higher when calculated as fertility during the childbearing age than when averaged over all women. In contrast, for chimpanzee populations, whether baby production is distributed over all the adult females or only those of childbearing age makes little difference because so few females are alive after terminal fertility in that species. The results do not support our previous claims that human fertility is high for our late maturity, but they underline the distinctively long adult lifespans and old age structure of human populations. We put more into offspring and produce them at a slower rate, as expected for a primate with our old population age structure.

The old age structure of human populations is often mistakenly assumed to depend on long life expectancies. Life expectancies much over 40 may be a relatively recent novelty (Oeppen and Vaupel 2002), and many assume that long average adult lifespans are also recent (such as Washburn 1981; Crews and Gerber 2003; Kennedy 2003; Krovitz, Nelson, and Thompson 2003; Nelson, Thompson, and Krovitz 2003). But life expectancies are averages, strongly affected by the short lives of those who die as infants and juveniles. This is clearly illustrated by using the stable population models built by Coale and Demeny (1966) to examine the effects of varying mortality on population age structure. With fertility held constant, a tripling of life expectancy (from 20 to 60) makes little difference in the fraction of adults over 45 (Hawkes 2004a; Hawkes and Blurton Jones 2005). Moreover, the direction of the difference is opposite to usual intuition: the lower the life expectancy, the *larger* the fraction of women past the age of terminal fertility. In hunter-gatherer populations with life expectancies lower

than 40, a quarter or more of the adult women are past the age of 45 (Hawkes 2003, 2004a; Hawkes, O'Connell, and Blurton Jones 2003; Hawkes and Blurton Jones 2005).

To explain this pattern, the Grandmother hypothesis proposes that aging slowed and adult lifespans lengthened in our lineage when females who were slightly more vigorous as their fertility declined could have a novel effect on their own fitness. Without infants of their own, the older females could help their daughters feed weanlings who were unable to handle increasingly important food resources. As more vigorous elders helped more, their vigor was represented in more descendants. Slower aging and longer average adult lifespans decreased the risk of waiting longer to mature, resulting in a larger pool of adolescents who might also help their mothers and younger siblings, while remaining immature longer. Middle-aged adults and teenagers are conventionally described as post- and prereproductive, but they contribute to the reproductive success of childbearers. Sarah Hrdy (1999, 2001) has characterized humans as cooperative breeders, underlining the importance of flows of help to mothers and young juveniles. Resource transfers, not only from mothers to offspring but also from grandmothers, older siblings, and others, are long appreciated features of the socioecology of our species (see Sahlin 1972; Davis and Daly 1997; Ivey 2000).

COMPARING GRANDMOTHERING WITH THE HUNTING HYPOTHESIS

This Grandmother hypothesis is similar in some meaningful respects to the Hunting hypothesis summarized at the beginning of this chapter. Both point to the same expanding savannas to trigger adaptive shifts in the human lineage. Both hypotheses involve resource transfers between generations—maternal and especially paternal provisioning, on one hand, and maternal and grandmaternal provisioning, on the other. Changes in aging rates, which are central to the Grandmother hypothesis, are also major elements of recent versions of the Hunting hypothesis (Kaplan et al. 2000).

A central difference is the grandmothering model's focus on female life history trade-offs. In contrast, the Hunting hypothesis makes the productivity of hunting *men* the source of payoffs for longer juvenile

development (Hill and Kaplan 1999). Kaplan and colleagues have elaborated this in detail (Kaplan 1996, 1997; Kaplan et al. 2000; Kaplan and Robson 2002; Robson and Kaplan 2003), characterizing brains and learning as embodied capital that generates surplus production from hunting men. Using data on foraging return rates and time allocation among hunter-gatherers, they estimate average age- and sex-specific energy consumption and production rates and highlight the net energetic deficit imposed by juveniles, as well as the net energetic surplus provided by men's hunting. If the production profiles reflect age- and sex-specific capacities, then the surplus from men is necessary to pay the juvenile deficits. An economic and reproductive partnership between men and women is then required. Assuming that partnership, the number of new dependent juveniles that can be recruited depends on the size of the surplus. These assumptions justify the expectation that investments in growth and mortality reduction that maximize surplus production will be favored by selection. Modeling those investments, Kaplan and Robson (2002; Robson and Kaplan 2003) find that when greater investments in embodied capital raise the lifetime surplus produced, the surplus is further increased by investments in mortality reduction.

By contrast, the Grandmother hypothesis attends explicitly to female life history trade-offs, proposing that slowed aging is favored by the contribution that older females make to the survival and fertility of their junior kin. These fitness effects from grandmothering result in competitive advantages for lineages in which aging is slower than in ancestral populations. This results in lower adult mortality and consequent changes in other life history variables. Males and females are not assumed to have the same productive and reproductive interests.

Alternative models ignore different aspects of the empirical complexity. Choices among simplifications—as with everything else—involve trade-offs. The Grandmother hypothesis is built on a model of mammalian life history evolution, which, like much of life history theory, is about female trade-offs. For mammals, life history questions such as when to stop growing and have the first baby; whether to have singletons, twins, or more offspring per pregnancy; and when to wean and move on to the next baby are questions about female life histories. Males, of course, face life history trade-offs as well, but different ones.

Charnov's model of mammalian life history invariants provides "assembly rules" for female life histories. The variation in age at maturity and in annual fecundity across mammals generally, and within the order primates, is correlated with adult female lifespans—irrespective of male strategies.

In humans, as in most mammals, including other primates, child-care is women's work. Our species is unusual for the economic productivity of adult males. Men, unlike males in most primate species, expend substantial effort producing food that is consumed by females and juveniles. This surplus production, central to the Hunting or Embodied Capital hypothesis, plays no role in the Grandmother hypothesis. The omission is not because those investigating the evolutionary role of grandmothers dismiss the importance of hunting and men's activities. Our ethnographic experience confirms the high value placed on meat and hunting in modern hunting and gathering communities, prompting inquiry explicitly focused on hunting and men's foraging and reproductive strategies (Hawkes 1990, 1991, 1993, 2000, 2001, 2004a; Hawkes, O'Connell, and Blurton Jones 1991, 2001a, 2001b; Blurton Jones et al. 2000; Hawkes and Bliege Bird 2002; O'Connell et al. 2002). Main findings include the strategic adjustments that individuals of different ages and sexes make in their foraging behavior and the lesson that—in these ethnographic cases, at least—hunting is not satisfactorily explained as paternal provisioning. Hunting successes are unpredictable, meat is claimed by many, and the hunter does not control the distribution of shares. Differential nutritional benefits to the hunters (and their own wives and children) are all but absent. Yet, better hunters often spend more time hunting, inflating the contributions they make to others' consumption. Social benefits for the hunters seem more likely to explain the patterns than do nutritional benefits for their own families.

The ethnographic findings point to the importance of sexual selection (Darwin 1871) in human social behavior. Different reproductive interests of the sexes and the relevance of mating competition among males are central to the "ecological model" for explaining social arrangements in other primates (Wrangham 1979, 1980; van Schaik 1983, 1989, 1996; Kappeler and van Schaik 2002; Hawkes 2004b). Because the same things are important among modern humans, it is

especially likely that they were also important in the lives of our ancestors. The earliest assemblages of stone tools and large animal bones are associated with ancestors who lacked the projectile weapons that make modern men such effective hunters. But social benefits to males for showing off their qualities as competitive scavengers can explain otherwise puzzling aspects of the early archaeology (O'Connell, Hawkes, and Blurton Jones 2002). Social competition and costly signaling models promise to be more effective than paternal provisioning in explaining the evolution and character of men's work (Hawkes and Bliege Bird 2002; Bliege Bird and Smith 2005).

The Grandmother hypothesis is silent on male life history trade-offs, except in one way. Arguments about slowed aging in our lineage do have implications for male longevity. As greater allocation to somatic maintenance and repair is favored through the increased fitness of more vigorous grandmothers, those characteristics would be represented in their descendants of both sexes. Longer-lived women not only have more expensive and longer-lived daughters (and granddaughters) but also longer-lived and more expensive sons (and grandsons). Male-male competition can lead to higher adult mortality rates in males than females, with higher aging rates expected as a consequence, but longevity in females is a good general predictor of longevity in males across primate species (Allman et al. 1998).

CONCLUDING COMMENT

The fast-slow life history continuum implies the predominance of a few basic trade-offs across the broad range of mammalian life histories. The evolutionary optimization model that best explains the cross-species patterns assumes that continued growth is current reproduction traded for greater reproductive capacity in the future. The present value of those future benefits depends on adult mortality risk. Theory, combined with the empirical patterns, also links mortality risk to variation in somatic durability, maintenance, and repair. More fitness opportunities at later ages increase the net benefits for somatic maintenance. Slower life histories favor more effective intrinsic buffers against somatic damage, so much so that the rate of aging can become the main determinant of adult mortality rates.

Varying investment in somatic maintenance and repair should also

affect allocation to production of self (juvenile growth rate) and babies. In Charnov's 1991 model, he assumed growth and reproductive rates to be independent of mortality rates to explain the scaling regularities in adult lifespans, age at maturity, and annual fecundity. A recent (Charnov 2004) modification attends specifically to trade-offs between production (growth and reproduction) and maintenance, showing that longer-lived species may be more efficient at maintenance and repair. This model is of special interest because it also reproduces the scaling regularities in the life history variables that are real aspects of the empirical variation.

Ronald D. Lee (2003) has developed a formal theory of intergenerational transfers to explore the evolution of slower aging rates. Lee's model does not deal with scaling patterns among life history variables, but it does show that when intergenerational transfers of resources are important, it is the remaining economic productivity, not fertility, that determines the rate of senescence. Links between economic productivity and aging are also central to Kaplan and colleagues' Embodied Capital model. Their verbal arguments emphasize expanding brains and hunting, but their formal model (Kaplan et al. 2000; Kaplan and Robson 2002; Robson and Kaplan 2003) includes more general trade-offs among growth, mortality buffers, and production. Differences among these various approaches are not small ones. One of them, the role of the broader primate regularities in the Grandmother hypothesis, has been emphasized here. Also, there are important convergences. Kaplan and colleagues' models, the model built by Lee, and the Grandmother hypothesis recognize human longevity as an evolved trait—with help *from* elders, not *to* them—the source of the distinctive age structure of human populations. That agreement, in itself, indicates progress toward explaining our slow life histories as the evolutionary legacy of our ancestral past.

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