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Grandmothers and the Evolution of Human Longevity

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ABSTRACT Great apes, our closest living relatives, live longer and mature later than most other mammals and modern humans are even later-maturing and potentially longer-lived. Evolutionary life-history theory seeks to explain cross-species differences in these variables and the covariation between them. That provides the foundation for a hypothesis that a novel role for grandmothers underlies the shift from an ape-like ancestral pattern to one more like our own in the first widely successful members of genus *Homo*. This hypothesis links four distinctive features of human life histories: 1) our potential longevity, 2) our late maturity, 3) our midlife menopause, and 4) our early weaning with next offspring produced before the previous infant can feed itself. I discuss the problem, then, using modern humans and chimpanzees to represent, respectively, genus *Homo* and australopithecines, I focus on two corollaries of this grandmother hypothesis: 1) that ancestral age-specific fertility declines persisted in our genus, while 2) senescence in other aspects of physiological performance slowed down. The data are scanty but they illustrate similarities in age-specific fertility decline and differences in somatic durability that are consistent with the hypothesis that increased longevity in our genus is a legacy of the "reproductive" role of ancestral grandmothers. Am. J. Hum. Biol. 15:380–400, 2003.

Based on detailed examination of human reproductive aging, Gosden (1996:281) concluded that the "reproductive system ages faster than the body as a whole and by age 45 can be said to be in the state that a woman's other organs have reached by eighty." Here I review the foundation for linking that characteristic of humans to other life-history features that distinguish us from our nearest living relatives, the chimpanzees. After briefly outlining the problem, I consider the hunting hypothesis, an influential explanation for some of those features based on benefits from large-game hunting among our ancestors. Summarizing archaeological and paleontological evidence against the hunting hypothesis, I present a simplified outline of hominin evolution that highlights the life-history shift in genus *Homo*. Then I review ideas from evolutionary life-history theory and cross-species variations they explain. Theory and data linking adult mortality risk to both maturation and aging rates provide the foundation for a grandmother hypothesis to account for the life-history shift in our genus. I reprise that hypothesis and then report comparisons of age-specific changes in fertility, mortality, and aging between humans and chimpanzees. These data, while consistent with the hypothesis, are very limited, highlighting how much more we need to know about fertility and aging in chimpanzees.

PROBLEMS OF HUMAN LIFE HISTORY

In three of the classic articles that established evolutionary life-history theory (Cole, 1954; Williams, 1957; Hamilton, 1966), long postmenopausal survival was recognized as a striking characteristic of modern humans. Williams (1957:407), noting that selection could maintain "little or no post-reproductive period in the normal life-cycle of any species" proposed the most influential hypothesis to explain the apparent exception in humans: "At some time during human evolution it may have become advantageous for a woman of forty-five or fifty to stop dividing her declining faculties between the care of extant offspring and the production of new ones." This scenario implies that modern aging rates were established in ancestral human populations before subsequent adjustment in our age at menopause. Analytical attention thus focused on the optimal timing of fertility termination in humans (e.g., Hawkes et al., 1989; Hill and Hurtado, 1991, 1996, 1999; Rogers, 1993; Peccei, 2001; Shanley and Kirkwood, 2001),

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and comparisons among the lengths of postfertile lifespans across species (e.g., Finch, 1990; Pavelka and Fedigan, 1991; vom Saal et al., 1994; Caro et al., 1995; Packer et al., 1998).

But since Williams proposed this stopping early hypothesis, theoretical and empirical reasons have been accumulating to pose questions about human postmenopausal longevity in the opposite way. Our age at menopause may be the conserved ancestral trait, while our long potential lifespans are derived (Kaplan, 1997; Judge and Carey, 2000). Cross-species variation in longevity and links between that variable and other life-history features then become matters of central interest. Maximum lifespans vary more than 50-fold among mammalian species. That variation is positively correlated with differences in maturation rates, a relationship that persists even when the body size effects are removed (Harvey and Zammuto, 1985; Sutherland et al., 1986; Harvey et al., 1989; Read and Harvey, 1989; Promislow et al., 1990). Theory to explain the cross-species regularities (Charnov, 1991, 1993, 1997; Charnov and Berrigan, 1991) combined with a hypothesis about a novel role for ancestral grandmothers (Hawkes et al., 1998) and evolutionary theories of aging (Williams, 1957; Hamilton, 1966; Kirkwood, 1977; Ricklefs, 1998) can account for a shift toward greater longevity and later maturity, while ancestral age-specific fertility termination was conserved in our lineage. This hypothesis highlights ancestral socioecological circumstances that allowed more vigorous perimenopausal females to raise the fertility of their daughters, consequently strengthening selection against senescence (the greater vulnerability to mortality with increasing age) and increasing potential longevity in our genus.

Using a demographic approach to life histories, the hypothesis relies on models in which adult mortality risk determines selection on both age at maturity and rates of senescence. Theory and evidence highlight the increase in intrinsic (senescent) components of adult mortality risk when life histories are slow (Williams, 1957; Ricklefs, 1998). This is justification for the proposition that reduced aging rates can further lessen adult mortality risk in already slow life histories. The demographic approach contrasts with others that use variation in fetal, infant, and juvenile patterns of growth and development to explain maturation rates (e.g., Gould, 1977; Leigh, 2001; Lieberman, 2002). Starting with adult mortality reveals cross-species regularities that are extremely general and this approach to human life histories links four distinctive features to each other: our long potential lifespans, late maturity, short interbirth intervals, and midlife menopause.

The most influential and most fully elaborated alternative argument to explain the evolution of late maturity and high fertility in humans, the hunting hypothesis, proposes that these features are consequences of expanding human brains. I outline it and then sketch the current paleoanthropological record of the timing and character of transitions in human evolution. The summary highlights both lack of fit with the hunting hypothesis and the prominence of a life-history shift in the evolution of our genus. Then I assemble the combination of theory and evidence that justifies a grandmother hypothesis to explain the evolution of genus *Homo* and proceed to comparisons between humans and chimpanzees that help to reveal our design for fertility and longevity.

THE HUNTING HYPOTHESIS

The hunting hypothesis was the most influential argument about human origins throughout the second half of the last century (Dart, 1953; Washburn, 1960; Washburn and DeVore, 1961; Washburn and Lancaster, 1968; Isaac, 1978; Hill, 1982; Lancaster and Lancaster, 1983, 1987; Tooby and DeVore, 1987; Kaplan et al., 2000). There are good reasons for the persistence of this idea. Cartmill (1993:191), reviewing its history, noted that "the hunting hypothesis was the first truly Darwinian explanation of human origins to be proposed." Building on Dart's proposals (e.g., Dart, 1949, 1953), it was Washburn who articulated the hunting hypothesis most effectively within anthropology (e.g., Washburn and Avis, 1958; Washburn and DeVore, 1961). In this hypothesis, features of life history and social organization that distinguish humans from other primates evolved because increased reliance on hunting large animal prey gave net advantages to larger brains. Its main elements are these:

- Drying environments in the late Tertiary constricted African forests, making

capacities to use alternative foods more advantageous among ancestral apes.
- Bipedalism was then favored because it freed hands for tool use, which increased success at hunting big animals, and this put a premium on larger brains.
- But the mechanics of bipedal locomotion limited pelvic width, so brain expansion created an "obstetrical dilemma" requiring most brain growth to be postnatal. Consequently, children with developing brains were immature longer and were more dependent, for a longer time, on maternal care.
- The care requirements interfered with maternal hunting, so mothers relied on provisioning from hunting mates. This help from fathers allowed mothers to produce more surviving offspring.
- Thus, parents formed lasting bonds and nuclear families became the fundamental units of cooperation in which a sexual division of labor served familial goals of production and reproduction.

This hypothesis was especially influential because it tied together the earliest archaeology, the fossil evidence of human ancestry, and social organization differences between humans and other primates. Washburn championed investigation of all those lines of evidence.

THE EARLY ARCHAEOLOGY

Paleolithic archaeologists used the hunting hypothesis to interpret the rich east African PlioPleistocene sites that are roughly the same age as the first fossils assigned to our genus. Isaac (1978) saw these as living areas where ancient hunters brought their prey to provision families. This view was subsequently challenged on grounds that the bone assemblages were more likely the residue of hominin scavenging from remains abandoned after most meat was consumed by the primary carnivore predators (Binford, 1981; Blumenschine, 1987). Countering this, other analysts found cut marks on bones indicating that the hominins also had access to carcasses that were still well fleshed (Bunn and Ezzo, 1993). Some archaeologists interpret the damage patterns as plausible indications of hunting (Dominguez-Roderigo, 2002). O'Connell et al. (2002a,b) note the locations of these early sites to be likely settings for carnivores to ambush prey. They review the mixed patterns of tool and carnivore tooth marks on the bones and surmise variable hominin success at competitive scavenging. Using modern analogs to estimate the likely amount of meat hominins might procure in this way, they show amounts too variable to be a major source of nutrition (O'Connell et al., 2002a,b). Since Washburn's elaboration of the hunting hypothesis stimulated detailed examination of the early archaeology (Issac, 1984), consensus that it indicates big-game hunting by the first members of our genus has collapsed.

HUMAN PALEONTOLOGY

The fossil evidence of the hominins themselves has expanded substantially in the last three decades. Evidence that bipedalism was not linked to brain expansion came first (Johanson and White, 1979). Now three main radiations of fossil hominins can be (provisionally) distinguished in the record (Klein, 1999, 2000). First was the initial divergence of our clade from that of chimpanzees, marked by a shift to bipedality, perhaps around 6 mya (e.g., Haile-Selassie, 2001; Senut et al., 2001). The brain and body sizes of those australopithecines (used in the broad sense to include *Australopithecus, Ardipithecus, Kenyanthropus, Paranthropus*, etc.) were similar to modern chimpanzees (McHenry, 1994). Their maturation patterns were more similar to modern chimpanzees than to modern humans (Bromage and Dean, 1985; Smith, 1986, 1987, 1989, 1991, 1994; Smith and Tompkins, 1995; Dean et al., 2001). On those grounds, the several taxa in this australopithecine radiation have been characterized as "bipedal apes" (Klein, 1999). Bipedalism antedated both brain expansion and the archaeological record by millions of years.

The second radiation began around two million years ago with genus *Homo*. The first widely successful members of our genus, labeled *H. ergaster* in Africa (Wood, 1992) and *H. erectus* in Asia (Rightmire, 1990) showed a marked increase in brain size. Body sizes also increased substantially, making the *relative* brain size change less dramatic (McHenry, 1992, 1994; Aiello and Wheeler, 1995; Aiello and Wells, 2002; Wood

and Collard, 1999). Collard and Wood (1999: 324) go so far as to say 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.

Homo ergaster/erectus reached its larger size by maturing later than the australopithecines (Smith BB, 1991, 1993, 1994; Dean et al., 2001; Aiello and Wells, 2002). With body size and shape similar to modern humans, maturation patterns may also have been within the modern range (Smith, 1994; Clegg and Aiello, 1999; Tardieu, 1998; Anton, 2001).

Homo ergaster/erectus had smaller teeth and jaws and a narrower thorax than australopithecines, indicating reliance on foods that require less mastication and digestion (Aiello and Wheeler, 1995). Different foraging strategies are also indicated by expansion into a wide range of new habitats. This taxon is the first hominin found outside Africa, as far east as Indonesia and as far north as 45° (Gabunia et al., 2000; Larick et al., 2001; Swisher et al., 1994; Zhu et al., 2001). Both the smaller teeth and guts and the range expansion have been attributed to increased meat eating (e.g., Shipman, 1986; Shipman and Walker, 1989; Milton, 1999; Stanford and Bunn, 2001), an inference often linked to the assumption that the PlioPleistocene sites represent big-game hunting. As noted above, archaeological challenges to that claim are numerous. Big-game hunting is not reliably indicated in the archeology until the last half million years (Stiner, 2002). Isotope analyses of a sample of specimens show no more meat eating in *H. ergaster* than among contemporaneous australopithecines (Lee-Thorp et al., 2000; Lee-Thorp, 2002). Alternatives (Wrangham et al., 1999), including the grandmother hypothesis (O'Connell et al., 1999, 2002a,b), may better explain the morphological changes.

It was with the third hominin radiation, that of archaic humans which began about 0.5 mya, that brains reached the large size of moderns (e.g., McHenry, 1994; Ruff et al., 1997). So by this reading of the evidence, the second radiation, the appearance of genus *Homo*, is associated especially with a change in life history as indexed by body size and maturation rate and a change in resource use inferred from dentition, body form, and geographical range. Bipedalism precedes this life-history shift by millions of years. Big-game hunting and the very big brains that evolve with archaic humans (including both early near moderns and their contemporaries like the Neanderthals) do not appear until more than 1.5 million years later. The features linked in the hunting hypothesis do not evolve together and the large brains that are presumed to drive the evolution of other features emerge last.

Since Washburn elaborated the hunting hypothesis, not only the archeological and fossil evidence has changed. Theory and models unavailable then have become standard tools of analysis in behavioral evolution. Conflicts of interest among individuals have been recognized as especially important (Williams, 1966a; Trivers, 1972; Hrdy, 1981, 1999; Haig, 1993). Investigations into social organization in other primates reveal persistent male–female relationships that do not depend on economic cooperation (Smuts, 1985, 1992; van Schaik, 1996; van Schaik and Janson, 2000). Studies of many living populations now challenge previous assumptions that nuclear families evolve and persist as units of common reproductive interest (Gowaty, 1996; Mesnick, 1997; Palombit, 1998; Bliege Bird, 1999; Hawkes et al., 2001).

LIFE-HISTORY THEORY

At the same time that evolutionary anthropologists were especially stimulated by Washburn's extensions of the hunting hypothesis, MacArthur and Wilson (1967; Pianka, 1970) proposed r and K selection to account for the enormous interspecific variation in rates of maturation and offspring production among living things. They hypothesized that life-history tactics giving high maximal rates of population increase (r) would be favored when ecological disruptions result in population crashes and periodic opportunities for rapid population growth. These circumstances would favor early maturity at small size, with all effort expended in producing many small offspring, and so early death. On the other

hand, in "saturated" environments, with population densities near carrying capacity (K), they hypothesized that selection would favor life histories that maximize competitive capacities: late maturity at large size and the production of a few large, well-developed offspring over long adult lives.

While the logic of r & K selection is simple and compelling, both empirical and theoretical work has shown that density-dependent mortality has different effects on developmental strategies depending on which age classes suffer the mortality (Gadgil and Bossert, 1970; Charnov and Shaffer, 1973; Stearns, 1977). The ecological associations originally postulated for r & K selection do not hold empirically. Both clusters of characters can evolve and persist in stable populations (see Stearns, 1992, for review). But the fast/slow variation that was of initial interest (MacArthur and Wilson, 1967; Pianka, 1970) and correlations between life-history variables and body size (Bonner, 1965; Blueweiss et al., 1978; Western, 1979; Harvey and Clutton-Brock, 1985) are real regularities of the empirical world.

Adult lifespans and age at maturity

Life-history features are not only correlated with body size, they also correlate with each other when the effects of body size are removed (Harvey and Read, 1988; Read and Harvey, 1989; Promislow et al., 1989; Harvey et al., 1989). During the last decade, Charnov, building on his own earlier work and that of many others (e.g., Koslowski and Wiegert, 1987; Harvey and Purvis, 1999, for review), focused on only a very few tradeoffs that can account for these patterns. Within taxonomic groups, larger-bodied species live slower lives, but some taxa are relatively slower, and others faster, at similar body sizes. The explanatory models take account of both the within-taxon correlations with body size and the between-taxon differences (Charnov, 1991, 1993, 1997, 2001; Charnov and Berrigan, 1991; Purvis and Harvey, 1995).

Charnov's models capture both similarities in the relationships among life-history features in primate and nonprimate mammals and also some distinctive features of the primate order (Charnov, 1993; Charnov and Berrigan, 1993). The "mouse lemur to gorilla" curve describes allometric variation in the life-history variables with body size that parallels the "mouse to elephant" curve. In both cases larger bodies are associated with longer adult lifetimes (the inverse of the adult mortality rate: M), later ages at maturity (α), and lower annual fecundity (b). These features are not only correlated within each group, Charnov (1991, 1993) shows that their products are "invariant" even as the values of the variables themselves change across the range of body sizes.

Primates are slower than nonprimate mammals, taking longer to mature at a given size and reproducing more slowly for size, but relationships among the life-history variables are the same for primate and nonprimate mammals. Charnov explains the relationships using a simple growth model in which productive capacity is an allometric function of size. [Charnov (2001) modifies this assumption to allow more realistic sigmoid growth, a modification that does not change the basic results discussed here.] Juveniles are assumed to use their productive capacity to grow themselves, then, at maturity, adults redirect that capacity into producing offspring. The benefit of delaying reproduction is the greater productive capacity resulting from growth to larger size. The cost of delaying maturity is the risk of dying before reproducing. If adult mortality risk is high, selection favors maturing earlier. If adult mortality risk declines, selection favors growing longer before maturing to reap the productive benefits of larger size. Whether life histories slow down or speed up depends on adult mortality risk.

Adult lifespans and rates of aging

Adult mortality risk not only provides a key to variation in age at maturity and rates of offspring production, it is central to evolutionary explanations of senescence, defined as age-specific declines in adaptive performance (Williams 1957). Williams (1957:404) used the decline of selection pressures with increasing age to explain why "*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" [original italics]. Paradoxically, senescence is faster in species with fast life histories because individuals do not live long enough to grow old. When adult mortalities

are low, more individuals live long enough to display age-specific declines in performance; selection against senescence strengthens; aging slows. But age-specific mortalities are more strongly affected by senescence the lower the adult mortalities. An increasingly larger fraction of adult mortality is due to age-specific frailty. Ricklefs (1998) demonstrates how sharply this intrinsic component of adult mortality increases with lifespan in mammals and birds.

Fitting a parameter for age-specific changes in adult mortality rates to data on 18 species of birds and 27 species of mammals, Ricklefs (1998) found that, as predicted, species with longer adult lifespans age more slowly. He used the difference between the minimum mortality rate (the rate in young adults) and rates at older ages to estimate intrinsic (senescent) mortality, and measured the fraction of adult deaths due to senescence for different lifespans. In populations with average adult life spans just under 2 years, less than 8% of adult deaths are due to senescence, i.e., all but those few deaths would occur at the observed ages even if there were no senescence and vulnerability to mortality remained constant with age. On the other hand, in populations with average adult life spans just over 15 years (the range for modern great apes), 69% of the adult deaths were due to senescence. Selection can only slow senescence to the extent that the benefits of the physiological adjustments that retard aging outweigh the cost. Very high proportions of senescent deaths in long-lived organisms indicate that these costs are considerable (Ricklefs, 1998).

Disposable somas

Life-history theory assumes that the allocation of energy and time to somatic durability and maintenance is limited by fundamental tradeoffs. Kirkwood's "disposable soma" model (Kirkwood, 1977, 1981; Kirkwood and Holliday, 1979; Kirkwood and Rose, 1991) is a version of Williams' (1957) antagonistic pleiotropy theory of aging that highlights the tradeoff between somatic effort and current reproductive effort. Selection favors disposable somas rather than immortal ones because physiological adjustments that slow senescence leave less for current reproduction. Lower reproductive rates cannot be favored without other compensating effects on lifetime fitness.

The tradeoff is demonstrated in experiments on *Drosophila*, where artificial selection can increase lifespans over only a few generations and increased longevity is accompanied by decreases in reproduction (Rose, 1991). Heritable variation that could allow selection to shift longevities quickly is widely distributed. Both pedigree analysis and selection experiments indicate substantial within-population variation in longevity in a wide array of species, including humans (Herskind et al., 1996; Finch and Tanzi, 1997; Carey and Judge, 2001). Tradeoffs between longevity and fertility appear in human datasets as well (e.g., Westendorp and Kirkwood, 1998).

Many physiological processes contribute to somatic durability, processes that operate not only in adulthood, but throughout life (Service et al., 1985). Reviewing the history of ideas, Holliday (1995:102) noted that:

> Initially, the disposable soma theory took into account accuracy in macromolecular synthesis, . . . [Then] the metabolic cost of repair of macromolecules was an obvious inclusion (Kirkwood, 1981), and later on many other types of mechanism were discussed in terms of the maintenance of the adult organism . . . . Today the disposable soma theory includes the considerable metabolic expense of all such maintenance mechanisms and the tradeoff between this expense and the investment of resources into growth to adulthood and reproduction.

By focusing attention on these mechanisms, the disposable soma model reverses the question about variation in rates of senescence from "Why do organisms get old?" to the converse: "Why do organisms live so long?" Living processes result in damage that can be reduced or repaired, but always at some cost. Sometimes increased somatic maintenance and repair results in longer-term fitness benefits that outweigh those costs, including the cost of reduced, or postponed, reproduction (Williams, 1966b; Charnov and Schaffer, 1973; Stearns, 1992). Selection can therefore favor increased longevity, with lower rates of annual fecundity, as long as the overall rate of increase in the longer-lived lineages is higher than in competing lineages. The enormous empirical

variation in lifespans (Finch, 1990, 1997) and the generally lower rates of annual fecundity in longer-lived mammals and birds (Read and Harvey, 1988; Saether, 1988; Stearns, 1992; Charnov, 1993) are consistent with these tradeoff arguments.

GRANDMOTHER HYPOTHESIS AND THE LIFE-HISTORY SHIFT IN GENUS *HOMO*

Consider the evidence of human phylogeny in light of this broad and systematic variation in mammalian life histories. If australopithecines matured at about the same age as chimpanzees, implications follow for other aspects of their life histories. Modern chimpanzees mature very late and have maximum lifespans that are very long compared to other terrestrial mammals (Eisenberg, 1981). As noted above, *H. erectus* was larger, maturing later than the australopithecines (Smith, 1991, 1994; Clegg and Aiello, 1999; Tardieu, 1998; Anton, 2001; Dean et al., 2001). From a demographic perspective, later maturity implies even lower adult mortality and longer adult life spans than that typical of chimpanzees. The link between age at maturity and adult mortality poses the following question for the evolution of genus *Homo*: How could adult mortalities, already quite low in australopithecines, have been reduced even further in our genus?

Ethnographic clues

Observations among Hadza hunter–gatherers in northern Tanzania (Woodburn, 1968; Blurton Jones et al., 1992) suggest an answer. Among these modern people, women past menopause are productive and energetic foragers (Hawkes et al., 1989), and so are children (Blurton Jones et al., 1989). But young children are not very effective at handling an important diet staple, the large root of a plant that is deeply buried and requires some strength to excavate (Hawkes et al., 1995). Women past menopause are especially active tuber diggers (Hawkes et al., 1989). In this population, mothers' foraging effort has a measurable effect on their children's nutritional welfare except when those mothers are nursing new infants. Lactating women spend less time foraging, and it is the work of postmenopausal grandmothers that differentially affects the nutritional welfare of weaned children (Hawkes et al., 1997). Grandmaternal effects on the welfare of weanlings have been found in some other contemporary and historical populations (e.g., Sear et al., 2000; Voland and Biese, 2002; Jamison et al., 2002), but not all (Hill and Hurtado, 1991, 1996, 1999). The ecological circumstances faced by Hadza foragers highlight an especially important fitness opportunity for older females that would have arisen with the past climate changes that Washburn incorporated into the hunting hypothesis.

Drying environments in the Pliocene constricted the availability of foods that young juveniles can handle. Increasing aridity and seasonality favor 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, but only to those with the strength and skill to extract and process them. Young juveniles cannot do it. To rely on these resources and succeed in these environments, mothers have to provision offspring who are still too young to extract and process the foods themselves. The mother–offspring provisioning allows the occupation of otherwise inhospitable environments. It also creates a novel fitness opportunity for older females whose own fertility is declining. If the older females help feed their just-weaned grandchildren, the mothers of those weanlings can have shorter interbirth intervals without reductions in offspring survivorship. The more vigorous elders who have no nursing infants of their own will thus raise their daughters' reproductive success (Hawkes et al., 1998; O'Connell et al., 1999).

Grandmothers and Charnov's invariants

This grandmother hypothesis combined with Charnov's model implies particular relationships among life-history variables. If Charnov's tradeoff arguments apply, and if females past childbearing are contributing to their daughters' reproductive success, then human age at maturity (α) should be adjusted to the whole adult lifespan (1/M, the inverse of the adult mortality rate), preserving the relationship between α and M that is generally characteristic of primates. On the other hand, with grandmothers' help, human interbirth intervals during the childbearing years should be shorter than expected for a grandmother-less ape with our age at maturity.

In his 1993 book, Charnov displayed the correlation between age at maturity and average adult lifespan for 15 primate subfamilies. His analysis showed the relationship to be no different from the relationship between these variables in nonprimate mammals. Charnov's (1993) graph showing this pattern in primates included a data point for hominins, which comes from the only living member of the taxon: modern humans. While both lifespan and age at maturity are much higher in modern humans than in other primates, the human point falls just as predicted by the general primate αM invariant. As expected from this model, the αM numbers for three other living great apes, orangutans, gorillas, and chimpanzees, are similar to each other and also to an αM number calculated for a sample of modern human foragers (Hawkes et al., 1998). Alvarez (2000) confirms that the relationship between α and M in humans is well within the confidence interval of the general primate pattern based on data from 16 primate species. The fact that living primates *including modern humans* illustrate this αM invariance provides justification for the working assumption that the same relationships among life-history variables, and so the same fundamental tradeoffs, applied to ancestral hominins. If so, the ages at maturity of the fossil taxa imply characteristic longevities.

In addition to the αM invariant, Charnov's model highlights an invariant relationship between age at maturity (α) and average annual fecundity (b). For mammals generally and primates in particular, annual fecundity goes down as age at maturity is delayed. Later-maturing mothers are larger and nurse their babies to larger size before weaning. Consequently, they produce offspring at a slower rate. The grandmother hypothesis proposes that specific socioecological circumstances allow more vigorous elders to help their daughters provision weanlings, allowing those daughters to wean offspring earlier than without such help (Bogin, 1999, 2001). Consequently, selection favors increased vigor when fertility is dropping because the fecundity of the child-bearers in a grandmothering lineage is higher than expected for a grandmother-less one. Comparisons among orangutans, gorillas, chimpanzees, and humans show this to be the case (Hawkes et al., 1998). Alvarez (2000) confirmed that, as predicted by this hypothesis, the human rate of offspring production is above the confidence intervals determined by the regular relationship between age at maturity (α) and annual fecundity (b) across 16 primate species.

This apparent break with Charnov's "assembly rules" for mammalian life histories is, at the same time, consistent in a deeper sense with the general tradeoffs his model highlights. Humans have a higher probability of birth per year (b) than expected in the childbearing years, with b falling to zero a few years before menopause. The argument here proposes that grandmothering results in two interdependent components of adulthood, with higher than expected fecundity in one *because* there is no direct fecundity in the other. A grandmother-less primate, modeled according to these tradeoffs to have our age at maturity, would have constant fecundity throughout adult life instead. When human fecundity is calculated as an average over all adulthood, not just the childbearing years, our αb does approach the primate invariant.

Grandmothers and aging

This grandmother hypothesis proposes that when food resources that were difficult for young juveniles to exploit became increasingly important in ancestral diets, mothers shared more of the food they found and processed with their young offspring. Help with provisioning then allowed earlier weaning and reduced birth spacing. Older females who were becoming infertile, and so not encumbered with infants themselves, could supply that help and have a large effect on their daughters' fertility. By this pathway selection would have favored increased allocation to physiological processes that buffer adults against mortality risk, slowing senescence through increases in somatic durability, maintenance, and repair (Kirkwood, 1977; Kirkwood and Holliday, 1979; Kirkwood and Rose, 1991).

This scenario assumes that australopithecine populations were in evolutionary equilibrium for the tradeoff between somatic maintenance and current reproduction. At this equilibrium, further reductions in the rates of senescence were too costly in current reproduction to be favored by selection. But then, the ecological changes that increased the importance of foods difficult for young juveniles to handle and increased the role of

maternal provisioning changed these payoffs. Females who were slightly more vigorous as their own fertility was ending could increase the fertility of their daughters. Increased somatic effort would impose a cost on current reproductive effort: more vigorous longer-lived females would allocate less to their own current reproduction. But sufficient benefit from the help of elders could compensate that cost, resulting in selection for greater longevity. Net higher fertility for the child-bearers would give a selective advantage to lineages with increased vigor at later ages. This would slow further the already slow life history of our australopithecine ancestors.

Selection for greater longevity by this pathway could not delay the age-specific termination of fertility. Grandmothers with infants of their own would be less able to help their daughters. The cost of a more durable soma (lower reproductive effort from longer-lived mothers) in lineages with longer fertile spans would then go uncompensated. Thus, selection would favor less somatic effort and life histories collapse back to the ancestral chimpanzee-like equilibrium. Longer-lived lineages would maintain their slower life histories only by maintaining the ancestral age of menopause.

HUMAN/CHIMPANZEE COMPARISONS

This scenario depends on the inference that australopithecines had life histories similar to modern chimpanzees, while *H. erectus/ergaster* had life histories more similar to modern humans. Among the comparisons between humans and chimpanzees that are pertinent to it, I consider two here. If an ancestral age-specific fertility decline persisted through australopithecines to genus *Homo*, and if chimpanzees maintain a similar ancestral pattern, then we should be similar to chimpanzees in our age-related fertility declines. On the other hand, grandmothering in genus *Homo* is hypothesized to favor increased longevity. We should be different from chimpanzees in our somatic durability and age-specific somatic performance.

Age-specific fertility decline

Measurements of the day-specific probabilities of conception controlling intercourse behavior show fertility declines in women beginning just after the mid-20s (Dunson et al., 2002). In industrial societies women in their 40s have few babies (e.g., Fretts et al., 1995), a pattern that also holds among modern foragers (Howell, 1979; Hill and Hurtado, 1996). Almost all last births are before 45. Data on chimpanzees are extremely limited by comparison. Conceptions rates per cycle in captivity are reported to be five times higher in females age 15–25 than in those age 35–48 (Graham, 1986). The ages of older adults are often not known in captivity, and known more rarely in the wild. Goodall (1986) estimated that Flo, the famous matriarch of the Gombe population, had her last birth in her early 40s. Flo's daughter Fifi, whose estimated birth is 1958, had her eighth baby in 1998. Sugiyama (1994) reports a few births to mothers estimated to be between 40 and 44 at Bossou. Based on those data, Gage (1998:209) estimates that "the end of the reproductive career . . . is similar for *Pan* and humans."

Comparing ages at menopause is more difficult, in part because menopause is harder to document even in humans. Treolar's (1981) dataset, based on longitudinal records, remains one of the best. Subjects initially recruited as students at the University of Minnesota recorded each menstruation and their daughters enrolled at menarche. In this well-nourished population, normal menopause ranged widely from early 40s to late 50s, with mean and mode for women not using hormone interventions about 51. Samples from other countries suggest there may be variation among different human populations. Some show earlier peaks and nutrition and other health practices may affect the timing of menopause, although measurement problems plague these comparisons (Gosden, 1985; Wood, 1994; Leidy, 1994).

In contrast to the large human datasets, there are few data on menopause in chimpanzees — in part because chimpanzees rarely live to menopausal ages. However, endocrine profiles published on two long captive subjects show them to be perimenopausal at ages 48 and 50, respectively (Gould et al., 1981), near the central tendencies of the human distribution. In addition to the two chimpanzees (*Pan troglodydes*), this study also included one bonobo (*Pan paniscus*). Estimated to be over 40 but younger than the other two, she was no longer cycling. Her hormone profiles and ovarian histology

confirmed that she had passed menopause. This provides another data point consistent with the premise that genus *Pan* and genus *Homo* have similar menopausal ages.

These limited data on age-specific fertility declines and menopause are consistent with the proposition that the ancestral age at menopause has been maintained in our lineage. However, some biochemical constraint, not an absence of directional selection, could also account for these similarities. Mammalian reproductive physiology makes this seem initially plausible. Although women, like most other mammals, reach menopause when a fixed stock of oocytes is depleted (Gosden, 1985; Richardson et al., 1987; vom Saal et al., 1994; Wood 1994), the similarity between human and chimpanzee menopause could result from an upper age limit on oocyte viability of about 50 years. But two kinds of evidence, interspecific variation in the size and depletion rates of initial oocyte stocks, and the longer fertile spans in other species suggest that selection can delay the age at menopause when there is net benefit for continued fertility at later ages.

First, the variation between and within species in both the size of the initial stock of oocytes and the rate at which these are lost is substantial (Finch, 1990; Finch and Kirkwood, 2000). Humans begin with an average of several million oocytes attained in the first few months of fetal life, reduced to less than a million before birth, with most of the remaining lost before puberty (Block, 1953). Number of ovulations is not a likely predictor of age at menopause, since less than 0.01% of the initial stock is ever actually ovulated. Across the mammals, the store of oocytes remaining at maturity varies with lifespan (Gosden and Telfer, 1987). Those stocks are larger in species with longer potential lives and the rate of loss during adulthood may be slower in longer-lived species (Gosden and Telfer, 1987). The cross-species variation suggests that selection can and does adjust both of these variables.

The second line of evidence consistent with the view that menopause at 50 is not set by physiological constraints on oocyte viability comes from other long-lived mammals. Both Asian (Sukumar et al., 1997) and African elephants (Moss, 2001) continue to have successful births into their seventh decade, 20 years longer than women do. In some cetaceans fertility continues to even later ages. Antarctic fin whales are found pregnant into their 80s (Mizroch, 1981). With recent evidence that maximum lifespans reach well over 100 years in bowhead whales (George et al., 1999), data on late age fertility in this species will be of special interest.

Overall then, the available data on ages at last birth and menopause in chimpanzees show age-specific fertility declines in that species not substantially different from our own. Mammalian fertility, however, can extend to much older ages than it does in humans. This evidence is consistent with the argument that ancestral age-specific fertility declines have been maintained in our lineage, perhaps conserved by stabilizing selection.

Distinctively human design for longevity

While ages at terminal fertility and menopause appear to be similar in humans and chimpanzees, we differ from them and from other primates in our general vigor at these ages and our high probability of decades of menopausal survival (Pavelka and Fedigan, 1991). Goodall (1986:81) classified Gombe chimpanzees as moving from middle to old age at 33. Among the few in her study population who lived that long, she noted frailty, emaciation, slow movement, and difficulty in climbing. "Whether or not the lives of these individuals were complicated by disease during their last months, there can be little doubt that old age itself was primarily responsible for their deaths" (1986:104).

Wide recognition of human postmenopausal longevity has, however, not led to consensus that perimenopausal vigor and postcycling survival are distinctive characteristics of our evolutionary design. None dispute that we have "the maximum lifespan of the terrestrial animals" (Smith and Tompkins, 1995:258), but the reasons for this continue to be debated. I first review demographic data from various human populations to show that life expectancy at birth is a misleading index of longevity and that changes in mortality with age are regular enough to suggest a "characteristic pattern" in our species. Then, noting the possibility that a distinctively human pattern of adult mortality could be an artifact of our unusual economic interdependence and cooperation, I turn to physiological measures that index cross-species differences in the rate of senescence.

Demography. Some investigators argue that, rather than a species characteristic, human potential longevity is a recent novelty (e.g., Olshansky et al., 1998). In most of the US, western Europe, and Japan, newborns can now expect to live about 80 years, while historical demography and population profiles in traditional settings show life expectancies less than four decades. Life expectancies at birth, however, are strongly affected by rates of infant and juvenile mortality (e.g., Bailey, 1987; Smith DWE, 1993; Lee, 1997). There is no credible evidence of any change in maximum human lifespan over historical time (Austad, 1997).

French historical demography, for example, illustrates this point. Life expectancy was only 39 in 1850, compared to double that in 1985 (Keyfitz and Fleiger, 1968, 1990). The difference between these two time periods is not, however, a change in maximum life spans but a change in the rate of mortality at young ages. Since life expectancies are cohort averages, they reflect not only the long lives of those who die old but also the very short lives of those who die as infants and children. Even in mid-19th century France, with life expectancies less than 40, those who survived to adulthood had the prospect of a long life ahead. Of the girls who lived to adulthood, 72% then lived past 45, the age of terminal fertility, and the average number of years remaining for anyone who reached that age was more than two additional decades (Keyfitz and Fleiger, 1968, 1990).

Nineteenth-century France had an agrarian economy. If those vital rates depend on the use of domesticates, they would only have appeared within the last 10,000 years with the advent of agriculture. Demographic data from modern people who do not depend on domesticated food, however, show similar patterns of age-specific mortality. Three of the best-studied cases of modern hunter–gatherers, the !Kung of southern Africa (Howell, 1979), the Ache of South America (Hill and Hurtado, 1996), and the east African Hadza (Blurton Jones et al., 1992, 2002), represent populations in different environments with distinct recent genetic histories. All show patterns of age-specific survival very like those reported for 19th-century France. Life expectancies at birth are less than four decades in all these cases due to infant and juvenile mortality. On reaching adulthood most women live past their mid-40s, the age of terminal fertility, and those who do have an *average* of more than 20 years of life still ahead.

Using Hamilton's (1966) method for computing the force of mortality, the instantaneous death rate for each age class, underscores the similarity in patterns of senescent mortality in human populations with diverse economies and recent genetic histories. Figure 1 plots demographic data from France in 1985, 1950, 1900, and 1850. Although life expectancies double over this time period, the age-specific mortality risks among adults are similar. Figure 2 shows the force of mortality calculated for the three hunter–gatherer populations, the !Kung (Howell, 1979), the Ache (Hill and Hurtado, 1996), and the Hadza (Blurton Jones et al., 2002).

The unusual postmenopausal longevity of humans is especially clear when we are compared with other primates (Pavelka and Fedigan, 1991, 1999). In macaque and baboon populations (Pavleka and Fedigan, 1999; Packer et al., 1998), reaching the age of terminal fertility is the luck of only a very few individuals. Less than 5% of those reaching adulthood live past that. Chimpanzees mature later and have longer adult lifespans than do macaques and baboons. In captivity a few more females live past childbearing age (Caro et al., 1995). But among chimpanzees in the wild (Hill et al., 2001) only about 5% of the adult females are past the likely age of terminal fertility — so few they are all but invisible statistically. This contrasts with about one-third of adult females beyond childbearing age in human foraging populations (Hawkes et al., 2003).

Figure 3 combines the hunter–gatherer cases, mid-19th century France, and the Taiwanese peasants of 1900, the dataset Hamilton (1966) used himself. While the differences among these modern human populations are not negligible, the similarities are striking. Similarities in the shape of changes in mortality with increasing age, even when the particular causes of death vary (Preston et al., 1972; Finch et al., 1990; Gavrilov and Gavrilov, 1991; Hill and Hurtado, 1996) seem a "species signature" of aging that is especially clear when the human populations are contrasted with chimpanzees (Fig. 3; chimpanzee data from Hill et al., 2001). Senescent mortality (the fraction of deaths above baseline — the lowest age-specific mortality) increases much earlier and more steeply for chimpanzees.

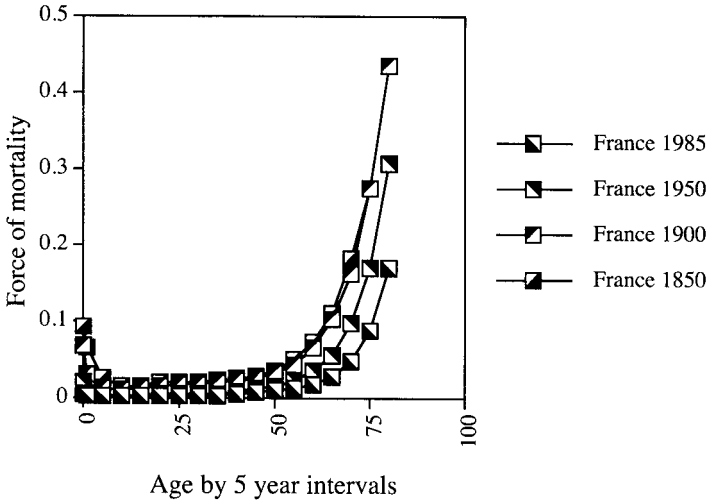

Fig. 1. Instantaneous age-specific mortality risk (Hamilton, 1966) calculated from historical demography (Keyfitz and Fleiger, 1968, 1990). Probability of death during a 5-year age class indicated on the y axis, age on the x axis.

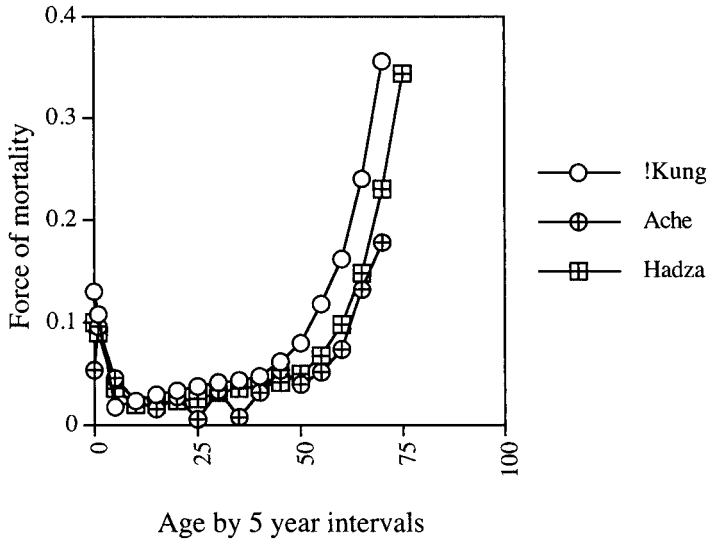

Fig. 2. Instantaneous age-specific mortality risk (Hamilton, 1966) calculated for three contemporary hunter–gatherer populations. Probability of death during a 5-year age class indicated on the y axis, age on the x axis. !Kung from Howell (1979); Ache from Hill and Hurtado (1996); Hadza data Blurton Jones et al. (2002).

The inference from historical and ethnographic demography that substantial longevity is a characteristic feature of human populations has been challenged by age distributions of archaeological skeletal assemblages (Weiss, 1973; Trinkaus, 1986, 1995; Austad, 1997). Remains from individuals estimated to be over 60 at death are rare, supporting skepticism about the generality of the demographic patterns found among living populations. But new sources of error are introduced in constructing population profiles from archaeological assemblages. Two especially important sources of bias are revealed by cases in which historical records provide independent evidence of

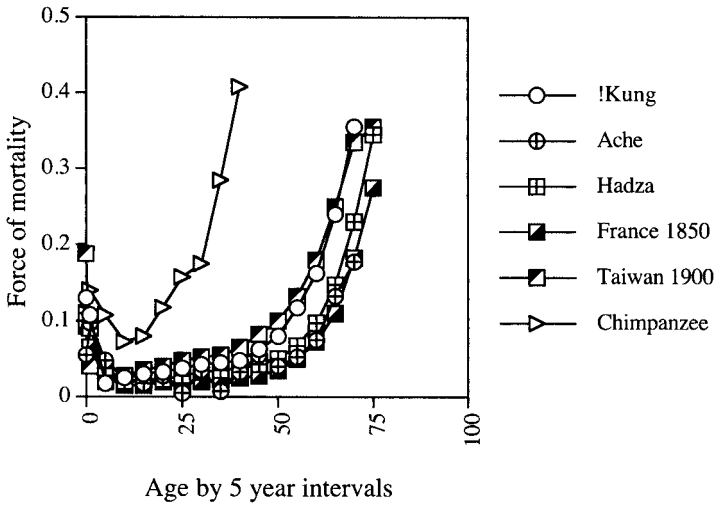

Fig. 3. Instantaneous age-specific mortality risk (Hamilton, 1966) for two preindustrial agricultural and three hunter–gatherer human populations and for wild chimpanzees. Probability of death during a 5-year age class indicated on the y axis, age on the x axis. French historical demograph from Keyfitz and Fleiger (1968); Taiwan peasant farmer data used by Hamilton (1966); !Kung from Howell (1979); Ache from Hill and Hurtado (1996); Hadza from Blurton Jones et al. (2002); chimpanzee data (smoothed) from Hill et al. (2001).

the age of individuals interred. Not only are the bones of the old and the young disproportionately unlikely to sustain long preservation, the ages of adults are also systematically underestimated (Walker et al., 1988; Paine, 1997). Standard aging techniques applied to samples of known ages illustrate the pervasiveness of the problem (Bocquet-Appel and Masset, 1982; Key et al., 1994). Analysts have repeatedly shown that the cemetery profiles do not represent sustainable living populations (e.g., Howell, 1982; Bermudez de Castro and Nicolas, 1997).

Biases in the other direction, novel features of the modern world that might extend longevities in ethnographically known foraging populations, have also been explored. Living people are now everywhere affected to some degree by global networks of interaction (Wolf, 1982; Schrire, 1994; Blurton Jones et al., 1996). Blurton Jones et al., (2002) examined the possible effects that interaction with agricultural neighbors as well as some access to Western medicines might have on Hadza demography. Even the most generous estimates of neighbor interactions, regional medical services, and the ethnographers' own possible effects make only negligible differences in the population parameters initially reported (Blurton Jones et al., 2002).

These patterns are consistent with the argument that modern humans have a distinctive design for longevity. But human dependence on culture might provide an alternative explanation for potentially longer human lives. When Lee (1968) estimated that 10% of the people in his !Kung study population were over 60, he used the estimate to underline his observation that hunter–gatherers meet their needs more easily than often expected. He emphasized not only the time they spent in food procurement, but also their economic cooperation and food sharing. The longevity of humans might result from resource pooling and community support that reduces fatalities otherwise likely to follow illness, injury, or foraging failures. If greater human longevity results from distinctively human patterns of cooperation and interdependence, the demographic differences between foraging people and chimpanzees might greatly overestimate underlying patterns of age-specific frailty. A cultural safety net, rather than a fundamental shift in rates of senescence, could be the reason for our longer lives.

Physiological aging. If the difference between chimpanzee and human longevities reflects real differences in aging rates, and mechanisms of maintenance and repair are similar, differences in allocation to these mechanisms might be directly measurable. Evidence is mounting that "modulators of the rate of aging" are conserved over large evolutionary distances (Partridge and Gems, 2002:165). I summarize ideas and evidence about three physiological systems related to maintenance and repair: antioxidants, DNA repair, and age-related neuropathologies. All illustrate differences between humans and chimpanzees in somatic durability.

The accumulation of damage to cells as a result of metabolism is an important contributor to aging (Harman, 1957; Beckman and Ames, 1998; Finkel and Holbrook, 2000). This may explain how dietary restriction slows aging in a wide array of organisms (Holliday, 1989; Sohol and Weinruch, 1996; Yu, 1996; Lin et al., 2000; Hulbert and Else, 2000; Partridge and Gems, 2002). Cell lines from different mammalian species vary in their resistance to a variety of oxidative and nonoxidative stresses and the variation is correlated with species longevities (Kapahi et al., 1999). Both rates of free radical production (Barja et al., 1994) and the production of antioxidant mechanisms (Ku et al., 1994) also correlate with species longevity. This work provides a foundation for expecting design for greater longevity in humans than chimpanzees to be reflected in differences in the management of oxidative damage at the cellular level. Some data are available on antioxidant production among primates showing that levels of the circulating antioxidants plasma urate (Ames et al., 1981) and super oxide dismutase (Tolmasoff et al., 1989) are substantially higher in humans than in chimpanzees.

Damage to DNA accumulates with time, so variation in cell responses to this damage should be correlated with aging rates (Tice and Setlow, 1985). Investigators have used UV-irradiation to damage cell lines and then measured subsequent DNA repair. Cell lines from different mammalian species show differing amounts of repair depending on species longevities. In human cell lines DNA repair is much greater than in cell lines from other primates (Hart and Setlow, 1974). That sample did not include chimpanzees but it did include gorillas. To the extent that gorillas can serve as a proxy for chimpanzees (who are themselves used here as a proxy for australopithecines), this is more evidence that humans are designed for greater longevity than were ancestral hominins.

Alzheimer-like neuropathologies increase with age in most primates just as they do in humans (Finch and Sapolsky, 1999). Unlike other primates, however, where the proliferation of senile plaques can begin before the age of menopause, these neuropathologies are generally delayed well past menopause in humans. When the same two chimpanzees found to be perimenopausal at ages 48 and 50 (Gould et al., 1981) died (at 56 and 59) they showed no sign of cognitive impairment (Gearing et al., 1994). However, autopsy revealed modest accumulation of brain amyloid (Gearing et al., 1994) which may represent presymptomatic or early symptomatic Alzheimer's disease (Morris et al., 1996). This would be consistent with earlier aging in chimpanzees than humans. Finch and Sapolsky (1999) suggest that the delay in this kind of age-related decline in humans is associated with our unusually long postmenopausal survival linked to the benefits of grandmothering. They also hypothesize that the several genetic variants of apolipoprotein E that are found in humans but not in other primates may be implicated in the protection against these age-related neuropathologies in our species. One of the apo E variants most like the single isoform found in other primates is associated with an array of disease susceptibilities that are consistent with this hypothesis (Finch and Sapolsky, 1999).

CONCLUSIONS

Like Washburn's elaboration of the hunting hypothesis, a grandmother hypothesis assumes that changing ecological circumstances favored a shift in foraging strategies that gave rise to our genus, with assistance for juveniles from kin other than mothers driving the shift. But this grandmother hypothesis uses a demographic perspective on life-history variation that gives our low adult mortality rates and remarkable longevity special importance.

The claim is that members of our genus began living longer *not* because they were kept alive when frail — although this does now happen, increasingly in many current populations. Instead, a change in selection pressures that favored increased

allocation to somatic durability resulted in slower aging in our genus. Greater longevity in turn favored later ages of maturity. Australopithecines with chimpanzee-like life histories had maintained a different equilibrium in which reproductive and other physiological systems senesced in tandem. When ecological changes resulted in more mother–child food sharing, the cost of variants that reduced senescent mortality in ancestral populations were outweighed by the benefits due to more vigorous grandmothers' help. This resulted in the evolution of genus *Homo*.

Instead of linking late maturity to the developmental requirements of large-brained juveniles, the grandmother hypothesis derives our late maturity from our design for longevity. Later maturity also means that older juveniles, gaining benefits from delaying their own reproduction, could provide another source of help for younger siblings. Fathers may have helped sometimes. But ancient men, like other males (Hawkes et al., 1995), especially other primates (Smuts, 1987, Smuts and Gubernick, 1992; Van Schaik and Paul, 1996), and like modern men (Hurtado and Hill, 1992; Bliege Bird, 1999; Blurton Jones et al., 2000; Hawkes et al., 2001), could not have escaped the pressures of male competition that often make mating effort take priority over paternal investment (Williams, 1966a; Trivers, 1972; Hawkes and Bliege Bird, 2002; O'Connell et al., 2002b). The grandmother hypothesis, unlike the hunting hypothesis, does not assume that cooperation in child-rearing either depends on or necessarily gives rise to nuclear families.

Hrdy (1999, 2001) has characterized humans as cooperative breeders. Noting that nonmaternal helpers, or allomothers, play an especially important role in rearing human children, she links a greater human ambivalence about maternal investment in particular offspring to our greater need for help. "A human mother's commitment to her infant should be linked to how much social support she herself can expect" (Hrdy, 2001:58). Most other primates postpone the next offspring as long as the previous one is still dependent. The grandmother hypothesis explains how we came to do otherwise, deriving the evolution of our need for help from the postmenopausal longevity that supplied more candidates for allomothering. Vigorous peri- and postmenopausal women, overlapping infants and toddlers, as well as prereproductive adolescents, are part of a systematically interrelated suite of life-history features. Together these features make social circumstances especially important, and so make maternal sensitivity to them especially advantageous.

A growing proportion of adults in senior age ranks in many contemporary human populations is clearly a recent novelty (Vaupel et al., 1998; Oeppen and Vaupel 2002). This large fraction of elders now surviving in spite of senescent frailty presents economic, medical, political, and social challenges that can hardly be overestimated (Tuljapurkar et al., 2000). But those facts should not obscure the strength of the evidence that adult lives substantially longer than those of the other apes are "normal" for our species, yet our age-specific fertility decline and menopause appears to differ little from chimpanzees. The models and evidence reviewed here employ the widely used assumption that chimpanzees can serve as a life-history analog to human ancestors. If australopithecines had ape-like life histories, the effects of senescence on adult mortalities could have been very large.

Demographic comparisons between foraging chimpanzees and people (Fig. 3) show not only the earlier, steeper climb in senescent mortality in chimpanzees, the baseline "initial mortality" rate is also higher for chimpanzees (Hill et al., 2001). While this lowest rate is conventionally used to estimate extrinsic mortality risk (e.g., Ricklefs, 1998), disposable soma ideas suggest that it would also include an intrinsic component in long-lived species. If mechanisms of maintenance and repair develop early in ontogeny, then more effective mechanisms for buffering mortality risks in adulthood would likely reduce the initial mortality rate as well (Finch et al., 1990).

Washburn argued effectively for the use of comparisons with other primates to discover what happened in human evolution. When he advanced the hunting hypothesis the frequency and importance of hunting among chimpanzees, now so well documented (e.g., Goodall, 1986; Boesch and Boesch, 1989; Stanford, 1996, 1999; Mitani et al., 2002), was unknown. In addition to the essential value of those and other behavioral comparisons between these species (e.g., de Waal 2001), genetic comparisons now promise the possibility of detailed evidence about human evolution (Varki, 2000; Gagneux and Varki,

2001; Enard et al., 2002). More work focused especially on life-history comparisons is also in order, including comparisons in age-related physiology (Erwin et al., 2001) and in molecular and cellular processes associated with somatic development, maintenance, and repair.

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