# 3

# Life History Theory and Human Evolution

## *A Chronicle of Ideas and Findings*

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**SUMMARY**

*Fertility ends at similar ages in women and female chimpanzees, but humans usually live longer and mature later. We also differ from our closest living relatives in weaning infants before they can feed themselves. The comparisons pose questions about when and why the distinctively human life history traits evolved in our lineage. Here I outline the basic framework of the field of life history evolution and, against that background, chronicle past inquiries into each of these distinctively human traits. The chronicle covers discovery and description, guided sometimes by hypotheses about underlying developmental mechanisms and sometimes by hypotheses about adaptive effects. Following the review, I discuss the continuing importance of distinguishing between questions about mechanisms and adaptive effects in light of accumulating fossil evidence and progress in genomics. I conclude with a brief reference to the most influential adaptive hypothesis to date, the Hunting hypothesis, and some of the accumulating empirical challenges to it, setting the stage for current debates addressed in subsequent chapters.*

Human life histories differ from those of our nearest living relatives in several striking ways (Smith and Tompkins 1995; Bogin and Smith 1996; Robson, van Schaik, and Hawkes, chapter 2, this volume). We can live twice as long as chimpanzees can, having the greatest potential longevity of the terrestrial vertebrates (Carey and Judge 2000). Yet, fertility declines to essentially zero at about the age of 45 in both women and female chimpanzees (Gage 1998; Hawkes 2003; Nishida et al. 2003). Other aspects of human physiology age more slowly than our ovarian systems (Gosden 1985; Finch 1990; Hill and Hurtado 1991; Hughes et al. 2001; Blurton Jones and Marlowe 2002; Walker and Hill 2003), so, unlike other primates, we reach menopause before our geriatric years (Pavelka and Fedigan 1991). We mature very slowly. Average age at first birth is about six years later for humans than it is for chimpanzees (Robson, van Schaik, and Hawkes, chapter 2, this volume). But we wean infants earlier than chimpanzees do and have shorter birth spacing (Galdikas and Wood 1990; Robson, van Schaik, and Hawkes, chapter 2, this volume). Unlike other primate juveniles, our children continue to depend on feeding assistance after they are weaned and their mothers are nursing new babies (Lancaster and Lancaster 1983, 1987; Bogin 1999a).

These features of human life histories not only describe important aspects of our individual lives but also determine the age structure of human families, communities, and populations. Age composition of social groups determines many of the problems and possibilities of both conflict and cooperation. Accumulating evidence that our life history features are both distinctive and typical of human experience prompts questions about when and why they evolved. When and why did ancestral populations begin to have characteristically human age structures?

Modern humans are the only living representatives of a clade that has included many other species. Those previous members of our lineage and their distribution in time and space are known only through the fossil and archaeological record. Without that record, we would have no clues to the existence and distinctive character of either australopithecines or any species in the genus *Homo* except our own. The fragmentary evidence of hominin fossils and the archaeology associated with past populations are the key lines of evidence about how they differed from one another, when and how much they differed from living species, and when and why human life histories evolved.

 

A focus on life histories highlights questions about longevity and aging, age at maturity, rates of offspring production, and the population age structures that these imply. Life history evolution is a field that seeks to explain variation in probabilities of survival and reproduction across the lifespans of living things, highlighting interrelationships among these timing and rate variables. Questions about physiological (and now, increasingly, molecular) mechanisms that underlie the rate and timing of growth, development, reproduction, and aging occupy many researchers. Life history evolution focuses less on mechanisms and more on their fitness-related effects, that is, whether they are likely to result in relatively more descendants in future populations. These effects can explain why natural selection adjusts the timing and rate variables (and therefore the mechanisms underlying them) differently in different species. The goal of the field is to discover the fitness costs and benefits, the trade-offs that explain the diversity of life histories across the living world. Applied to our lineage, the theory, the models, and other conceptual tools help link multiple lines of evidence about our evolutionary heritage.

Following this introduction, I describe the field of life history evolution by characterizing a few of its key assumptions and models (for a review of the field, see Stearns 1992). In preparation for subsequent discussion, I then summarize r and K selection, some critiques, and Charnov's alternative scheme of dimensionless invariants.

Next, I turn to the history of ideas and findings about the appearance of four distinctive features of human life history: our late maturation, our potentially long lifespans, our slow aging, with fertility ending in women while other physiological systems maintain a substantial fraction of peak performance, and our children's continued dependence on feeding assistance after they are weaned.

After summarizing work on the evolution of each of those four features, I explicitly distinguish different kinds of explanations. The discussion follows the review of work because a different mix of explanatory frameworks has influenced inquiry into each of the four features of human life histories. Questions about causal mechanisms and about adaptive function have both been important. Distinguishing between them is too. Another distinction, between homologous features (those shared with immediate common ancestors) and homoplasies (similarities that newly appear in descendants), has also become

 

especially important with the rise of cladistics and the explosive development of molecular techniques. The distinction between homologies and homoplasies is of primary import for investigators seeking to establish phylogenetic relationships among both modern and ancient taxa. Expansions of the hominin fossil record have exposed a great deal of independent variation in measurable traits, complicating phylogenetic assignments. More generally, developments in genomics reveal similar genetic pathways for phenotypic functions employed by species that are phylogenetically very distant from one another. These findings make mechanism questions seem potentially tractable and especially tantalizing. Only adaptive hypotheses, however, seek to explain why particular life history features evolve instead of others, why human life histories have taken their distinctive shape.

One adaptive hypothesis has provided especially influential guidance for research exploring changes in the human lineage. I turn to it in a brief concluding section. As Cartmill (1993:191) pointed out, "the hunting hypothesis was the first truly Darwinian explanation of human origins to be proposed." S. L. Washburn's (1960; Washburn and DeVore 1961; Washburn and Moore 1974) especially influential version of this hypothesis linked work in paleoecology, paleontology, Paleolithic archaeology, comparative primatology, and hunter-gatherer ethnography. Now the empirical record in each of the fields on which Washburn relied is much richer and more complex. The evidence no longer shows the temporal relationships among the appearances of human characteristics, which once seemed clear. Nevertheless, discovering what actually happened in the evolution of our lineage still involves pursuing Washburn's general research strategy. The same lines of inquiry he championed, as well as genomics, provide the evidence to build, correct, and revise hypotheses about what happened in the past. Explicit use of models that link life history features to one another is an additional tool to help us extract more from the hard evidence on the evolution of our lineage.

## LIFE HISTORY EVOLUTION

Although the foundation goes back to Darwin (1859) and further to Euler (1760), the field-defining publications in life history evolution are mostly mid-twentieth century, including Fisher (1930), Cole

 

(1954), Williams (1957, 1966b), Hamilton (1966), MacArthur and Wilson (1967), Lack (1968), Tinkle (1969), and Gadgil and Bossert (1970). However, the pace of work accelerated so much in the late eighties and nineties that Stearns, in his 1992 textbook, could write that "analysis of the evolution of fitness components is a new field, life history evolution" (Stearns 1992:10).

**The Demographic Foundation**

Partridge and Harvey's (1988:1449) succinct definition of life histories as "the probabilities of survival and the rates of reproduction at each age in the life-span" highlights the demographic framework of the field and its ties with the conceptual tools of population genetics.

Stearns (1992:10) explained the framework this way:

> Life history evolution makes the simplifying claim that the phenotype consists of demographic traits—birth, age and size at maturity, number and size of offspring, growth and reproductive investment, length of life, death—connected by constraining relationships, tradeoffs…including those between current reproduction and survival, current reproduction and future reproduction, number, size, and sex of offspring.

The power of this simplification comes from models of population growth in age-structured populations (Cole 1954; Hamilton 1966; Gadgil and Bossert 1970; Charlesworth 1994).

The basic equation of both demography and life history evolution was discovered by Euler in the eighteenth century and rediscovered by Lotka in the twentieth (Euler 1760; Lotka 1922; Keyfitz 1977). Population growth rate (r) is determined by age-specific mortality and fertility rates. It depends on the probability of survival ($l_x$) and the average number of offspring produced ($m_x$) by those at each age (x). These variables are necessarily interdependent because the size of each newborn cohort depends on the number of females in the offspring-bearing ages and their fertility rates. Those females come from cohorts of past newborns. Thus, the number of females in the fertile ages depends on the number and fertility of females in the past, as well as the mortality rates of their offspring. When fertility and mortality rates

 

are sustained for a few generations, they result in a population with a stable age distribution. This means that all the age classes then grow (or decline) at a constant exponential rate (r) and the fraction of the total population in each age class remains unchanged.

"Although most natural populations are rarely in stable age distribution, moderate deviations from stable age distributions do not often change qualitative predictions. The Euler-Lotka equation captures robust features of demography" (Stearns 1992:25). A mutation that alters the rate of fertility or mortality at any age can affect the population growth rate. Comparing the growth rate of the mutant with the growth rate of the background population therefore indicates the lifetime fitness effect of the mutation and whether it would spread or decline against the common type.

An additional simplifying assumption is sometimes used. Because stable populations grow (or decline) exponentially, growth rates must average near zero most of the time. Otherwise, populations either disappear or overrun the planet. In stationary (that is, nongrowing) populations ($r = 0$), each adult female, on average, exactly replaces herself. The number of surviving daughters produced by a female over her lifetime ($R_0$) is one. When $R_0 = 1$, the relationships among certain life history variables are necessarily fixed; therefore, assuming that populations are nongrowing can simplify modeling and analyses (Charnov 1991, 1993, 1997). Stationary populations also have standing age distributions that mirror the mortality schedule, a useful way to underline the fundamental link between life histories and the age structure of populations.

Life history analyses explore the costs and benefits of shifts in mortality and fertility by measuring the magnitude of their lifetime effects. Because time and energy are limited, more of one thing generally means less of something else (Maynard Smith 1978; Seger and Stubblefield 1996). Any increase in fertility or decrease in mortality at one age likely entails changes in those variables at other ages. Changes have different consequences for lifetime fitness, depending on which ages they affect (Cole 1954; Williams 1957; Hamilton 1966; Charlesworth 1994). These consequences and the life histories of ancestral populations determine the range of possible life histories in immediate descendants.

 

**r and K Selection**

Over the past several decades, r and K selection has been the most widely used theory of life history variation. The labels were introduced by MacArthur and Wilson (1967; Pianka 1970) to capture Dobzhansky's (1950) suggestion that in environments subject to extreme variation, populations will likely "crash." Mortality would then be independent of density and largely independent of individual competitive abilities. Under those conditions, selection would favor features that maximize the intrinsic rate of population growth (r). In relatively constant environments, populations would saturate carrying capacity, K (the conventional symbol for this variable in density-dependent models of population growth). Then mortality would be density-dependent and would differentially affect individuals, depending on their efficiency in acquiring resources. Under those conditions, selection would favor increased competitive ability. Pianka (1970) enumerated the contrasting characteristics expected for each kind of selection, with K-selected species investing more in fewer offspring that develop more slowly and delay reproduction to gain greater competitive ability and larger body size, living long lives and reproducing repeatedly.

The r and K scheme is intuitively appealing. As Stearns (1992:307) wrote, it "was suggestive and influential but incorrect." Problems with it include the following three (for example, see Promislow and Harvey 1990, 1991; Roff 1992; Stearns 1992; Charlesworth 1994). First, the associations postulated in the theory are often not found in the world. Stearns (1977:168) tested the dichotomy on published data on a wide array of taxa: "In about half the studies…the organisms fit the accepted scheme…; in the other half they did not." Promislow and Harvey (1991:124) noted that "K selection should, by definition, give rise to increased carrying capacity.… But larger individuals using a greater amount of resources might actually reduce carrying capacity." In fact, maximum densities of different species are negatively correlated with body mass, larger-bodied species living at lower maximum densities (Damuth 1981, 1987).

In addition to empirical failures, a second important problem is the coherence of the assumptions. As Stearns (1977:206) put it:

> Unlike r, K cannot be realistically expressed as a function of

 

> life history traits.…Thus r and K cannot be reduced to units of common currency. If they do trade off, so that higher r's imply lower K's, the mechanisms by which that tradeoff is accomplished are not demographic, but are bound up in physiology and social behavior, and as such could be expected to change from taxon to taxon.

It is especially important that the r and K distinction ignores differences in mortality rates with age, because changes in mortality rates have different consequences for life history evolution, depending on which ages are affected. A population in which growth is held in check by equal mortality increases on all age classes can be comparable to a population that is allowed to grow unchecked. Selection has the same effects on life history traits under both conditions (Charlesworth 1994). Equal mortality increases across all age classes are the same as no mortality changes at all.

A third problem is that the characteristics expected with either r or K selection can be generated and maintained in stationary (nongrowing) populations by models in which mortality varies with age (Kozlowski and Weiner 1997; Harvey and Purvis 1999). The circumstances postulated in the r and K scheme to explain each set of features are therefore not required for the evolution or maintenance of either one.

Nevertheless, "fast-slow" variation in life cycles and associations between timing variables and adult body size are empirical regularities in the living world (Bonner 1965; Western 1979; Clutton-Brock and Harvey 1983; McMahon and Bonner 1983; Peters 1983; Calder 1984). Scaling patterns are pervasive (Brown and West 2000). Roff (1992:45) criticized Pianka for using body lengths of vertebrates and insects to demonstrate r versus K selection, because "to compare vertebrates and insects is to compare apples and oranges." Still, within taxonomic groups, life history variation does generally fall along a fast-slow continuum that is associated with variation in body size. As Promislow and Harvey (1990:418) emphasized, however, "it is important to distinguish between the empirically observed fast-slow continuum in mammal life histories and the theoretically derived r-K continuum. There is no good evidence that increased competitive ability among mammals results in higher K."

 

**Charnov's Symmetry Approach**

An alternative scheme for broad regularities in life history variation has been developed by E. L. Charnov (1993, 2002), who has shown that mammals, birds, and fish can be distinguished by characteristic relationships among life history traits. The relationships among the traits reflect distinctive symmetries in each taxonomic group by remaining approximately constant—invariant—when the traits themselves change in value. For example, the ratio between species' average adult lifespan and average age at maturity is about 1.4 for mammals, 2.3 for birds, and 0.5 for fish (Charnov and Berrigan 1990a; Charnov 1993). To explain these taxonomically distinctive patterns, Charnov (2002:753) has constructed explanatory models in which "the optimal life history adjusts some life-history variables…in the face of tradeoffs with others.…[T]he dimensionless approach to life histories looks for *invariants* in the *outward life history*…and in the *tradeoffs* that generate the set of optimal life histories" (italics original).

Unlike r and K selection, Charnov's approach emphasizes both the differences among and the similarities within taxonomic groups. Three "dimensionless numbers, each a benefit-cost ratio summarizing reproductive timing, allocation and demography, are invariants and thus are useful to classify life histories"(Charnov 2002:749). He uses them to represent the distribution of life histories in three dimensions, a "life history cube." Mammals, birds, and fish occupy distinct positions in the cube, defined by their characteristic values on the life history ratios that delineate the edges: (1) the ratio of offspring size to maternal size, (2) the ratio of relative reproductive effort to adult mortality rate, and (3) the ratio of adult lifespan to age at maturity. The values for each variable are averages calculated over age-structured populations. Charnov (2002:757) wrote:

> This classification scheme for life histories differs from those such as "*r*- and *K*-selection"…in that these other schemes invariably use axes with dimensional magnitudes such as time or mass. Elephants and squirrels are at opposite poles in these schemes, and the suggestion is made that natural selection operates in fundamentally different ways when we contrast them (opportunistic versus equilibrium,

 

> for example)….But when we remove absolute magnitude for time and mass, squirrels and elephants look a lot alike, and look different from fish or altricial birds. Selection may well operate similarly on squirrel and elephant life histories in the sense that…the trade-offs have the same dimensionless features. My working hypothesis…is that the trade-off features are the same *within* (say) altricial birds, mammals or indeterminate growers like fish, with major differences *between* these groups.

To the extent that this holds empirically, the distinctive characteristics of mammalian (or perhaps, more narrowly, primate) trade-offs should help students of human evolution hypothesize the ranges of possible life histories and population age structures of our ancestors and the extinct taxa in our lineage. A Grandmother hypothesis for the evolution of human life histories (Hawkes, O'Connell, and Blurton Jones 1997, 2003; Hawkes et al. 1998; Blurton Jones, Hawkes, and O'Connell 1999; O'Connell, Hawkes, and Blurton Jones 1999; Alvarez 2000; O'Connell et al. 2002; see Hawkes, chapter 4, this volume, for more detailed discussion) explicitly builds on Charnov's mammalian invariants.

## THE EVOLUTION OF FOUR DISTINCTIVE FEATURES IN THE HUMAN LINEAGE

The empirical record of life history variation among living populations, the theory to explain that variation, and the fossil record of our own lineage have all expanded in the past few decades. For a much longer time, questions about the evolution of particular human life history features have preoccupied the curious. I largely restrict attention to work that addresses questions about the appearance of these features in the evolution of our lineage, and I discuss the four features in turn. Investigators often addressed these features two at a time, so the sections overlap. But each highlights different problems and different aspects of the record.

Models of life history evolution generally assume that mortality schedules determine optimal ages of first birth and rates of offspring production. On those grounds, I should discuss our low adult mor-

 

tality (our long lifespans and slow aging) first, with postmenopausal longevity following, then slow maturation, and finally weaning age. I begin instead with slow maturation because S. J. Gould's (1977) *Ontogeny and Phylogeny* highlighted that feature especially, sketching a broad history of ideas about evolutionary change and raising issues that I touch on again in subsequent sections of this chapter. Gould's book continues to influence research on the evolution of human growth and development.

**Slow Maturation**

Gould is especially well known for his interest in the role of developmental mechanisms in shaping the diversity of life and for his critiques of "the adaptationist program" (for example, Gould and Lewontin 1979). *Ontogeny and Phylogeny* (1977), however, emphasized the fundamental adaptive importance of life history variation. Here are Gould's (1977:289–290) own words:

> I regard the rise of theoretical population ecology as one of the most significant events in evolutionary theory during the past twenty years. For…it has proved that the components of life history strategies—timing of reproduction, fecundity, and longevity, for example—are adaptations in themselves, not merely the consequences of evolving structure and function.…In short, theoretical population ecology has given us a new set of parameters for assessing adaptation.

Gould (1977:2) wrote that he began his book about "the ancient subject of parallels between ontogeny and phylogeny" to rescue an important topic that was neglected at the time because of associations with erroneous ideas about evolution: "Properly restructured, it stands as a central theme in evolutionary biology because it illuminates two issues of great contemporary importance: the evolution of ecological strategies and the biology of regulation" (1977:2).

Gould (1977:2) characterized *Ontogeny and Phylogeny* as "primarily a long argument for the evolutionary importance of *heterochrony*—changes in the relative timing of appearance and rate of development for characters already present in ancestors." From the start of the book (here quoting Gould 1977:8), however, he emphasized that although

 

"classical arguments [for the importance of heterochrony] are based upon the macroevolutionary significance of morphology," he had a different kind of evolutionary importance in mind: "I focus upon the immediate significance of acceleration and retardation in the evolution of life-history strategies for ecological adaptation. In this context, the timing of maturation assumes special importance" (1977:8).

Although morphological development is very much the subject of the book, the life history implications of morphology are repeatedly given priority of importance, for example, "the timing of maturation (rather than the morphology obtained by speeding up or slowing down)" (1977:289). In his treatment of human evolution, Gould (1977:399) reiterates that in discussing "the evolutionary significance of heterochrony, I have been trying to de-emphasize the traditional arguments of morphology while asserting the importance of life history strategies."

The r and K scheme was in wide use in the seventies and is a major theme of the book. Gould (1977:399–400) proposed that it had great promise for explaining macroevolutionary patterns:

> I have linked accelerated development to r-selective regimes and identified retarded development as a common trait of K strategists.…I have also tried to link K selection to what we generally regard as "progressive" in evolution, while suggesting that r selection generally serves as a break upon such evolutionary change. I regard human evolution as a strong confirmation of these views.
>
> To begin with, we belong to a class of animals in which K selection dominates (Pianka 1970).…We belong to an order of mammals distinguished by their propensity for repeated single births, intense parental care, long life spans, late maturation, and a high degree of socialization—a point for point agreement with Pianka's listing of traits common to K strategists (1970).
>
> Human evolution has emphasized one feature of this common primate heritage—delayed development, particularly as expressed in late maturation and extended childhood. This retardation has reacted synergistically with other hall-

 

> marks of hominization—with intelligence (by enlarging the brain through prolongation of fetal growth tendencies and by providing a longer period of childhood learning) and with socialization (by cementing family units through increased parental care of slowly developing offspring).

Gould (1977:9) argued that "neoteny has been a (probably the) major determinant of human evolution." In documenting both the history of the idea and the empirical evidence for it, he surmised:

> The notion of human neoteny has it roots in two obvious facts: the striking resemblances between juvenile pongids and adult humans and the obliteration of this similarity during pongid ontogeny by strong negative allometry of the brain and positive allometry of the jaws.…[T]hese phenomena…had been recognized as soon as juvenile pongids had reached the zoos and museums of Europe. (Gould 1977:353)

In the 1920s, L. Bolk (for example, 1926) developed his fetalization theory of human evolution; the distinctive features of human body form are seen as fetal conditions in other primates but remain permanent in humans. Gould (1977:356) justified extended attention to the case Bolk made: "His insight has been ridiculed in the light of modern doctrine and dismissed in toto because he linked valid and important data to evolutionary views now rejected.…[But] the data that he presented can survive the collapse of his explanatory structure."

While the work of A. H. Schultz is under no such danger of dismissal, Gould's caveat about the value of the data, in spite of an unsustainable explanatory structure, applies to him as well. Schultz had a very orthogenetic view of primate evolution, but those errors in no way reduce the value of many of his insights, based as they are on his meticulous attention to measurements and patterns of variation both within and among populations. Gould uses Schultz's work to support his arguments about delayed maturation in humans.

Noting our common ancestry with the anthropoid apes, Schultz (1950:428) had this to say:

> Man became distinguished chiefly in connection with his

 

> three outstanding specializations; the early and undoubtedly rapid acquisition of the erect posture, the later, gradual and ultimately great increase in relative brain-size, and the comparatively very recent prolongation of his main periods of life.
>
> With the gradual accumulation of large series of monkeys and apes it has become possible to compare man with other primates on the basis of statistically adequate observations and at all stages of growth. Only in this way can it be decided whether a particular character is really distinctive in all cases within a representative, normal range of variations, or differs merely in regard to the frequency of occurrence, thus being of lesser significance. With the realization that any evolutionary change, apparent in the adult, is the result of some primary alteration in the processes of growth and development, it seems highly desirable to discover the condition in which human growth differs from the growth of non-human primates.

Schultz (1956:890) underlines the importance of variation in age at maturity across the primates:

> In regard to all parts of post-natal life one can recognize a clear trend toward prolongation, beginning in monkeys, as compared to lemurs, more pronounced in gibbons, still more in all three great apes and by far the most marked in man. For instance, general growth, at least in length, is completed in only 3 years in prosimians, in 7 years in Old World monkeys such as macaques, in not over 9 years in gibbons, in 11 years in the large man-like apes, and in as much as 20 years in recent man. Of special biological significance is the fact that with the advance on the evolutionary scale of primates, puberty and the beginning of fertility become more and more chronologically retarded and thereby the interval between succeeding generations becomes steadily lengthened.

Schultz's view that these differences in the time to skeletal maturation represent a trend of progressive evolution is not consistent with modern

 

evolutionary theory, but the differences he points to are real empirical phenomena. As the final sentence of the passage shows, he recognized that the demographic implications of age at maturity are especially important.

Schultz might have agreed with Gould's endorsement of Bolk's central point. Gould ends his chapter on human evolution by reasserting Bolk's (1926:470) question and answer: "What is the essential in man as an organism? The obvious answer is: the slow progress of his life's course." But Schultz's work also showed that slower life histories did not mean slower growth and development in all corresponding body parts. "With his fetalization theory, Bolk (1926) had called attention to ontogenetic retardation as a phylogenetic process. It can be shown today that accelerations in development have also played a role in human evolution" (Schultz 1956:888).

Well aware of this, Gould still claimed that neoteny has been the major determinant of human evolution, a claim that has been actively debated with many demonstrations of non-neotenous processes in human growth and development (for example, Shea 1989; McKinney and McNamara 1991; Godfrey and Sutherland 1996). Gould's 1977 book might have stimulated more attention to life history, but particular kinds of morphological heterochrony have been the focus of attention among students of growth and development (for example, Parker, Langer, and McKinney 2000; Minugh-Purvis and McNamara 2002; Thompson, Krovitz, and Nelson 2003).

Much of what has happened in studies of heterochrony is quite difficult for nonspecialists because of the emphasis on distinguishing different kinds of morphological heterochrony. Shea (2002:79), appraising the proliferation of recent work, commented:

> Certainly, one area where much attention has been focused involves the recognition and definition of the myriad types and categories of morphological heterochrony.… [While] debates over appropriate definitions and classifications are certainly a necessary and productive component of heterochronic research…they often seem, to insiders and outsiders alike, as exercises that dwell excessively on ever-growing mounds of tongue-twisting jargon of uncertain biological relevance.

 

Shea (1981, 1983a) demonstrated that no single heterochronic shift could account for the differences in either growth trajectories or adult form among the African apes (Leigh and Shea 1996). Subsequent work has underlined the generality of that finding. Leigh and Park (1998:348), investigating "the evolution of human growth prolongation," compared velocity curves of growth across many species of primates on an array of dimensions and found that

> relative size, velocity relative to size-for-age, and estimates of the pace of ontogeny (timing variables) show appreciable amounts of variation independent of adult size. Variation that is unrelated to adult size is typical during early periods of primate ontogeny, and may reflect a high degree of adaptive variation. Low phylogenetically adjusted correlations for some of these variables suggest that attributes of early growth periods and adult body size are often uncoupled, and evolve independently.

Such independence has repeatedly confronted analysts looking for correlated adjustments in morphological variation. Investigators exploring the mismatch between phenotypic and molecular similarities in papionin primates find substantial variation in growth patterns across the tribe (Collard and Wood 2001). Collard and O'Higgins (2001) and Leigh, Shah, and Buchanan (2003) measured crania from ontogenetic series of various genera to characterize the growth patterns that result in adult faces. Both studies not only improve understanding of the ontogeny and evolution of papionin faces but also reveal diversity in the patterns of growth and development. Leigh, Shah, and Buchanan (2003:307) concluded that "ontogenetic allometric data may not be informative regarding papionin taxonomy."

Schultz had pointed out that even though prolonged life periods distinguish people from other primates, this does not mean that all aspects of human growth and development are slower. In his 1960 review of "age changes in primates and their modification in man," Schultz (1960:19) referred to a large study of skeletal changes with age in young American males, which found maturation changes to share a common, orderly process. He remarked:

> Closely corresponding to this intraspecific constancy in gen-

 

> eral sequence and variability in timing of the age changes of man we find intergenerically among primates few major deviations from one common pattern of development, but many marked modifications in the relative timing during the maturation of epiphyses, sutures, dentition, hair, etc. (Schultz 1960:29)

Gould (1977) appreciated this variability. Recognizing that not only neoteny but also multiple heterochronic processes are implicated in human evolution, he wrote:

> No Darwinian supporter of retardation as a major element in human evolution can deny that many distinctive features are not peadomorphic; the concept of mosaic evolution practically requires such a belief.…The evolutionary direction of each feature is controlled by natural selection; the capacity for independent variation of characteristics is very great. (Gould 1977:364–365)

Yet, at the same time, Gould pinned substantial hope on finding that developmental mechanisms limit that independence. His claim that "features of an organism are bound (often quite loosely) in covariant sets, and these sets are often dissociable as blocks" (Gould 1982:341–342) is cited by Shea (1983b:522), who explicitly hoped that "this 'shuffling' of the developmental trajectories of various body regions may provide new adaptive morphological configurations with minimal genetic changes."

*Ontogeny and Phylogeny* was published just after M.-C. King and A. C. Wilson's (1975:107) summary showing that "the genetic distance between humans and the chimpanzee is probably too small to account for their substantial organismal differences." King and Wilson (1975:107) hypothesized that "regulatory mutations account for the major biological differences between humans and chimpanzees." Citing their work, Gould (1977:9) wrote:

> Humans and chimpanzees are almost identical in structural genes, yet differ markedly in form and behavior. This paradox can be resolved by invoking a small genetic difference with profound effects—alterations in the regulatory system that slow down the general rate of development in humans.

 

> Heterochronic changes are regulatory changes; they require only an alteration in the timing of features already present.

The emphasis is on mechanisms. While Gould appreciated that demographic processes shape life histories and he recognized the capacity of selection to modify individual morphological features independently, he expected developmental mechanisms to explain human differences. The mechanisms that give rise to a variant and the net fitness benefits that spread and maintain it are equally important domains of inquiry. But they demand different kinds of research. Life history evolution generally seeks to explain species differences in age at maturity through the fitness effects of shifts in that trait under prevailing demographic constraints.

**Long Lifespans and Slow Aging**

Gould (1977) cited G. A. Sacher (1959; Sacher and Staffeldt 1974), who made especially important contributions to the evolution of longevity. A footnote in *Ontogeny and Phylogeny* (1977) mentioned the associations Sacher found between brain size and maturation time across the mammals as corroborating evidence for Gould's neoteny argument. Gould (1977:371n) concluded that "the neotenic hypothesis applies whether brain enlargement precedes more general retardation or vice versa." The associations between brain size and maturation time that Sacher discovered became a component of his inquiry into the evolution of longevity. Sacher (1975:417) argued that human longevity "is distinctive and important enough to merit being listed, along with symbolic and tool making behavior, as a distinguishing feature of the human species."

Finding a strong relationship between brain size and maximum lifespan across mammals, Sacher (1975:426) favored two mutually compatible hypotheses "about the causal processes responsible for the relation." One is that the brain "participates in the stabilization of the life processes of the organism.…In other words, the brain is postulated to be an ORGAN OF LONGEVITY" (1975:426, emphasis original). The other is that "the evolution of a larger brain imposes on the species an added metabolic and developmental burden, and a consequent

 

decreased reproductive rate that can only be compensated by means of an extension of the reproductive span, and hence of lifespan" (1975:426). He considered the second hypothesis to be confirmed by his finding that two brain size variables, neonatal brain weight and the ratio of neonatal to adult brain weight, are correlated with two life history variables, gestation time and litter size across ninety-one mammal species, including nineteen primates (Sacher 1975).

Sacher had earlier concluded (Sacher and Staffeldt 1974) that a common growth law for mammalian brains is the rate-limiting process for all other aspects of somatic growth. Therefore, brain size "provides an objective basis for the estimation of relative maturation times of related species that differ in brain size" (Sacher 1975:427). On those grounds, Sacher used cranial capacity to estimate both lifespan and maturation times in fossil hominin taxa. From the data available at the time, he estimated that australopithecines were later maturing and longer-lived than modern chimpanzees, that Neanderthals were similar to modern humans, and that *Homo erectus* was intermediate between australopiths and modern people.

Sacher's hypothesis that brains controlled both maturation and longevity was partly based on his (1959) finding that although brain weight is correlated with body weight across the mammals and body weight is correlated with lifespan, the correlation between lifespan and brain weight was significantly stronger. In response to Sacher, others showed that spleen, adrenal, and kidney mass are as well correlated with lifespan as is brain mass (Calder 1976; Economos 1980a; Prothero and Jürgens 1987). One recognized reason why organ weights show stronger correlations with life history variables than do body weights is that organ masses vary less among adults over time, making them more stable measures of animal size.

> The reason for the better prediction of life span from brain mass is that brain mass has less variability than body mass. The body mass of any individual or species can vary widely, depending upon its energy balance situation.... [W]ithin species, or when the mammals are treated together, the coefficient of variation for brain mass is less than half that for body mass. (Lindstedt and Calder 1981:12)

 

There is a substantial literature on brain-size/body-size measures and the relationships between them (for example, Prothero and Jürgens 1987; Pagel and Harvey 1989; Harvey and Krebs 1990; Barton 1999). Sacher (1959) devised an "index of cephalization" to measure brain size relative to body size, and much subsequent work has focused on the evolution of brains that are relatively large for body size, especially in primates (R. Martin 1990) and in the human lineage (for example, Jerison 1973; Aiello and Wheeler 1995; Allman 1999). Increases in this relative measure are correlated with increased lifespan in primates, but not in other mammalian orders (Austad and Fischer 1992; but see van Schaik et al., chapter 5, this volume).

Sacher's ideas about the role of brains in determining the pace of life histories do not include the usual assumption that the function of delayed maturity is to allow more time to learn. He cited Dobzhansky (1962) for the widespread claim "that the maturation times of hominids get longer because they have more to learn" (Sacher 1975: 429). "Hominids do indeed have more to learn," Sacher (1975:429) agreed, "but the essence of hominization is that they are able to learn more rapidly." Still, he favored the hypothesis that age at maturity depended on brain size: "A more reasonable assumption…is that the learning process speeds up to fit into the maturation period defined by an optimum pattern of mammalian brain growth" (1975:429).

Sacher's hypotheses about the causal mechanisms linking brains to life history variables have not fared well (see, for example, Allman 1999; Deaner, Barton, and van Schaik 2003; and Robson, van Schaik, and Hawkes, chapter 2, this volume, on brain growth patterns in humans and chimpanzees), but his discovery that lifespan is one of the life history features generally predictable from organism size remains extremely important. Calder (1984:4) summarized the importance of size this way:

> Suppose we encounter a new beast that we wish to understand.…If we know only its "weight" we can predict (give or take 25% or so) a wide variety of its specifications and requirements: home range, heart and metabolic rates, life span—each from an empirical allometric equation based on body size.

 

Lifespan, usually (but not always) meaning observed maximum lifespan, has come to be a widely used measure of longevity (D. W. E. Smith 1993). But starting with Sacher's use of observed maximum lifespan in the 1959 Ciba symposium, it has drawn objections. G. C. Williams (1999:405) framed his long-standing complaint this way: "It is clearly true that different species have different demographies.…This does not mean that any maximum observed age can be identified as a species characteristic." Williams (1999:405) worried that a specified maximum lifespan suggests erroneously that "death is a programmed event in life history," an idea inconsistent with evolutionary theories of aging. There are, nevertheless, ways that maximum lifespan is very useful in comparisons among populations and across species. The population average is the best single summary of lifetime survival, so life expectancy at birth or mean lifespan may seem a better single parameter than observed maximum lifespan to characterize a population's mortality experience. But mean lifespan does not distinguish differences in mortality with age. Consequently, it obscures both important cross-species differences and important intraspecies similarities.

Life expectancy is the probable number of additional years of life remaining for a cohort. Given a life table, which records the rates of death at each age, it can be calculated from any age. For example, life expectancy at the beginning of adulthood (at about 19 or 20 years of age for women [Robson, van Schaik, and Hawkes, chapter 2, this volume]) is the expectation of life for those who have reached adulthood, the average adult lifespan—a very useful parameter. When used without an age qualification, however, life expectancy refers to the expectation of life from birth. Because it is an average for an entire cohort, life expectancy at birth includes the lives of all who die as infants and juveniles. These short lives have large effects on the estimate.

Sacher (1959) focused attention on the important difference between life expectancy at birth and how long *adults* are likely to live. He said that he used "maximum documented longevity" in cross-species comparisons not only because complete life tables—necessary to calculate life expectancy—were available for so few species but also because life expectancy is not a stable parameter. "This is clearly seen in the life-tables of human populations in different countries or in the same country in different historical periods. Instances can be found in

 

which life expectations vary by more than a factor of two, but even in these extreme cases the lifespans do not differ by as much as 20 percent" (Sacher 1959:115).

Williams also seconded the common concern that maximum observed age at death might be so highly sensitive to sample size that it could not be a stable index of adult mortality rates. Sacher (1959, 1975) showed that this concern was misplaced. The relevant probability estimates for the behavior of a variable like maximum recorded lifespan come from the statistics of extremes. When adult mortality follows a Gompertz function—increasing exponentially with age (as fish, bird, and mammal mortality schedules do)—the exponential term always dominates at advanced ages. Consequently, "the characteristic oldest age increases as a double logarithmic function of the cohort size" (Sacher 1959:115). "In consequence the maximum expected survival increases very slowly" with sample size (Sacher 1975:419). "What this means in practice, as I have now confirmed for a large number of species, is that as the number of maximum survival times from different zoos increases, the maximum of this set of values increases very slowly and becomes virtually stationary for practical purposes" (1975:419).

Sacher's attention to maximum lifespan as an especially useful variable played a significant role in developments in life history theory. R. J. H. Beverton and S. J. Holt participated in the 1959 Ciba conference where Sacher reported his methods and findings. Studying the effects of harvest rates on fish life histories, Beverton and Holt (1959; Beverton 1963) required an estimate of adult mortality rates. They found data available, even approximately, for very few populations of interest. Citing Sacher (1959) for pioneering the technique of using maximum lifespan as a stable measure of longevity, Beverton (1963) showed that the "characteristic maximum age," the oldest individual observed, provided a useful index of the adult mortality rate in a population. Beverton both evaluated and exploited Sacher's argument about the statistics of extremes, and Charnov subsequently used Beverton's work as a foundation for his symmetry approach. Following Beverton, Charnov (1993) found that maximum lifespan provided a useful index of average adult lifespan, the inverse of adult mortality rates.

Adult mortality is, in turn, linked with aging. It was G. C. Williams

 

(1957) who first used life history theory to lay out basic expectations about the evolution of senescence, defined as declines in fitness-related performance with age. Williams (1957) showed how the theory of natural selection can explain why organisms get old and why members of different species age at different rates. His model of "antagonistic pleiotropy" is an evolutionary explanation for aging that is built on the decline in force of selection with age. Because some mortality risk is inevitable—even without senescence—cohorts necessarily become smaller at each successive age. Consequently, selection acts more strongly on mutations that affect phenotypic performance at younger adult ages. When the same genes have effects at different adult ages, positive effects earlier in life are favored at the expense of consequent negative effects on performance at older ages. One expectation about rates of senescence that comes from evolutionary life history theory is that selection against senescence should be stronger—and therefore rates of aging slower—in species with low rates of adult mortality. Another is that selection against senescence should be stronger when fitness-increasing opportunities rise with adult age. W. D. Hamilton (1966) provided the classic formal demonstration of demographic constraints on senescence, using data on a human population to do so.

T. B. L. Kirkwood's (1977, 1981; Kirkwood and Rose 1991) "disposable soma" model has been an especially productive elaboration on these arguments because it focuses on the demographic effects of trade-offs between repair of somatic damage that slows senescence, on one hand, and investments in current reproduction, on the other. The processes of life result in inevitable damage to cells (for example, Beckman and Ames 1998). That damage can be lessened or repaired, but allocation to those processes leaves less for growth and reproduction. Given such a trade-off, selection can never favor perfect repair. Variants that invest too much in somatic maintenance have lower lifetime fitness and are outcompeted by alternatives that put less into somatic maintenance and more into producing descendants. Reviewing the history of ideas, R. 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.

Kirkwood's model speaks to Williams' question, why do organisms age? and shows why disposable somas result from natural selection. It also speaks readily to the questions, why does aging ever slow? how can increased longevity evolve? If the lifetime fitness benefits for a marginally increased investment in somatic repair outweigh the benefits for the same investment in reproduction at younger ages, selection favors more somatic maintenance. If adult mortality rates go down, the chances of staying alive longer to benefit from a more durable soma increase, and selection favors increased somatic effort. As Williams (1957:404) noted, "*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). Subsequent work (for example, Ricklefs 1998) has confirmed that empirical correlations between adult mortality rates and aging rates across samples of mammals and birds are consistent with the theory (see Hawkes, chapter 4, this volume, for further discussion).

Long average adult lifespans characterize living human populations (Robson, van Schaik, and Hawkes, chapter 2, this volume). For past populations, the ages at death represented in skeletal assemblages would seem the obvious source of information about mortality rates. Researchers investigating aging have often relied on these data to estimate past mortality rates (Williams 1957; Austad 1997). Increasingly, though, paleodemographers have come to appreciate why the archaeological assemblages are not a straightforward reflection of population mortality rates (see Konigsberg and Herrmann, chapter 9, this volume). "[A] number of natural and cultural filters conspire to produce archaeological skeletal samples that cannot be considered as random samples of all members of a population who died within a certain period" (Konigsberg and Frankenberg 1994:92).

 

Among these filters, the following three are especially important for estimating rates of death at different ages from archaeological samples. First, taphonomic processes differentially affect bones by sex and age (P. L. Walker 1995). The surprising magnitude of possible preservation bias was demonstrated by Walker, Johnson, and Lambert (1988) on a cemetery in California where mission records showed that 53 percent of the adults interred in the cemetery were older than 45 years of age but only 7 percent of the adult skeletons recovered were older than 45. As the investigators cautioned, other things being equal, the magnitude of the age-related bias should be "roughly proportional to the length of time a group of burials has been in the ground" (Walker, Johnson, and Lambert 1988:188), a stark warning for Paleolithic samples.

The second problem of bias in the age-at-death distribution of skeletal assemblages is also serious. Remains of individuals of different ages and sexes may be deposited in different places. Among modern people, for example, juveniles and adults may be interred in cemeteries, but infants may not. Most Neanderthal remains have been discovered in cave deposits, and as E. Trinkaus (1995:138) noted, older individuals may be underrepresented if "the elderly were dying in or adjacent to shelters less frequently than younger members of the population."

Of remains that actually are recovered, age-at-death misestimation is a third substantial problem. J.-P. Bocquet-Appel and C. Masset's "Farewell to Paleodemography" (1982) was prompted by comparisons between the mortality schedules of Europeans constructed by historical demographers working with texts and those constructed by paleodemographers working with skeletal material often from related populations in similar time ranges. Bocquet-Appel and Masset showed the mismatch in estimated mortality schedules and found that the distinctive age structures produced by paleodemographers resulted from systematic biases due to the characteristics of the reference samples used in aging adult skeletons. They concluded that "early mortality of adults, overmortality of women, lack of old people in those populations, whether prehistoric or medieval: all these hackneyed notions were born from the misinterpretation of data. As they are in no way vindicated, we must get rid of them" (Bocquet-Appel and Masset 1982:329).

Assessing responses to these difficulties, L. W. Konigsberg and

 

S. R. Frankenberg (1994:93) concluded that "unfortunately there are no 'magic bullets' that can be uniformly applied to remove the biases caused by biological and cultural filters." Nevertheless, they pronounced paleoanthropology "not quite dead" because emerging methods promised much improved age-at-death estimates. Increasingly sophisticated mathematical tools extract more accurate, if less precise, information about the distribution of ages in modern human skeletal assemblages (Hoppa and Vaupel 2002a; Konigsberg and Herrmann, chapter 9, this volume). Analysts now recognize that they must *start* with a model of "how the chance of death varies with age" (Hoppa and Vaupel 2002b:3). This echoes a point N. Howell (1976a:25) made about the necessary reliance of paleodemography on the "uniformitarian assumption" that "the human animal has not basically changed in its direct biological response to the environment in processes of ovulation, spermatogenesis, length of pregnancy, degree of helplessness of the young and rates of maturation and senility over time."

If this holds across modern humans, what of other members of our evolutionary lineage? Konigsberg and Frankenberg (1994:92–93) assumed that "the evolutionary details that modified a basic pongid life history into a hominid one remain obscure, but aspects of recent demographic history are assailable. Study of the last 10,000 years or so is an important part of anthropological discourse." They went on to say that "with relatively new appreciation of the problems of age estimation, sample bias, and the complexity of relationships among fertility, mortality, population growth and life table analysis, we see paleodemography as just beginning to embark on what should be a truly productive phase of research" (Konigsberg and Frankenberg 1994:104).

Yet, some of the complexity they note, long recognized by demographers, still remains to be appreciated by students of human evolution. Of particular importance here, the erroneous inference that life expectancy (at birth) is an index of adult lifespans leads to mistaken assumptions about age structures in past human populations. The error was committed by S. L. Washburn (1981:11) decades ago: "In the last 100 years, the expectation of length of life for human beings has increased dramatically.… The result is a situation that is entirely new from the point of view of evolution—a very large number of human beings living to ages far beyond those that were normal for the species."

 

As Sacher had already observed, though, life expectancy is not a good index of longevity (and see D. W. E. Smith 1993). It is strongly affected by infant and juvenile mortality rates. Recently, J. Oeppen and R. W. Vaupel (2002) demonstrated the remarkably steady increase in the global record for national life expectancy, essentially linear since 1840. Emphasizing the importance of this persistent recent trend, they also noted that "before 1950, most of the gain in life expectancy was due to large reductions in death rates at younger ages" (Oeppen and Vaupel 2002:1029). In those populations with much lower life expectancies than the current record holders, people who did not die in childhood had long average adult lifespans, with good chances of living to a ripe old age (see Paine and Boldsen, chapter 10, this volume).

Demographers long ago demonstrated that variation in life expectancies in human populations have surprisingly small effects on adult age structure. Fertility levels have large effects (for example, Coale 1956; Coale and Demeny 1966 [second edition, 1983]). The Coale and Demeny model life tables for stable (human) populations show what initially seems counterintuitive. Holding fertility constant and tripling life expectancy from twenty to sixty years, the fraction of adults (those older than 15) who are older than 45 differs within only five percentage points. Also—perhaps initially paradoxically—the proportion of elders is largest when life expectancy is lowest (Hawkes 2004b; Hawkes and Blurton Jones 2005). Careful ethnographic demography of modern hunter-gatherers repeatedly finds life expectancies shorter than four decades and also finds the characteristic and distinctive feature of human age structures: about a third of the adults are older than 45 (Howell 1979; Hill and Hurtado 1996; Blurton Jones, Hawkes, and O'Connell 2002).

Both models and data show that it is incorrect to infer that people could not have had long lifespans in prehistory, because "life expectancies over forty years were never reported for any population prior to the late nineteenth and twentieth centuries" (Crews and Gerber 2003: 20). It is misleading to reason that "if typical life expectancy among our recent hominid predecessors was only three or four decades…less than half of the total lifespan of a typical individual would have been spent as an adult" (Krovitz, Nelson, and Thompson 2003:1).

The difficulties in estimating adult mortality rates from age-at-

 

death estimates of past populations are increasingly well understood. Investigators continue to work at reducing biases in age estimation (for example, Aykroyd et al. 1999; Miles 2001; Hoppa and Vaupel 2002a; Konigsberg and Herrmann, chapter 9, this volume), and perhaps the taphonomic biases can be estimated. But neither of these correctives removes the "cultural filter" (Konigsberg and Frankenberg 1994) that makes skeletal assemblages a biased sample of deaths. As a consequence, body size continues to be a favored index, even by paleodemographers (Konigsberg and Frankenberg 1994), for estimating variation in lifespan across hominins. The cross-species associations between size and maximum lifespan recognized by Sacher have been confirmed in greater detail in expanded and improved data sets (Harvey and Clutton-Brock 1985; Finch 1990; Allman, McLaughlin, and Hakeem 1993; Hammer and Foley 1996; Barton 1999; Judge and Carey 2000; Deaner, Barton, and van Schaik 2003), and maximum lifespan is a useful index of average adult lifespans and adult mortality rates (Beverton 1963; Charnov 1993).

Size, however, is a variable with problems of its own, as noted above in regard to brains and bodies. Additional difficulties arise in estimating it from fossil specimens (for example, Kappelman 1996; R. Smith 1996), but there are some strong patterns. McHenry (1991, 1992) demonstrated that australopiths, even those with very robust crania, were substantially smaller in body size than early members of genus *Homo* (Wood and Collard 1999b). The change from a chimpanzee-size australopith ancestor to the first widely successful members of our genus, in which both brain size and maternal body size approximately doubled, signals a shift in life history (Smith and Tompkins 1995; O'Connell, Hawkes, and Blurton Jones 1999; Aiello and Key 2002; Aiello and Wells 2002; Hawkes, O'Connell, and Blurton Jones 2003).

Within genus *Homo*, brain size and body size do not always change together (Hawkes, O'Connell, and Blurton Jones 2003; Skinner and Wood, chapter 11, this volume). Ruff, Trinkaus, and Holliday (1997: 173–174) estimated body mass and cranial capacities for genus *Homo* across the Pleistocene and concluded that an apparent increase in body size from Early to Late Pleistocene "is largely an artifact of two confounding variables: sex and geography.… There is a bias toward males in our Late Pleistocene sample…[and] the apparent sudden

 

increase in average body mass during the latter Middle Pleistocene is largely a result of the inclusion of higher-latitude specimens."

As to relative brain size, they found that

> early to early Middle Pleistocene (1,800–600 kyr BP) *Homo* was about one third less encephalized than recent humans, and there was no increase in encephalization quotient (EQ) throughout this time period. By the early Late Pleistocene (150–100 kyr BP), EQ had increased to values within about 10% of those of recent humans. (Ruff, Trinkaus, and Holliday 1997:175)

Finally, they show "that a decrease in average absolute brain size over the past 35,000 years within *H. sapiens* was paralleled by a corresponding decrease in average body size, supporting earlier suggestions of a general correlated size reduction in the human skeleton since the early Upper Paleolithic" (Ruff, Trinkaus, and Holliday 1997:175).

How big is a modern human? To the extent that body size can be estimated for a taxon and this can index longevity, the sample and estimates provided by Ruff and colleagues suggest no large differences between the early members of our genus (as defined by Wood and Collard 1999b), archaics, and Upper Paleolithic moderns. Brains, however, are larger and encephalization greater in Middle Paleolithic humans. If it is brain size that predicts longevity, then (as Sacher concluded) Neanderthals and moderns must have similar lifespans, longer than those of earlier members of the genus, with those, in turn, longer than those of australopiths.

**Postmenopausal Longevity**

When lifespans are longer—adult mortalities lower—selection against senescence is stronger, and aging is slower. However, the ovaries of living humans do not age any more slowly than those of living chimpanzees (Hawkes 2003; Robson, van Schaik, and Hawkes, chapter 2, this volume). We establish our maximum store of oocytes in fetal life, and the number of follicles remaining declines from birth onward (Block 1952; Richardson, Senikas, and Nelson 1987; O'Connor, Holman, and Wood 2001). Recent evidence in mice (Johnson, et al. 2004; Johnson, et al. 2005) challenges long-standing orthodoxy that, in

 

mammals, no new follicles are produced during adulthood. Still, the net number of follicles declines with age across all mammalian clades (vom Saal, Finch, and Nelson 1994). The physiology of menopause, in particular, is known to be quite similar in humans, chimpanzees, and several species of macaques (Graham, Kling, and Steiner 1979; Gould, Flint, and Graham 1981; Nozaki, Mitsunaga, and Shimizu 1995; M. L. Walker 1995). Unlike other primates, however, humans reach terminal fertility and then menopause when aging is less advanced in other physiological systems (Gosden 1985; Hill and Hurtado 1991; Pavelka and Fedigan 1991; Caro et al. 1995).

Williams (1957) raised the topic of human mid-life menopause explicitly when he addressed the problem of aging from the perspective of evolutionary life history theory. Theory predicts that "there should be little or no post-reproductive period in the normal life of any species," but "at first sight it appears that this prediction is not realized. Long post-reproductive periods are known in many domesticated animals and in man himself. In man it may be even longer than the reproductive period" (1957:407). But Williams (1957:407) went on to say that "these observations lose much of their seeming importance when it is realized that they are largely artifacts of civilization. In very primitive conditions, such as prevailed throughout almost all of man's evolution post-reproductive individuals were extremely rare."

The evidence Williams (1957:407) cited for this rarity was a tabulation of estimated ages at death in a series of Paleolithic and Mesolithic skeletons in which only 3 of 173 specimens "were over fifty and none was much older than this." Twenty-two years later, Williams (1999:407) reiterated the same conclusion: "Young-adult mortality rates in the Stone Age were such that only a trivial minority would live beyond what we now call middle age." I reviewed biases inherent in estimating adult mortality rates and population age structures from such data above. Those known biases, combined with the data from careful hunter-gatherer demographies, are grounds for disputing the common claim, here coming from Williams, that old people are an artifact of civilization.

Williams' other hypothesis (1957:407–408) about the evolution of mid-life menopause in women was this:

> 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. A termination of increasingly hazardous pregnancies would enable her to devote her whole remaining energy to the care of her living children, and would remove childbirth mortality as a possible cause of failure to raise these children. Menopause, although apparently a cessation of reproduction, may have arisen as a reproductive adaptation to a life-cycle already characterized by senescence, unusual hazards in pregnancy and childbirth, and a long period of juvenile dependence.

This "stopping early" hypothesis has stimulated work on the optimal timing of menopause. Trade-offs like those hypothesized by Williams might explain why selection maintains the observed modal age of menopause in women (for example, Hill and Hurtado 1991, 1996; Rogers 1993; Packer, Tatar, and Collins 1998; Peccei 2001; Shanley and Kirkwood 2001). Noting that the age of fertility decline and menopause appears to be about the same in chimpanzees and people whereas human adult mortalities are much lower, Hawkes and colleagues (1998) pointed to the simplest phylogenetic inference: our longevity, not our age at menopause, is derived. Ovarian ontogeny may have remained more or less the same throughout both hominin and chimpanzee lineages from our common ancestor. If so, the age of menopause did not alter; it became a mid-life process when greater longevity evolved in the human lineage. That makes the evolutionary question, what could favor slower aging while not favoring later ages of childbearing at the same time?

E. Trinkaus and R. L. Tompkins (1987) proposed that our slower aging and longer lifespans may not have evolved until the appearance of moderns. They estimated the ages at death of individuals assumed to be among the oldest Neanderthal adults and, combining their findings with other reports, noted "the extreme rarity and possible absence of Neanderthals greater than 40 to 45 years in the fossil record" (Trinkaus and Tompkins 1987:128). Using these data and other lines of evidence, they proposed "the intriguing possibility" that "the significant postreproductive survival of recent humans had not yet

 

emerged among these late archaic humans"; instead, "archaic members of the genus *Homo* [may have] had lifespans similar to those of wild chimpanzees" (Trinkaus and Tompkins 1990:174). They recognized that if this were so, age at maturity in Neanderthals must have been earlier as well. "Retarded developmental rates among Nean-derthals approaching that presumed for recent humans" could not have been favored if adult mortalities were so high (1990:159). Subsequently, concern about the age-at-death estimates on which they relied and about the viability of populations with chimpanzee adult mortality profiles and even slightly slower maturation than chimpanzees led Trinkaus to reconsider those earlier lifespan estimates. Revisiting the evidence, Trinkaus (1995:139) still surmised that young adult mortality must have been high compared with ethnographically known hunter-gatherers, but he concluded "that the Neanderthals had a demographic pattern similar to those of at least some modern human populations."

The under-representation of older adults in fossil remains of both Neanderthals and Neanderthal ancestors is a recurrent finding. Analyzing the assemblage of Middle Pleistocene Neanderthal ancestors from the Atapuerca Sima de los Huesos, J. M. Bermúdez de Castro and M. E. Nicolás (1997:333) reported that "longevity was probably no greater than 40 years." On the basis of more complete analysis of the assemblage and comparison with other Middle Pleistocene European assemblages, Bermúdez de Castro and colleagues (2004:22) concluded "from the fossil evidence that the effective life span of Middle Pleistocene populations in Europe probably did not exceed 40–45."

Employing the same toothwear seriation technique (Miles 1963) used by Bermúdez de Castro and colleagues to age adults, R. Caspari and S.-H. Lee (2004) produced an "OY ratio" of older to younger adults for skeletal dentitions from an array of hominin taxa: australopithecines, *Homo erectus*, Neanderthals, and Upper Paleolithic moderns. Their young adult category consisted of dentitions with the third molar erupted but limited tooth wear. "Older adults were defined as twice the age of reproductive maturation, the age at which one could theoretically first become a grandmother" (Caspari and Lee 2004:10896). The ratio of older to younger adults was taken to be an index of longevity. Caspari and Lee (2004:10898) wrote:

 

> Two important conclusions emerge from this study: first, there is significant increased longevity between all groups, indicating a trend of increased survivorship of older adults through human evolution. Second, the increase is by far the greatest in the early modern humans of the Upper Paleolithic, when for the first time there are a larger number of older adults than younger adults in the death distribution. Whereas high levels of young adult mortality have been noted for Neanderthals (Trinkaus 1995), the magnitude of the increase in OY ratios in the Upper Paleolithic is nevertheless surprising.

Caspari and Lee's OY ratio was 0.39 for Neanderthals and 2.08 for Upper Paleolithic moderns, a fivefold difference.

Problems of taphonomic bias and age-at-death estimations, as well as the unknown relationship between the age distribution of skeletal assemblages and the mortality experience of populations, would apply to these dentitions. Another problem is specific to their definition of old and young adults. The scaling relationships in primate—and therefore probably in hominin—life histories mean that such an OY ratio would likely be insensitive to longevity.

Caspari and Lee chose this ratio because age at maturity is not known for the nonmodern taxa, but as Schultz (1956, 1960) and then B. H. Smith (1989a) had shown, eruption of the third molar is a useful marker of maturity across the primates. By choosing a trait that is "independent of actual ages," Caspari and Lee could avoid contention about the actual age estimates. However, the "invariant" relationship between age at maturity and adult mortality highlighted by Charnov's model of mammalian life history evolution implies that when old is defined as twice the age at maturity, the ratio of old to young adults might be similarly invariant. Because longevity and age at maturity are positively correlated across the mammals, including the primates—even modern humans (Charnov 1993; Charnov and Berrigan 1993; Hawkes et al. 1998; Alvarez 2000)—the same invariance likely held among past hominins. If the relationship between these two variables is approximately constant, then the fraction of adults that are more than double the age at maturity might be about the same, whether average adult lifespans are short or long.

Data on living populations illustrate exactly this (Hawkes and O'Connell 2005). The proportions of adults in the old categories defined as twice the age of M3 eruption can be calculated for standing populations of foraging people and chimpanzees, using data in Smith, Crummett, and Brandt's (1994) compendium of tooth eruption ages in primates, life tables for modern chimpanzees in the wild (Hill et al. 2001), and life tables for three modern hunter-gatherer populations (!Kung: Howell 1979; Ache: Hill and Hurtado 1996; Hadza: Blurton Jones, Hawkes, and O'Connell 2002). The central tendency of M3 eruption in Smith and colleagues' sample of female chimpanzees (n = 7) is 10.7 years; in their sample of women (n = 663), it is 20.4 years. Using Caspari and Lee's definitions, young adult chimpanzees are 10–19 and old chimpanzees, 20 and older. Young women are 20–39 and old women, 40 and older. Assuming that the life tables characterize stationary populations, the ratio of old adults to young adults is 1.09 for chimpanzees and 1.12 for people, very similar proportions of old adults in two species with different longevities.

Adding macaques underlines the point. For Smith, Crummett, and Brandt's (1994) sample of *Macaca fuscata* (n = 570), the central tendency of third molar eruption is 5.7 years. By Caspari and Lee's definition, young adults would be 5–9 and old adults, 10 and older. Using Pavelka and Fedigan's (1999) life table for *M. fuscata* at Arashiyama West and again assuming that the population is stationary, the ratio of old adults to young adults is 0.97. For people, chimpanzees, and Japanese macaques—species with widely differing longevities—the ratio of old to young adults is very similar because the definition of old is scaled to age at maturity.

Konigsberg and Herrmann (chapter 9, this volume) point out other reasons to be cautious about Caspari and Lee's analyses. Their chapter illustrates some of the increasingly sophisticated methods for dealing with the multiple and inevitable sources of error in estimating the age characteristics of death assemblages. Difficulties confronting paleodemographers are daunting but increasingly well recognized. Some, at least, can be corrected so that this line of hard evidence may begin to return solid findings.

More investigation of the similarities and differences among living species will improve understanding of the evolution of our postmenopausal longevity. For example, chimpanzees in the wild rarely

 

live to menopausal ages. This mortality pattern, as well as numerous anecdotal descriptions of chimpanzees beginning to display geriatric symptoms in their mid-30s (Goodall 1986; Huffman 1990; Finch and Stanford 2004), supports the expectation that chimpanzees age faster than people do. In captivity, survival improves at all ages (Courtenay and Santow 1989; Dyke et al. 1995; Hill et al. 2001), so more will live to menopause. As increasing numbers of captives of known birth dates move into the ages of interest (Erwin et al. 2002), we have the opportunity to record variation in ages at menopause and to measure performance of other physiological systems across these ages. Systematic comparisons between humans and chimpanzees should show which functions decline with age at a slower rate in our own species.

**Weaning before Independent Feeding**

Humans wean infants earlier than they can feed themselves and at younger ages than chimpanzees or orangutans do (Robson, van Schaik, and Hawkes, chapter 2, and Sellen, chapter 6, this volume). This relatively early weaning runs counter to the broad pattern across primate species, in which longer adult lifespans and later ages at first birth are usually associated with later weaning ages (Smith and Tompkins 1995; Hawkes et al. 1998; Hawkes, O'Connell, and Blurton Jones 2003; Hawkes, chapter 4, this volume). Variation in rates of maturation to feeding independence has been a topic of special interest in primate life history evolution beginning with Schultz's use of dental markers to identify and compare aspects of primate ontogeny. Schultz (1960:11–13) wrote: "Of the many age changes in development, those regarding the dentition have been studied most intensively, not only because they can be readily observed but also because they serve generally for the estimation of the physiological age of living animals as well as skulls."

In particular, Gould (1977) mentioned Schultz's (1949) observation that in faster-maturing primates the molars appear before the deciduous teeth are shed, so when nursing ends, the molars are ready for mastication. By contrast, in humans the molars emerge after the anterior permanent teeth. Gould (1977:380) cited, in full, Schultz's interpretation that "this alteration is an adaptive requirement of delayed development." Schultz (1950:440) wrote:

> It is tempting to speculate that this human distinction is the

 

> result of some natural selection, directly connected with the extreme prolongation of the period of growth in man. The deciduous teeth of man are not more durable than those of other primates, yet they have to serve in the former for much longer periods than in the latter. Hence this newly acquired precedence for the replacement of milk teeth over the addition of molars is undoubtedly beneficial, if not necessary, for man.

B. H. Smith (2000) has called this "Schultz's rule" and has used the differences that Schultz described in the dental emergence patterns of great apes and humans to assess the relative development of juvenile hominid fossils (B. H. Smith 1986). In Schultz's schematic depiction of variation in duration of life periods across the primates (for example, Schultz 1960, 1969), he used "the appearance of the first permanent teeth" to mark the termination of infancy (1969:147). Although we wean infants earlier, the eruption of the first permanent molars is much delayed in humans, compared with the living great apes (Robson, van Schaik, and Hawkes, chapter 2, this volume). Smith showed that juvenile *A. afarensis*, *A. africanus*, and *H. habilis* specimens fit pongid emergence standards better than human standards whereas a Neanderthal specimen fit the modern human pattern. Noting that "prolonged infant and child dependency appears consistently in theories of early cultural evolution," she concluded that her analysis did not support A. Mann's (1975) view that this was characteristic of hominids before the appearance of *Homo erectus* (B. H. Smith 1986:329).

Smith then investigated relationships between aspects of dental development and life history variables in a sample of twenty-one primate species (including prosimians and both New and Old World anthropoids) to build on Sacher's finding that brain size, longevity, and age at maturity are correlated across the mammals (B. H. Smith 1989a). She found that the ages of eruption of the first molar and of the last permanent teeth were especially well correlated with brain size, as well as with life history variables such as age at weaning, age at first birth, and lifespan. "The dentition has advantages over other markers of maturation in that it is robust to environmental perturbations and has relatively low variance. Thus the dentition provides a growth

 

marker that is reliable…[and] can be extended to species in the fossil record" (B. H. Smith 1989a:686).

Smith used these associations, especially age of emergence of the first molar, to interpret hominin schedules of development, concluding that australopiths displayed maturation rates like modern apes whereas *Homo erectus* and Neanderthals had rates more similar to modern humans (B. H. Smith 1989b, 1991a, 1993, 1994; Smith and Tompkins 1995; Bogin and Smith 1996). Her findings then guided others seeking to explain the evolution of human life histories (Bogin 1999a; O'Connell, Hawkes, and Blurton Jones 1999; Kaplan et al. 2000; Hawkes, O'Connell, and Blurton Jones 2003).

Dental markers do not, however, escape the difficulties with other growth and development variables discussed under heterochrony above. Gould (1977) had used Mann's (1975) assessment that australopithecine juveniles had dental developmental patterns like modern humans and unlike modern apes as evidence of greater retardation in our lineage from its initial radiation. Smith's analyses challenged Mann's conclusions by showing that the relative timing of first molar eruption in australopiths was similar to modern apes and unlike modern humans. Only fossil specimens assigned to genus *Homo* displayed emergence sequences similar to modern humans (B. H. Smith 1994). Other aspects of dental development, however, do not show the same differences, so specialists have found the emphasis on first molar eruption difficult to justify (Macho and Wood 1995b). Macho (2001:189) wrote:

> The pattern and timing of tooth emergence is highly correlated with life-history variables and brain size. Conversely, a firm relationship between molar formation time and life-history variables has not yet been established. It seems counterintuitive that one aspect of dental development should be correlated with life history variables while the other should not.

Schultz had made the point that, compared with other modern primates, some aspects of skeletal development are slowed down in humans and others are not. The same holds for dental development. Crown formation is a variable of special interest because the process is

 

punctuated by daily shifts that leave a record of the number of days it takes a tooth to form. Tooth microstructure thus provides an actual developmental clock for aging juvenile fossils (Beynon and Dean 1988) and perhaps for identifying absolute weaning ages (Katzenberg, Herring, and Saunders 1996; Wright and Schwarcz 1998; Rabb et al. 2004). Macho and Wood (1995b:23) report crown formation data for humans, chimpanzees, gorillas, orangutans, and two fossil hominins: "[A]vailable data on permanent incisor formation times indicate… considerable overlap among great apes and humans.…Canine formation times vary substantially both within and between species.…On the other hand crown formation times of molars are more similar between hominoid species."

With a phylogenetically wider sample of living and extinct primates covering a broader range of body sizes, Macho (2001) found correlations of crown formation times with life history variables and with brain size as well. But "the correlations are relatively high for only a few variables, notably age at weaning, brain size and body mass" (Macho 2001: 196). Considering only her subsample of humans, chimpanzees, gorillas, and orangutans, even these variables are not correlated with molar crown formation times (Robson, van Schaik, and Hawkes, chapter 2, this volume).

Examining a sample of modern humans and fossil relatives and African apes, Dean and others (2001) found enamel formation rates to be faster in anterior teeth of australopiths than in African apes. Although they confirm Smith's picture of eruption sequences in genus *Homo*, commenting that "radiographs, as well as direct observations of developing teeth, show that the sequence of key events during tooth growth in *H. erectus* was identical to that of modern humans" (Dean et al. 2001:629), they found enamel formation times for two *H. erectus* specimens to be faster than times for modern humans. Their analysis of crown formation rates found that only a Neanderthal specimen overlapped the modern human range (Dean et al. 2001). In a larger sample of Neanderthal anterior teeth and measuring a different feature of enamel formation, Ramírez Rozzi and Bermúdez de Castro (2004) found clearly faster formation in Neanderthals than in modern humans. From this, they infer a faster rate of maturation in Neanderthals "because dental growth is an excellent indicator of somatic

 

development" (Ramírez Rozzi and Bermúdez de Castro 2004: 936). But this component of dental development, crown formation times, is not related to differences in age at maturity among living people and other great apes.

Other aspects of dental development and life history variation across primates have been explored by Godfrey and colleagues (2001). They measured dental development in two ways: (1) dental precocity, the fraction of postcanine teeth erupted at various ages, and (2) dental endowment at weaning, the fraction of adult postcanine occlusial area that was present at weaning. A strong association between these measures and adult brain size, but not a close correlation with life history traits, led them to "underscore just how variable dental development at weaning can be. One cannot assume…that dental maturation will be linked in a consistent manner to skeletal growth or reproductive maturation" (Godfrey et al. 2003:197). The details of particular measures, samples, and tests in these important challenges merit careful attention.

As shown by Robson, van Schaik, and Hawkes (chapter 2, this volume), commonly used markers of dental development are not correlated with weaning age across the living hominoids. Weaning does, however, leave an isotope signature in the microstructure of tooth enamel (Katzenberg, Herring, and Saunders 1996; Wright and Schwarcz 1998; Rabb et al. 2004). That could provide a way to identify this important life history variable (see Sellen, chapter 6, this volume) in fossil specimens.

## DIFFERENT KINDS OF EXPLANATIONS BEFORE AND AFTER GENOMICS

Finding systematic relationships between life history features and variables that can be measured in bones and teeth is a necessary foundation for extracting life history information from the fossil record. Different frameworks of ideas have motivated that inquiry and will surely continue to do so in the future. Distinction between the proximate mechanisms that generate a morphological, physiological, or behavioral feature and the adaptive effects that explain why selection favored, spread, and maintained it goes back to Darwin. The apparent simplicity of this distinction seems increasingly deceptive, but that does not make it less important.

 

**Causal Mechanisms, Adaptive Explanations, and Distinctions between Homology and Homoplasy**

N. Tinbergen (1963) famously reminded evolutionary biologists that answers to proximate mechanism questions are not answers to questions about adaptive function, and vice versa. Different kinds of research programs, different data, different hypotheses address these different questions. Tinbergen distinguished four different questions researchers have in mind when they seek to explain a biological feature, four different "whys." One addresses the proximate mechanisms that cause the feature, how the underlying physiology works. This contrasts with functional questions that focus on adaptive effects, asking why selection maintains the feature of interest in the form observed. Perhaps paradoxically in this context, Tinbergen also distinguished both of those from questions about ontogeny or phylogeny. A different research agenda is necessary to discover how a feature develops over the life course of an individual organism, and another to determine when the feature arose in ancestral populations. Both mechanism and adaptive effect questions can also be asked about ontogenetic or phylogenetic patterns.

In the past few decades, the dominance of cladistics (Hennig 1966), the explosion of molecular techniques and data sets (Hillis 1994; Klein and Takahata 2002), and the addition to the comparative method of systematic procedures to correct phylogenetic sampling biases (Harvey and Pagel 1991) have highlighted new complications associated with the distinction between causal mechanisms and adaptive effects. On the one hand, cladistic methods assume that only similarities between taxa that are shared with their last common ancestor (homologies) are relevant to identifying phylogenetic relationships. Similarities between taxa that are not shared with their last common ancestor (homoplasies) are considered potentially misleading for phylogenetic questions.

On the other hand, current practice in testing adaptive hypotheses with cross-species comparisons requires the use of phylogenetically corrected data sets in which species that are homologous for the features of interest are not counted because they are not considered independent cases. Only homoplasies offer independent trials for testing adaptive hypotheses. Classic persuasive illustrations of the power of natural

 

selection show different proximate mechanisms producing similar adaptive effects, for example, adaptations for flight in birds and bats, male and female individuals produced by diverse sex-determining mechanisms, and the convergent evolution of cephalopod and vertebrate eyes.

The distinction between homologous and homoplastic similarities can seem straightforward, but it has become increasingly vexed as phylogenetic relationships are explored in light of evidence of similarities and differences at many levels. Wake (1999:30–31) gives the following example:

> The condition of permanent larvae in salamanders has arisen many times independently…some bones never form in larvae, but even should a permanently larval species reproduce, the bones remain as latent elements. Imagine that [these bones] reappear in a derivative species. These would be identified as homoplasies…in phylogentic analysis, but a morphologist would insist that [each bone is the same] as in distantly related salamanders. It must be a homologue!

Lockwood and Fleagle (1999), reviewing treatments of homoplasy in primate and human evolution, noted that ideas about intrinsic progressive tendencies supported early twentieth-century expectations of widespread homoplasies in primate lineages because each lineage was expected to progress through time from lower to higher grades. After the modern synthesis, orthogenesis was abandoned, but not the idea of adaptive trends recognized as grade shifts progressively evolving more humanlike characteristics. As the primate fossil record expanded, however, "emphasis was placed on identifying unique features of modern taxa in fossils, without undue concern for the implications this may have for parallel evolution or reversals in other features" (Lockwood and Fleagle 1999:194).

With the expansion of cladistics into anthropology, "homoplasy was generally seen [at first] as the result of error due to bad choice of characters and misidentified homology. However, the 'reality' of homoplasy can be seen in the effect that fossil discoveries have had on phylogenies in recent years" (Lockwood and Fleagle 1999:194).

 

> [It] has become common for fossil taxa to be found that belong to a particular clade based on selected data sets or traditional "key" characters, but at the same time reveal parallel evolution among various other traits.…In other words, the combination of traits in the fossil taxon sets up a situation where one body of evidence (e.g., cranial, dental, or postcranial) is substantially homoplastic, and it is unclear which data set to prefer.…Computer programs that are now available permit analyses of large data sets (numbers of taxa and characters) that often generate numerous equally parsimonious trees. (Lockwood and Fleagle 1999:196)

Unusual features of the primate fossil record, or of the features often chosen, might make anthropological cladistics especially thorny. But "statistics…are readily calculated to express the proportion of character change that reflects homology or homoplasy.…Primates do not exhibit unusually high or low levels of homoplasy, and within primates, no single type of data appears to be less homoplastic than other types of data" (Lockwood and Fleagle 1999:196).

The problem is central for establishing phylogentic relationships, but it also plagues phenotypic reconstructions. Life history models show why, given stable population theory and assuming populations to be generally nongrowing, only certain combinations of life history traits can persist—but which, if any, features of the fossil specimens provide reliable signals of any of them?

**After the Genomics Revolution**

Problems of the homology/homoplasy opposition have emerged with special force from discoveries in evolutionary developmental genetics. The complexities apply not only to morphological and behavioral data sets but to molecular ones as well (Sanderson and Donoghue 1989; Patterson, Williams, and Humphries 1993). In light of "evo-devo" findings, Gould (2002:1062) appraised his treatment in *Ontogeny and Phylogeny* as reading

> like a quaint conceptual fossil from an "ancient" time of cross bows and arquebuses.…I can only express my joy and astonishment at a subsequent speed of resolution and dis-

 

> covery that has sustained my predictions, but also made my earlier book effectively obsolete…the field of evolutionary developmental biology…has invented the tools…for decoding the basic genetic structure of regulation.

Because "extensive genetic homology for fundamental features of development does hold across the most disparate animal phyla" (Gould 2002:1066), Gould (1123) concluded that evo-devo has finally laid "selectionist orthodoxy" to rest:

> This general shift in viewpoint—from a preference for atomistic adaptationism (favoring the explanation for each part as an independent and relatively unconstrained event of crafting by natural selection for current utility) to a recognition that homologous developmental pathways (retained from a deep and different past, whatever the original adaptive context) strongly shape current possibilities "from the inside"—has permeated phylogentic studies at all levels, from similarities among the most disparate phyla to diversity among species within small monophyletic segments of life's tree.

Gould (1123) summarizes "the homology of developmental pathways in homoplastic eyes of several phyla" to demonstrate that selection is subordinate to developmental mechanisms. However, the findings also highlight the power of natural selection. Reviewing the book, D. Futuyma (2002:661) wrote that "Gould's excitement (which I share) about contemporary 'evo-devo' is palpable.… (I, however, do not agree that the convergence of vertebrate and cephalopod eyes, in which some 'master' genes play common roles, has lost its role as testament to the power of natural selection)." Patterns of growth and development vary among closely related taxa with different elements of that variation uncoupled from one another, while the same genetic control mechanisms are found among taxa not only in different phyla but in different kingdoms.

> "The more we learn about the genome, the more it teaches us about our own place in the web of life," said Robert May , an evolutionary biologist and president of the Royal Society in Great Britain. "For example, we share half our genes with

 

> the banana. (Actually, it would be more accurate to say bananas share half their genes with us, because their genome is smaller.) This is a fact more evident in some of my acquaintances than others." (*Discover* 2001:62)

The genomics revolution has provided a radically new view of the diversity of life (for example, Adoutte et al. 2000), to which phenotypic characteristics can be a poor guide. J. Klein and N. Takahata's (2002: 199) comments capture a "post genomics" view of human/chimpanzee comparisons:

> All the estimates obtained by both older and newer techniques have yielded very similar figures: the genomes of these two species differ at approximately 1–2 percent of their nucleotide sites.…Science writers and even some scientists appear to be flabbergasted when confronted with the sequence divergence between the human and chimpanzee genomes, but they shouldn't be, because the observed value matches the expectation very well. It would be indeed astounding if the value had turned out to be much greater than 2 percent.

They suggest that "the impression of a gap between the small differences at the molecular level to the seemingly large differences at the phenotypic level may arise because the intervening steps between the genotype and the phenotype, between the DNA and the appearance of an organism, remain unidentified" (Klein and Takahata 2002:204).

The difference between people and chimpanzees also seems remarkable because of the tendency to misestimate the rate of change required to produce the phenotypic differences. As G. C. Williams (1992:132) pointed out:

> Data on Pleistocene human evolution are interpretable in various ways, but it is possible that the cerebrum doubled in size in as little as 100,000 years, or perhaps 3000 generations (Rightmire 1985). This according to Whiten and Byrne (1988) is a "unique and staggering acceleration in brain size." How rapid a rate is this really? Even with conservative assumptions on coefficient of variation (e.g., 10%) and her-

 

> itability (30%) in this character, it would take only a rather weak selection (s = 0.03) to give a 1% change in a generation. This would mean a doubling in 70 generations. An early hominid brain could have increased to modern size, and back again, about 21 times while the actual evolution took place.

Thirty years ago, King and Wilson (1975) surmised that differences in regulatory mechanisms mostly account for the phenotypic differences between people and chimpanzees. This hypothesis continues to be a useful guide as new technology allows much more precise comparison of genomes. The two species differ in levels of gene expression, the location of recombination "hot spots," and the evident strength of selection on particular genes (Enard et al. 2002; Caceres et al. 2003; Olson and Varki 2004; Nielsen et al. 2005; Winckler et al. 2005). Insertions, deletions, and duplications account for more differences than single nucleotide substitutions (Cheng et al. 2005; The Chimpanzee Sequencing and Analysis Consortium 2005).

One genomic signal of the differences in life history is revealed by analyses of changes in gene expression with age in human and chimpanzee brains (Fraser et al. 2005). Now the power of molecular techniques to measure both similarities and differences far outstrips understanding of their phenotypic consequences. Positive selection can be detected in the absence of clues about the actual fitness costs and benefits conferred. The challenge is to link the evidence of selection to phenotypic consequences (Li and Saunders 2005). As S. B. Carroll (2005:1164) noted in an Allan Wilson Memorial Lecture, "the great and difficult challenge, with the genome sequences of humans, chimpanzees, and other mammals now available, is to map changes in genes to changes in traits."

Study of genetic correlations with intraspecific variation in aging rates among humans and among chimpanzees will also provide more evidence of how rates of aging shifted in our lineage. Genetic diversity in living human populations may even contain evidence of differences among nonmodern populations (Eswaran, Harpending, and Rogers 2005). For the most part, however, learning about the phenotypes of our ancestors and extinct cousins necessarily remains the "ancient"

 

problem that Gould laid out in 1977: "The data of heterochrony represent the only confident estimate that classical macroevolutionary morphology can supply for the importance of changes in regulation" (Gould 1977:408). It is the fossil and archaeological record that can show where and when brains, bodies, and life histories changed in the hominin clade. Discovering what the lives of the ancestors and cousins of modern humans were like, how they differed from us and from other modern primates, how they differed from one another, what happened in our lineage before the appearance of modern humans, where, when, and why, requires placing the fossil and archaeological clues within frameworks for describing and explaining the variation we can examine in living species. To discover what phenotypes selection favored and when and why, we need well-warranted hypotheses about the variation in the present and also ways to take those hypotheses to the tangible remains of a different past.

## ADAPTIVE HYPOTHESES ABOUT THE EVOLUTION OF HUMAN LIFE HISTORIES

Gould in 1977 had expected morphological variation to be mosaic because "the evolutionary direction of each feature is controlled by natural selection; the capacity for independent variation of characteristics is very great" (Gould 1977:364–365). He had also cited Cole's (1954) demonstration that life history features cannot vary independently. Subsequent work on evolution in age-structured populations has elaborated both the theory and the empirical record on relationships among age at maturity, annual fecundity, and adult and juvenile mortality. The life history models do not depend on particular mechanisms, applying as they do across species in which those mechanisms might (or might not) be different. That gives them a welcome generality for applications to a different past. At the same time, the estimates of life history variables in past populations must come from fossil evidence, so lineage-specific mechanisms that link morphological features to life history variables in primates—size and aspects of dental development, for example—become crucial tools. Genomics promises to add to this a much better understanding of what changed in the evolution of our own lineage and that of our sister species, chimpanzees. The fossil and archaeological records tie those changes to time and place, and a life

 

history framework supplies the help of linking features to one another. When some life history traits can be confidently estimated, the result is a strong prediction about others. Explicit attempts to take advantage of those interrelationships in constructing and revising adaptive hypotheses are recent, but they build on a venerable foundation.

In the fifties and sixties, S. L. Washburn departed from most anthropologists in arguing that comparative morphology supported a quite recent common ancestor of modern humans and African apes, something the molecular evidence subsequently confirmed (Goodman 1962; Wilson and Sarich 1969; King and Wilson 1975). As Lockwood and Fleagle (1999:195) noted, Washburn and his students repeatedly pointed out that the "fossil phylogenies based on dental similarities [like tooth size] between particular 'Miocene apes' and living hominoid genera implied tremendous parallel evolution of postcranial similarities among modern hominoids." Contrary to many anthropologists, Washburn preferred a "more parsimonious phylogeny: a clade of Miocene apes preceding the radiation of living hominoids. This view in many ways laid the groundwork for current understanding of fossil hominoid relationships" (Lockwood and Fleagle 1999:195).

Washburn had the widest influence on thinking about human evolution through his elaboration of the Hunting hypothesis. Its satisfying, adaptive logic is one of the reasons it is so widely enlisted to explain human evolution. His emphasis was on links among hunting, tools, bipedalism, and brains, but he used these to explain what Gould called the "hallmarks of hominization." Building on Dart's (1949) savanna hypothesis about the likely importance of hunting and therefore tools and brains to explain the bipedalism of australopithecines, Washburn (1960) proposed that the combination of enlarging brains and a pelvis shaped for bipedalism created an "obstetrical dilemma" for mothers. Shifts in the timing and rate of fetal, infant, and juvenile development were necessary consequences. Most brain growth had to be postnatal, slowing other aspects of infant development. Undeveloped neonates required more maternal protection and support, and juveniles needed a longer developmental and learning period before reaching adulthood. The conflict between maternal care requirements and hunting led mothers to pair with hunting mates who supplied paternal provisioning to fund the greater dependence of human infants and juveniles for a

 

longer time. A sexual division of labor with paternal provisioning made nuclear families into units of economic and reproductive cooperation. Gould (1977:400) mentioned Washburn as one who had recognized the significance of delayed maturation. But Washburn did more than that. He elaborated an adaptive hypothesis that linked slow development to the fitness benefits for an ape that could eat better by hunting in savanna environments (Washburn 1960; Washburn and Lancaster 1968; Washburn and Moore 1974).

Washburn drew comprehensively on hominid fossils, Paleolithic archaeology, comparative studies of nonhuman primates, and studies of modern hunter-gatherers. He used the consensus picture that had emerged in each of these fields through the sixties and early seventies to fill out the scenario. Ethnology gave a fundamental role to sexual divisions of labor, making nuclear families the basic elements of human social organization (for example, Murdock 1949; Sahlins 1972). Other primates generally, and other great apes in particular, lacked nuclear families and were not known to hunt (Lancaster and Lancaster 1983). The paleontological record suggested that bipedal locomotion and enlarged brains emerged contemporaneously, and this appeared to be more or less coincident in time with the earliest archaeology (Washburn 1960).

Challenges began to mount in the late seventies, with clear evidence that bipedalism preceded the appearance of stone tools and expanding brains by at least a million years (Johanson and White 1979; see Skinner and Wood, chapter 11, this volume). Chimpanzees were discovered to be regular hunters (Goodall 1968, 1986; Stanford 1999). The home base interpretation of concentrations of stone tools and the bones of large animals in Lower Paleolithic sites was critically questioned and largely rejected (Binford 1981; O'Connell, Hawkes, and Blurton Jones 1988; O'Connell et al. 2002). Archaeologists now recognize that clear evidence of hominin big-game hunting does not appear until the late Middle Pleistocene, more than 1.5 million years after the earliest stone tools and at least a million years after the appearance of genus *Homo* (O'Connell et al. 2002; Stiner 2002). Systematic study of foraging and food-sharing patterns among living hunter-gatherers showed that hunters provide little of the meat eaten by their own wives and offspring; most is eaten by consumers outside the hunter's own

 

family (Kaplan and Hill 1985; Hawkes, O'Connell, and Blurton Jones 1991, 2001b; Hawkes 1993).

Some view the challenges as decisive grounds for rejecting the Hunting hypothesis (for example, Zihlmann and Tanner 1978; Dahlberg 1981; O'Connell, Hawkes, and Blurton Jones 1999; Hawkes, O'Connell, and Blurton Jones 2001a; O'Connell et al. 2002). Others argue that the Hunting hypothesis still captures major components of what happened in human evolution (Tooby and DeVore 1987; Deacon 1997; Kaplan et al. 2000). Adaptive hypotheses about the evolution of human life histories that are founded on these alternative assessments are discussed in chapter 4.

A lesson of this review is that what we know about the past is much more complicated than it used to be. Each line of inquiry develops conceptual and modeling tools and bodies of evidence that highlight different questions, driving what practitioners investigate, what they debate, and what they take for granted. That is supposed to be how knowledge increases. But the difficulty of conversation across lines of evidence grows as a consequence, and notions falsified in one field of inquiry remain common assumptions in others. Some things that seem contradictory may be what we should expect of a different past, as Gould anticipated in 1977 about heterochronic changes in human evolution. The problem of discovering which aspects of the fossil and archaeological record give the best indices of ancestral life history variables has no clear general solution, at least not yet. Alternative hypotheses applied to multiple lines of evidence expose many contradictions and pose new measurement and modeling challenges. The one certain thing is that recent developments give us all a lot to do.

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Life-history theory can help us identify the genetic and environmental factors that favored the evolution of the unusual human life-history, which has changed drastically in the last two centuries. We discuss why a quantitative genetic perspective can provide insight into human life-history evolution and review recent studies revealing how quantitative genetic techniques have determined how cultural and environmental changes have altered selection pressures. These studies illustrate the difficulty of predicting evolutionary change in future generations but nevertheless provide a means to study