Human Growth: Evolutionary and Life History Perspectives

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Abstract

Evolutionary and life history perspectives allow a fuller understanding of both patterns of growth and development and variations in disease risk. Evolutionary processes act to ensure successful reproduction and not the preservation of health and longevity, and this entails trade-offs both between traits and across the life course. Developmental plasticity adjusts the developmental trajectory so that the phenotype in childhood and through peak reproduction will suit predicted environmental conditions – a capacity that may become maladaptive should early-life predictions be inaccurate. Bipedalism and consequent pelvic narrowing in humans have led to the evolution of secondary altricialism. Shorter inter-birth intervals enabled by appropriate social support structures have allowed increased fecundity/fitness. The age at puberty has fallen over the past two centuries, perhaps resulting from changes in maternal and infant health and nutrition. The timing of puberty is also advanced by conditions of high extrinsic mortality in hunter-gatherers and is reflected in developed countries where a poor or disadvantaged start to life may also accelerate maturation. The postpubertal individual is physically and psychosexually mature, but neural executive function only reaches full maturity in the third decade of life; this mismatch may account for increased adolescent morbidity and mortality in those with earlier pubertal onset.

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Some General Evolutionary Principles

The application of evolutionary thinking to human biology and medicine can be beset by misconceptions. This is because evolutionary processes are not directly concerned with issues of health, disease or longevity but with ensuring successful reproduction. Selective processes operate to maximize reproductive success either directly or indirectly (e.g. through kin selection; see glossary in the appendix), whereas genetic drift and population bottlenecks operate through stochastic processes to exclude some genotypes from successive populations. Selective processes (natural, sexual or social) depend on variation in traits, heritability of some underpinning elements of a trait, and differential reproductive success associated with the trait. Human fitness is to a significant extent determined by survival in the first two decades of life as well as reproductive success following puberty, and is much less influenced by later life events [1]. In contrast, later health and longevity are influenced by such events, exacerbated by the decline in repair processes associated with antagonistic pleiotropy [2].

Key to understanding both macro- and microevolution is the concept of trade-offs. No trait evolves in isolation, and constraints thus exist that limit the range of solutions possible. For example, the size and shape of an animal can be limited by both ecological and energetic constraints, and in the case of terrestrial animals by skeletal constraints [3]. Life history theory considers trade-offs from another perspective, namely the interaction between traits defining a population's life history, and these are discussed below.

Co-evolutionary pressures also need to be considered. Humans did not evolve in isolation from other species; we recognize this well in terms of the defense systems such as stress responses and innate and acquired immunity, and more recently the importance of the gut microbiome to human biology has started to be appreciated. Humans also evolved within social frameworks and with the capacity to create technologies that changed their physical, biotic and social environments. In doing so, potential mismatches between their evolved biology and their environment and lifestyle have been created, and this can affect their health. In recent times, the health impact of such changes with respect to metabolic disease has become particularly apparent.

Microevolutionary processes presumably continue in humans, but the extent to which they are important in a world where technological control of reproduction and survival is widespread, at least in Western societies, remains controversial. One of the problems is that ‘fitness’ is an inclusive concept; it effectively refers to reproductive success which in humans is influenced by both biological and cultural evolution. Estimates of fitness taken from modern populations do not easily distinguish between these two components. The impression gained from an analysis of data derived from the Framingham Heart Study, namely that humans are evolving to be shorter [4], highlights the problems of conflating these concepts. The method of calculation of fitness is sophisticated, but there
can be many reasons why fitness is greater in shorter people. As Nettle [5] has pointed out, fundamental changes in life history strategies are associated with being poor or disadvantaged, and these could be manifest as a relatively greater reproductive success.

The Role of Developmental Plasticity

Genotypes do not have a one-to-one relationship with phenotypes. Environmental factors may disrupt phenotypic development, acting as teratogens and leaving the organism to accommodate phenotypically, assuming that the disruption does not lead to loss of viability. With social support and medical technologies, humans can survive to reproduce even with significant developmental disruption, and in that sense phenotypic accommodation can be seen as potentially adaptive [6].

More universally, development is influenced by an ecological range of external environmental influences. In the case of the mammal, a physiological range of maternally created intrauterine environments influences the developmental trajectory such that a range of mature phenotypes can appear. This range is termed the reaction norm, that is the range of environmentally inducible phenotypes that can be elicited from a single genotype. The underlying mechanisms of developmental plasticity include epigenetic change, and are found across all taxa and have the adaptive potential to adjust the phenotype to the actual environment.

We have classified developmental plasticity as being of two types [7]. First is where the adaptive response is closely linked in time to the inducing environments; fetal growth retardation can thus be viewed as an adaptive response of the fetus to a disturbed nutritional environment, and the adaptation is reduced growth to allow it to survive to birth and have the opportunity to reproduce. The second and more frequent form of adaptive developmental plasticity need not induce immediate responses, rather the phenotypic difference appears later in the life course. For example, it is now apparent that relatively unremarkable changes in maternal nutrition can induce phenotypic changes in the offspring which emerge some time after birth [8]. Such developmental programming can be seen as adaptive if it can enhance the potential of the individual to reproduce successfully. We termed this type of response a predictive adaptive response, because the adaptation is made in anticipation of a predicted later environment where a particular range of phenotypic traits is anticipated to be more advantageous to fitness [9]. Bet-hedging is an alternative strategy used in fast-reproducing organisms to enhance fitness in variable environments [10]. In contrast, where the environmental change is in the order of generation times, and particularly in species with a low number of offspring such as the human, modeling shows that even somewhat inaccurate predictions offer a fitness
advantage [11]. It is important to reiterate that the adaptive responses of importance are those that occur during childhood and the early reproductive years if they are to promote successful reproduction [1]. Later life consequences need not be adaptive.

Evolution is to a large extent blind to what happens later in life. Thus early life insulin resistance may be an adaptation of advantage to allow metabolic survival and promote accelerated maturation in nutritionally poor circumstances; however, this response later in life may be maladaptive if the prediction is wrong and the nutritional environment is obesogenic – this will be manifest as metabolic disease. In this situation, the metabolic disease is a consequence of the mismatch between the predicted environment and the actual environment, but the adverse health consequences are likely to occur only after peak reproduction and thus are of little consequence to fitness. Indeed in our evolutionary past, developmentally induced insulin resistance would have been of little consequence given the low likelihood of exposure to an obesogenic environment.

This delayed anticipatory response may occur in association with, or in isolation from, more immediate adaptive responses. For example, children who are born small are more likely to have permanent growth retardation. The initial growth retardation is an immediate adaptive response, while the persistent growth failure in childhood and stunting can be envisaged as a predictive adaptive response to anticipated nutritional deprivation. The apparently delayed obesity and insulin resistance in children born small [12] can be similarly interpreted as a predictive response in association with the immediate fetal growth response. The recent observation of an increased risk of obesity in adults who were first-born children [13] highlights how subtle developmental influences (the increased maternal constraint associated with primiparity) can induce predictive adaptive responses, in this case a metabolic phenotype more appropriate for a lower nutritional plane then mismatched in an obesogenic environment. There is growing evidence for the central role of epigenetic mechanisms in adaptive developmental plasticity [14].

**Life History Theory**

Life history theory considers the evolution of, and the trade-offs between, traits directly related to reproductive success. This evolved suite of traits defines the basic characteristics of a species: their patterns of growth and maturation, their longevity and phases of development, the number of offspring they have and so forth. Such considerations cannot be isolated from the ecology of the species and the social structure of the population. Subject to nutrient availability, being large as an adult is in general an advantage for survival, but growth takes time. Thus, the longer it takes to reach the age of maturation,
the greater the risk of dying before reproducing. Consequently, a key trade-off is that between investing to grow larger and investing in reproduction. Similarly, there is a trade-off between the quality and number of offspring that themselves need to survive to reproduce. In addition, there is a further trade-off between current and future reproduction – in humans this can be manifest in the lower viability of offspring associated with shorter inter-birth intervals. It is important to emphasize that evolution can only act on traits that vary and have a heritable element. There is an extensive literature on the evolution of life history traits [15].

**Heritability, Genetics, Non-Genetic Inheritance and Phenotypic-Driven Evolution**

Heritability is a concept which, while often used to reflect genetic determinism, simply refers to the extent to which phenotypic variation can be explained by intergenerational influences. Certainly, the genetic basis of heritability is dominant, but is not the only factor. Estimates of heritability are dependent on the environment of the lineage – the heritability of height is far greater in conditions of high nutrition than in those of changing or poor nutrition where stunting may affect different generations/individuals to different extents. There is increasing interest in the role of non-genomic inheritance through trans-meiotic passage of epigenetic marks, or indirectly through mechanisms which lead to the re-creation of the inducing environment in each successive generation [16]. For example, stunted women are more likely to give birth to children who are small because of greater maternal constraint, and children who are born small are themselves more likely to grow up to be small, particularly if the environment remains disadvantageous. The less than optimal intrauterine environment may thus induce epigenetic changes in the offspring which are again induced in successive generations, but the epigenetic mark has not passed meiosis. West-Eberhard [17] in particular drew attention to the potential for developmentally induced changes to be fixed in a population by the poorly elucidated processes of genetic assimilation, which may play a role in evolutionary processes [6].

**An Evolutionary and Life History Perspective on Human Growth**

The human life course and patterns of growth and development must be understood in the context of the above discussion. However, one of the difficulties of applying evolutionary biology to human biology and medicine is that while it is easy to develop evolutionary arguments and hypotheses, testing such hypotheses is in general indirect. Nevertheless, a conceptual framework for doing so does exist [18].
The human can be characterized as a long-lived species that has low fecun-
dity, a long pre-reproductive phase compared to the life span, with relatively
altricial offspring who have a high dependence on its parents for many years
after birth. It is also a generalist species capable of living successfully in a range
of ecological environments, and one with particular potential to undergo rapid
cultural evolution as a result of its capacities for language, social structure and
technology.

The hominin clade has been bipedal for perhaps at least 4.4 million years. As
the hominin clade has evolved, it has grown in stature such that skeletal remains
of *Homo erectus* members who lived some 1.8–1.3 million years ago suggest a
final height of between 150 and 185 cm. But perhaps of greater importance in
understanding the human life history has been the growth of the human brain
from a volume of about 450 cm³ in the earliest *Australopithecines* to about 1,250
cm³ in modern *Homo sapiens*. The development of a bipedal gait, the adaptive
advantage of which remains speculative, requires a relatively narrow pelvis to
be able to run, and this presumably had adaptive advantage both for hunting
and escaping predators. But the human infant cannot be delivered through the
pelvic canal at the same level of brain maturity as are other primate infants born
to quadripedal mothers with a wider pelvis. Thus there has been a trade-off –the
human life history is based on secondary altricialism, that is giving birth to very
dependent immature offspring, at a shorter gestational length and thus smaller
head size. This prolongs the time of absolute dependence of the offspring on its
mother for mobility and sustenance. In turn this may have played a role in the
development of the human social structure, which in turn allows for infants to
be well supported through this period of immaturity. There is evidence relat-
ing the complexity of human social structure to mature neocortical and thus
total brain volume [19]. In comparison to other members of the primate clade,
a larger proportion of human brain growth occurs postnatally, and this in turn
may provide an explanation for why humans are the fattest mammalian species
at birth. Kuzawa et al. [20] have suggested that neonatal and infant obesity is an
adaptation to provide a metabolic buffer for the brain in infancy, when diarrhea
or malnutrition may otherwise threaten brain development. In this regard, it is
interesting to note that infantile adiposity falls at about the age when infants are
presumed to have been weaned in the Paleolithic.

Gestational length itself is subject to environmental effects. There is evidence
for example that maternal nutritional state at conception can influence gesta-
tional length, with mild undernutrition associated with longer gestational length
and more severe undernutrition with shorter gestational length. Pediatricians
have long recognized the relatively precocial maturation of mildly premature
infants. This suggests that in the event of nutritional stress, the fetus can accel-
erate its maturation and shorten its gestational length in the anticipation that
a longer pregnancy is more likely to lead to intrauterine death. The potential
adaptive advantage to both mother and fetus is apparent.
Humans have relatively short inter-birth intervals given this prolonged period of infant dependency, and this provides a fitness-enhancing strategy because fecundity is relatively greater. However, this is only possible because the social structures allow weaning to occur before the infant is able to fend for itself nutritionally and in other ways, but yet can be supported by adults. This demonstrates how the evolution of human life history has been influenced by the evolution of social structures, and vice versa.

Bogin [21] divides the prepubertal period of *H. sapiens* life cycle into three phases: infancy, childhood and the juvenile phase. In turn, he and Hochberg [22] have related these phases to distinct transitions in growth. In this model, infancy is seen as a continuation of the fetal period of growth; the rate of linear growth has started to decline in fetal life and while still rapid is declining through infancy, lasting until weaning. Based on contemporary hunter-gatherer societies, this is assumed to have been generally between 2 and 3 years of age. However, the endocrine control of growth changes during that period, with growth hormone dependency emerging in the first year of life [23]. Further, Karlberg [24] would argue that phases of growth as illustrated by the infant-child puberty model of growth show a transition from the infant to childhood phase earlier than that suggested by the timing of weaning. This simple debate highlights the difficulties of directly linking one aspect of maturation to a particular single circumstance; life history itself represents the outcome of a set of trade-offs to optimize fitness.

Childhood is generally defined by the period between weaning and adrenarche [25] and is a period of continued parental dependency. A key component of this phase is the replacement of deciduous teeth with permanent teeth – by adrenarche the first four molars have generally erupted. The period between adrenarche and the onset of puberty is increasingly recognized as a distinct phase of development, termed the juvenile period. In behavioral terms, the juvenile has some level of independence but is neither reproductively competent nor socially independent; a similar phase is also seen in many other species. In the human, brain size is maximal by the start of the juvenile period, although brain maturation continues into the third decade of life [26]. It has been frequently suggested that this is a critical phase for developing social skills for living in a group. But earlier experiences can also influence the development of such executive functions.

It has been suggested that prolonged childhood is a unique feature of the human life cycle [21]. In part, this apparent prolongation may simply reflect the relatively short infantile phase which allows the mother to reproduce earlier and thus increase her fitness. It also reflects the longer duration of postnatal brain growth which is not complete until the end of the childhood phase, which is in turn a reflection of both the secondary altricial nature of human gestation and the relatively large mature brain size of humans.
**Puberty**

In life history terms, survival to the age of pubertal maturation allows the individual to reproduce; we have discussed above the trade-off between the age at reproductive maturation and mature body size across species. The timing of puberty shows considerable variation within and between human populations, and this plasticity may be adaptive in that it allows a number of developmental factors to influence the timing of puberty. Two uncertainties enter this discussion: the longevity of humans in evolutionary history, and thus, the timing of puberty. While the average life expectancy in the Paleolithic is generally thought to have been low (perhaps 25 years at birth, or about 35 years if one survived infancy), there is ample paleological evidence suggesting that a significant number of individuals did survive into the sixth and seventh decades of life. Arguments over the evolutionary origins of the menopause, not the topic of this paper, rely in part on the assumption of there being a fitness advantage of living into old age; that generates an indirect fitness effect of the grandmother assisting her daughters with their mothering duties, thus promoting grandchild survival.

There is much more uncertainty over the timing of puberty in the Paleolithic. Based on the timing of the third molar (M3) eruption, which in other primates is generally at the time of sexual maturation [27], it has been generally stated that humans evolved with the age of pubertal completion late in the second decade of life. However, if in the Paleolithic where the average life expectancy is shorter, perhaps the age of M3 eruption and pubertal maturation became disassociated. Given the high risk of not surviving to reproduce, an earlier age of puberty would have had a beneficial fitness effect. Evidence to support this comes from a study of extant hunter-gatherer populations which demonstrates a broad range of ages at menarche, including some with menarche at as low as 13 years of age [28]. Such studies have their limitations, even though they are often used to extrapolate backwards into evolutionary time, but the finding that the age at menarche inversely correlated with the chances of surviving to the age of 15 in these populations does support the hypothesis that earlier maturation is an adaptive plastic response to situations of high mortality. These studies also suggest, along with observations of the secular trend in the declining age at puberty and the effect of migration in infancy, that the reaction norm for the age at puberty extends to a young age. This likely reflects the protection of the capacity to respond in this way, and suggests previous periods of young pubertal ages across evolutionary time.

There is a well-documented secular decrease in the age at puberty in females based on the timing of menarche [29]; more recently a similar trend in the male has been documented by the declining age of peak pubertal growth spurt [30]. In historical terms, this appears to reflect the increase in child health and nutrition during the late 18th century, but it may actually be driven in part by the change in maternal nutrition and early developmental effects, as recently
demonstrated by accelerated puberty in the rat following enhanced maternal nutrition [31]. Given that child health likely declined following the development of agriculture and increasingly dense urban living, we have suggested that the secular trend may be taking human maturation back to what it was during the Paleolithic [32].

The plasticity of human maturation is highlighted by the evidence of prenatal and postnatal nutritional and postnatal stressor influences on the age of puberty. Puberty is accelerated by prenatal undernutrition, as reflected in low birthweight, in both humans [33] and rats [31] and by postnatal stress [34]. Relative adiposity in the juvenile period is associated with accelerated puberty particularly if low birthweight is also present [33]. The most obvious manifestation of this is in the very early puberty seen in children who migrated from underdeveloped countries to Western environments in infancy [35]. Such patterns of maturation can be interpreted in evolutionary and life history terms using the predictive adaptive framework, as children who start their life predicting a high risk of extrinsic mortality accelerate their maturation to reduce that risk and increase the likelihood of reproducing. The predictive model makes it more likely that such children will become obese if exposed to an obeso-genic environment. Conversely, if there is juvenile undernutrition, puberty is delayed in the expectation that the delay may allow nutritional circumstances to improve and thus allow the mother-infant dyad a better chance of survival. Similar arguments can be developed for pubertal responses to abuse and deprivation in infancy.

It has been suggested that the pubertal growth spurt is unique to humans. But sexual dimorphism becomes exaggerated or appears in many mammals during puberty. Indeed, skeletal and body size changes are common in many mammals during puberty. The key difference is that in humans, linear growth is the most obvious change, although human puberty as in other species is also associated with gender-dependent changes in body composition. While the growth spurt is particularly prominent in the human, this may be the inevitable outcome of bipedalism. All other primates are quadripedal, and the simple mechanical differences in how increases in body size might affect locomotion may constrain such increases in size, which are important to fitness – the increase in body size being largely linear in humans and more cuboidal in other species. It is clear that large body size provides a fitness advantage to males due to its impact on sexual competition, and in females it provides greater likelihood of successful support of pregnancy.

The relative roles of natural and sexual selection in determining pubertal growth patterns and the development of sexual characteristics remain speculative. Presumably sexual selection played a significant role in the patterns of hair distribution and the development of body shape. The nature of adult body proportions in different members of the Homo genus has been the subject of extensive study by paleoanthropologists and by those who have used climatic
and environmental arguments to posit explanations for the very different body shapes and sizes seen across modern human populations. For example, adaptive changes in response to cold exposure have been suggested as the basis of shorter limb length and flattened nasal and facial shape in populations living in very cold climates, such as the Inuit.

Thus, while the pubertal linear growth spurt is essentially unique to the hominin clade and probably evolved in early *Homo* species, puberty itself is a prolonged process in other primates. There remains a period of ongoing maturation which, depending on the reproductive strategy of the species, can extend for some time before the role as an adult is completely established. But it is particularly in the modern human that this prolonged phase of behavioral maturation extending beyond physical puberty becomes prominent.

**Adolescence**

While puberty is a solely biological process, the completion of adolescence is both biological and cultural. We have defined adolescence as being completed when the individual is fully accepted as an adult within society. The extent to which such acceptance is a biological and/or a social construct is not resolved. We now know that in Western societies, neural executive function is not fully mature until the third decade of life. This is reflected in structural changes in the frontothalamic pathways which extend well into the third decade [26]. During this period of postpubertal adolescence, the individual is exposed to a mature endocrine milieu and has a relatively mature body habitus, but has immature self-control and emotional control and the ability to show judgment is incompletely matured. Risk taking behaviors in this period are enhanced [36]. This postadolescent phase appears to have become particularly prolonged in recent decades, in part because the age at puberty has fallen and in part because in Western societies the age of acceptance as an adult has risen.

The key evolutionary question here relates to the age of full maturation of frontothalamic pathways. Is it that in the evolutionary past the age of brain maturation was the same as it is now, but in the simpler social conditions of clan societies, the advanced functions were of lesser importance and the individual was accepted as an adult at an earlier age? Is it that the period required for frontothalamic maturation takes longer in a more complex modern society? Or that some aspect of how infants and children are reared in modern societies delays maturation of frontothalamic pathways? We have raised the question of whether the shift in focus of early childhood experience from executive function (social) learning to cognitive learning may play a role [37]. These questions are amenable to empirical study and are of importance because the answer has practical implications. It is reported that children with earlier ages of puberty, that is with greater mismatch to their sociological environment, have far greater
morbidity in adolescence – for example the rate of attempted teenage suicide is much greater in boys who have an earlier age of puberty [38]. Girls who have menarche at a very early age have a greater likelihood of developing eating disorders [39].

**Final Remarks**

Physicians are used to thinking about their patients in proximate terms – that is the mechanisms and causes of disease – in a manner that leads to direct therapeutic intervention. But as this paper has suggested, there is a second layer of interpretation needed – that of understanding ultimate causation in evolutionary and phylogenetic terms – if we are to have a full understanding of the human condition [40]. A life history perspective gives valuable and utilitarian insights into the understanding of growth, both in terms of skeletal growth and body composition and in terms of functional maturation of the individual. But as Nettle [5] has recently noted, a life history perspective also allows a broader interpretation of many aspects of human reproductive biology and behaviors in populations who are disadvantaged.

**Appendix**

*Glossary of Evolutionary Terms Used*

**Adaptation** – refers to the selection of a trait which has been shown to have a fitness advantage. This is a difficult test, and is often inferred rather than proven from data suggesting that the trait promotes the organism's (and its offspring's) survival and reproduction in the environment in which they live.

**Antagonistic pleiotropy** – a selected trait may have beneficial effects that promote fitness in early life but may incur costs later in life.

**Fitness** – generally refers to the successful survival and reproduction of an organism allowing gene flow to the next generation.

**Genetic drift** – refers to the stochastic nature of the chances of an allele being passed onto the next generation. At a population level, it leads to changes in gene frequency not based on selection.

**Heritability** – the degree of phenotypic variation which can be accounted for by intergenerational influences. In quantitative genetics, it refers to the ratio of genotype-induced variance to the total phenotypic variance of a population.

**Kin selection** – the concept where an organism adopts evolutionary strategies that promote its relatives' fitness, even if it is at the expense of its own individual fitness. It is based on the idea that an organism shares a greater proportion of its genes with a relative than with another unrelated organism; hence, it may operate particularly in social animals wherein a high degree of relatedness is present.

**Maternal constraint** – maternal and uteroplacental factors that modulate fetal size. This limits fetal growth to allow birth through the narrow pelvic canal.
**Microevolution** – evolution of altered characteristics within a species due to genotypic and phenotypic change.

**Macroevolution** – evolution of a species; essentially an accumulation of microevolutionary change resulting in reproductive isolation between populations.

**Natural selection** – the process by which natural variation in a trait affects survival and fitness and leads to alterations in gene frequency within a lineage over time.

**Phenotypic accommodation** – phenotypic adjustment in response to a developmental disruption.

**Pleiotropy** – the gene product of a locus having multiple effects on physiology and phenotype.

**Predictive adaptive responses** – the usage of environmental cues by a developing organism to forecast the future environment and to attempt to match its physiology accordingly by modulating its developmental trajectory.

**Reaction norm** – the range of phenotypes that can be induced from a single genotype upon responding to variation in the developmental environment.

**Sexual selection** – a form of selection in which genes underlying a trait that is present in an organism that is sexually preferred – either because of intra-gender conflict or mate choice – become concentrated in the population over time.

**Social selection** – a form of natural selection similar to sexual selection where the preferred characteristics for mate choice reflect eusocial characteristics.

**Trade-off** – a situation where change in one trait benefits the organism (generally by increasing fitness) but concomitantly incurs a cost in another trait.

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