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Evolution, development and timing of puberty

Peter D. Gluckman[1] and Mark A. Hanson[2]

[1]The Liggins Institute and National Research Centre for Growth and Development, University of Auckland, Private Bag 92019, Auckland, New Zealand
[2]Centre for Developmental Origins of Health and Disease, University of Southampton, Southampton, SO16 5YA, UK

The age of menarche has fallen as child health has improved. Although there is ample evidence of delayed puberty being associated with poorer childhood nutrition, menarche is also influenced by prenatal factors. In particular, early onset of puberty is reported in children who have migrated from developing to developed countries. Evolutionary perspectives suggest that these effects can be explained by adaptive mechanisms. They also provide an explanation for the human pubertal growth spurt. In the past few decades, as puberty has advanced, biological maturation has come to precede psychosocial maturation significantly for the first time in our evolutionary history Although this developmental mismatch has considerable societal implications, care has to be taken not to medicalize contemporary early puberty inappropriately.

Introduction

There is considerable public and medical interest in the falling age of menarche [1]. This reflects an increasing awareness of the consequences of the psychosocial 'mismatch' which arises from early biological reproductive competence in societies in which young women do not obtain psychological or social maturity until at least their late teens [2]. Precocious puberty might be a result of identifiable central or peripheral pathology (Box 1). Most cases of central precious puberty are 'idiopathic', and within this group of children are many in which the progression of puberty is unremarkable other than being somewhat earlier in onset.

Particular attention has focused on an apparently greater incidence of early-onset puberty in girls who migrated at a young age from a poor to a developed country [1]. One explanation is that this phenomenon results from greater environmental exposure to developmental endocrine disruptors, such as derivatives of dichloro-diphenyl-trichloroethane, thus accelerating hypothalamic maturation [1]. Such considerations imply a pathological origin. Generally, a medical approach is taken to early menarche. The use of suppressors of gonadotrophin secretion or action has increased. For example, there is an increased use of such agents in association with growth hormone therapy for impaired skeletal growth, although the evidence for auxological efficacy is debatable [3,4]. A more parsimonious explanation for both the secular trend and the observations made in the migrant population is that these are reflections of the underlying evolved life history strategies of *Homo sapiens*.

Here, we review evidence suggesting that the timing of puberty and its other characteristics can be better understood by reference to evolutionary principles. These considerations place early-onset menarche in perspective, challenge the concept that it is necessarily pathological and suggest a need for a greater societal awareness of the biological inevitability of early menarche in the 21st century. Although many similar considerations might apply to males, the reproductive and life history strategies of the two genders are quite distinct and this might be reflected in the more frequent presentation of females with early-onset puberty. Further, the absence of an easily assessable marker of potential reproductive competence means that discussion will focus on the female.

Life history, reproductive competence and puberty

The attainment of reproductive competence is central to the life history of all sexually reproducing organisms. In mammals, this manifests as puberty. Natural selection favours individuals who optimize their capacity to pass their genes to the next generation. Reproductive fitness is enhanced if there is an adaptive match between the life history traits of the organism and the environment in which it has evolved to live. Key traits include the pattern of growth, timing of growth phases, optimal body size and longevity, and determinants of fecundity and reproduction, including the age of reproductive competence and number of progeny [5]. Environmental factors to consider include the physical environment, the interspecific biological environment (e.g. food availability, risk of predation) and the intraspecific environment (e.g. competition for food, reproductive success).

The life history strategy of H. *sapiens* evolved with constraints imposed by two distinct characteristics [6]: the upright posture leading to a distorted pelvic canal, and large brain size at maturity. Thus, in contrast to other primates, *H. sapiens* is secondarily altricial, being born relatively immature, and much development of the central nervous system is postnatal. Even after weaning, the

Corresponding author: Gluckman, P.D. (pd.gluckman@auckland.ac.nz).
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Box 1. Precocious and early-onset puberty

Sexual precocity is generally defined by the appearance of secondary sexual characteristics in females before eight years of age and in males before nine years of age. These ages are set by clinical convention, based on the 95% confidence limits from somewhat dated epidemiological studies [54]. Generally, pubertal development starts with breast development in girls and genital growth and pubic hair development in boys. Puberty is considered to be abnormal if the consonance of development is lost or if the events of puberty occur unusually early or late.

Precocious puberty might occur as a result of premature activation of the hypothalamic–pituitary–gonadal axis (termed *central precocious puberty*) or more rarely because of inappropriate androgen or oestrogen exposure – for example as a result of autonomous hormone production by the gonad. Most cases of central precocious puberty are reported in females, the majority of which have no identifiable cause (termed *idiopathic central precocious puberty)* and a minority are due to a variety of cerebral lesions.

Premature thelarche refers to isolated early-onset breast development and is generally benign, although it has been reported to be associated with exposure to oestrogenic environmental pollutants [1].

The major clinical concerns related to premature puberty are the psychosocial consequences of early maturation in relation to peers and reduced final height due to earlier fusion of the epiphyses.

There is a gradation between severe disturbance to the timing of the onset of puberty and mildly early but otherwise normal puberty, where 'early' is defined in terms of historical norms in a sentinel population. We suggest the use of descriptors such as *precocious* and *early-onset* to distinguish between these.

human infant is not self-sufficient and is dependent on its parents for nutrition and protection for several years. This extended childhood period is unique to humans [6]. Furthermore, after a juvenile period in common with other primates, humans undergo a unique pubertal growth spurt [7] before, in females, attaining reproductive competence.

Genetic influences on the timing of puberty

About half the variance in the timing of menarche is genetically determined [8], and there is evidence for a dominant inheritance pattern [9]. Several polymorphisms including shorter TAAAA repeats in the promoter region of the gene for the sex hormone binding globulin [10], and single nucleotide polymorphisms in CYP17 [11,12] and CYP3A4 [13] have been implicated. Evidence for genetic effects on the timing of puberty can also be inferred from the secular trend in the age of menarche. Northern Europeans to have later menarche than those from southern Europe but both have shown similar and parallel reductions in the age of menarche over recent decades [1,14,17].

Environmental influences on the timing of menarche

Life history theory suggests that nutritional influences, both in early development and in childhood, would have significant effects on the timing of puberty [18]. In epidemiological studies, a common finding is a trend towards earlier menarche in those of lower birth weight or low body mass [19–23]. As the timing of adrenarche appears to be similarly influenced [24], it suggests a general effect on maturation rather than a specific effect on the hypothalamic gonadostat [25]. This prenatal influence must be contrasted with the large body of literature relating poor childhood weight gain to delayed puberty [1]. Some studies demonstrate these to be independent effects [22].

Prenatal influences

Recent advances in our understanding of developmental plasticity and life history biology provide one explanation of why puberty might be advanced in those with evidence of an impaired intrauterine environment as reflected in birth size. The developing organism can respond to environmental cues with two potential classes of adaptive responses [26], both of which can influence the timing of menarche. These provide either immediate or delayed advantage. An immediate response to nutritional imbalance might permit immediate survival but nonetheless leave the organism to cope with the consequences; indeed, there is often a trade-off between an early adaptive response and longer-term effects that can reduce fitness. Many trade-offs in biology involve the interaction between growth and maturation. Early metamorphosis from a tadpole to a toad might be an immediately adaptive response to overcrowding (e.g. the pond drying up) but toads undergoing earlier metamorphosis are smaller and at greater risk of predation and are less competitive as mates [27]. Similarly, in mammals, intrauterine growth retardation can be the result of an immediately adaptive response to limited nutrition, which then has consequences for neonatal and infant survival [28].

Moreover, the developing organism can also make a delayed response to an environmental cue with the induction of an adaptive trait that becomes manifest later in the life cycle. This is advantageous if the developing organism has 'predicted' its future environment correctly [26,29]. The embryo or foetus senses its nutritional environment, 'predicts' the same after maturity and adjusts its developmental trajectory accordingly [30]. These processes, underpinned, at least in part, by epigenetic change [31], have the effect of shifting the settings for the control of metabolic homeostasis such that if the foetus 'predicts' postnatal deprivation, it sets its metabolic homeostasis for such an environment. Metabolic compromise and disease ensue if the 'prediction' is inaccurate – for example, if the postnatal environment is much richer than anticipated. Such predictive adaptive responses have been used to explain the relationship between a suboptimal foetal environment and the later risk of metabolic disease [30–32].

Life history theory suggests that if the individual 'predicts' a deprived future environment, then several components of its life course strategy will be adjusted in a coordinated manner [18]. Provided that postnatal energetics enable successful reproduction, protection of intergenerational gene transmission in a poor environment might be enhanced by earlier maturation, with less investment in growth, development and repair. Such strategies are demonstrable in the trade-off between

early growth, as influenced by maternal nutrition, and the age of maturation seen in many species, including insects [5,33]. This strategy is most clearly evolved in species which produce many offspring but invest little in each, and whose offspring mature quickly and have short lives. Gene transmission is maintained by a small fraction of a large number of offspring surviving. We suggest that echoes of such a shift in life-history strategy might underpin the relationship between low birth weight and age at menarche.

Postnatal influences

Humans are a species that matures slowly, lives a long time and has a high parental investment in a few progeny, of which each has a high probability of surviving. Such a strategy requires high sensitivity to a potentially variable postnatal environment. A period of postnatal nutritional deprivation in such an organism close to sexual maturity is likely to delay reproductive competence in the hope that the environment will subsequently improve. This strategy evolved because pregnancy incurs a high energetic cost; hence the trade-off between longevity and fecundity demonstrable in the human female [34]. This provides an evolutionary basis for the strong epidemiological evidence that poor weight gain in childhood is associated with delayed menarche, and more rapid weight gain with earlier puberty [1,14,35,36]. There is increasing evidence of a close interaction between neuroendocrine systems controlling gonadotrophin release and those controlling metabolic regulation, including the hypothalamic leptinergic pathway [35–37]. We anticipate that the postnatal effect would predominate over prenatal effects in humans, and this indeed appears to be the case [22].

The interaction between prenatal and postnatal influences

These two developmental effects become manifest most dramatically in the combination of prenatal early life deprivation with childhood nutritional excess, as seen in adopted children migrated from poor to rich countries [1,38]. Both environmental effects are predicted to advance menarche. We anticipate that the prenatal effect would be stronger in those children born small who are in a nutritionally enriched postnatal environment, as reflected in good weight gain in childhood, and indeed this is what is observed – the prenatal effect on age of menarche is much stronger in females in the highest quintile of childhood weight gain [22]. There might be a further interaction: animal [39] and human neonates [38] are more likely to develop childhood obesity because predictive adaptive responses induce both central and peripheral drivers to obesity [31]. These processes together offer an explanation of earlier puberty associated with migration.

Given the close interaction between energetics and reproduction [17], we anticipate that those environmental factors that determine later metabolic homeostasis would interface with those influencing the timing of sexual maturation. Using the epidemiological data referred to above, we have demonstrated (Figure 1) the suggested relationship between the foetal and postnatal nutritional effects and the timing of menarche. Although we have focused on the influence of nutrition, access to food and stress caused by predation and competition are ecologically linked. In studies of developmental induction of metabolic control, both nutritional and glucocorticoid manipulation have comparable effects and might act through common pathways [30,31]. There is evidence that postnatal stress can affect pubertal timing [1], presumably through comparable processes.

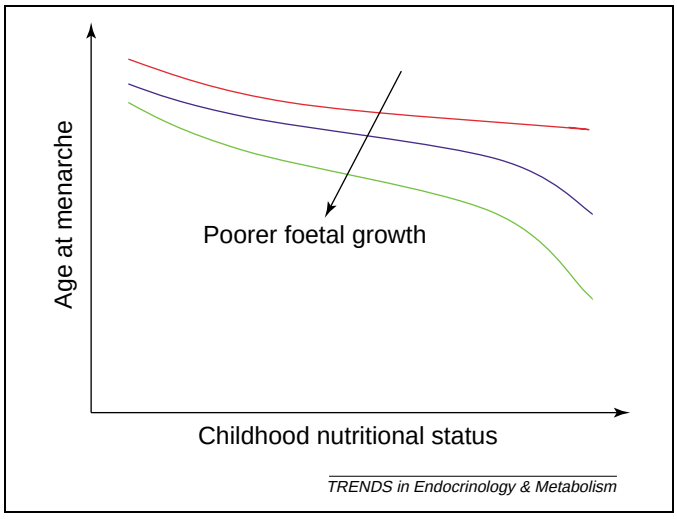

Figure 1. The relationship between childhood nutrition and the age of menarche is shown for children of different birth sizes as a series of contour lines. There is an inverse relationship between the age of menarche and childhood nutritional status but being born smaller also advances menarche. The prenatal influence is more marked at higher postnatal nutritional levels.

The secular trend in menarche and the mismatch produced

There is a consensus that in developed countries there has been a marked secular trend for a reduction in the age of menarche over the past 100 years [1,14–17]. In populations of Northern European origin, the age of menarche is approximately three years earlier than it was 100 years ago [1,40]. Similar but less dramatic trends can be observed in other populations. This trend is generally interpreted as being a reflection of improved nutrition and reduced infection in childhood over the past two centuries. Indeed, life-history modelling predicts that a reduction in juvenile mortality will reduce the age of menarche [5].

In a stable environment, evolutionary pressures operate to select traits that match the organism to its environment. It seems probable that the timing of reproductive competence would be linked to social maturation in most mammalian species, including *H. sapiens*. Thus, the timing of puberty evolved to match the age of reproductive competence to the social ecology of the evolving human. In Paleolithic times, it is a reasonable presumption that human females reached sexual maturity at about the age when they were psychosocially mature: this is the case in modern hunter-gatherer societies [41]. This synchrony would have been selected because reproduction is energetically costly and carries a risk for the female, and because it would be disadvantageous for reproductive competence to precede the capacity to function adequately as an adult and as a mother.

Although there are obvious limitations to interpretations obtained from skeletal remains, we estimate that menarche would have occurred between the ages of 7–13 years in Paleolithic times. Several grounds exist for this estimate: chimpanzees progress through puberty at 6–9 years of age [42], and the major auxological difference between them and humans is the 3–4 year delay in maturation (as, for example, indicated by the age of molar eruption) owing to our additional childhood growth phase [6]. Using estimates of life expectancy at birth for Neolithic humans [43], corrected for likelihood of death in pregnancy, the risks of death in childhood and the likely interbirth interval, and assuming 1% population growth per generation, we arrive at estimates of full reproductive competence in Neolithic females of 9–14 years of age. This would place menarche at 7–13 years, assuming a 1–2 year gap between menarche and reproductive competence, and suggests that menarche in Neolithic times could have been in the range now being observed in developed nations.

We propose that human females evolved to enter puberty at a relatively young age and progressed to reproductive competence at 11–13 years of age. This would have matched the degree of psychosocial maturation necessary to function as an adult in Paleolithic society based on small groups of hunter-gatherers. With settlement, childhood disease and postnatal undernutrition became common [44,45] and therefore the average age of menarche was delayed. This matched the increasing complexity of society following the development of agriculture, settlement and population aggregation, which in turn led to the differentiation of social tasks and the creation of societal hierarchies. Roman law assumed that females matured by the age of 12 [46]. As hygiene deteriorated with increasing population density, the age of menarche was delayed, particularly in Europe. However, once again, this delay was matched to the increased complexity of medieval society, and even more so in the industrial revolution from the 18th century. With modern hygiene, nutrition and medicine, these pathological constraints on puberty have been removed and the age of menarche has fallen to its evolutionarily determined range. But now the complexity of society has increased enormously and psychosocial maturation takes longer. Magnetic resonance imaging studies show that brain maturation extends into the third decade [47]. For the first time in our evolutionary history, biological puberty in females significantly precedes, rather than being matched to, the age of successful functioning as an adult (Figure 2). This mismatch between the age of biological and psychosocial maturation constitutes a fundamental issue for modern society. Our social structures have been developed in the expectation of longer childhood, prolonged education and training, and later reproductive competence. This emerging mismatch creates fundamental pressures on contemporary adolescents and on how they live in society.

The pubertal growth spurt

Another unique feature of humans is the pubertal growth spurt, there being no evidence of such spurts in skeletal

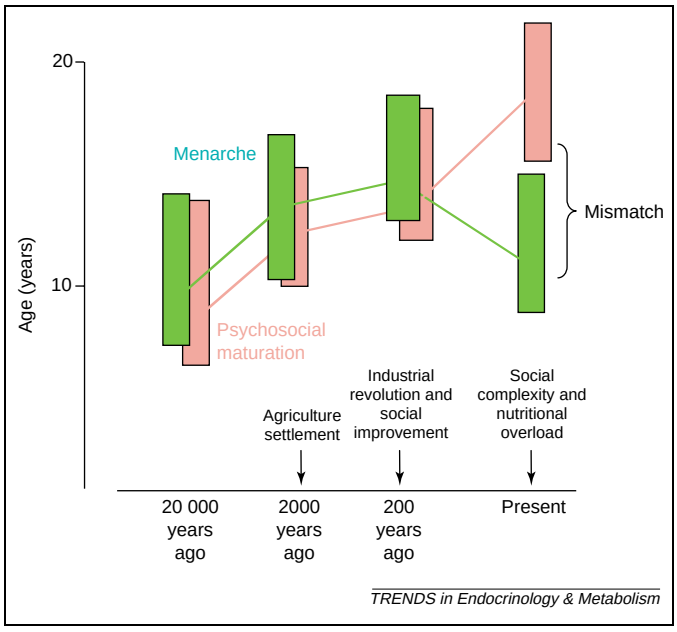

Figure 2. . The relationship between the likely range of ages of menarche (green) and achievement of psychosocial maturity (pink) from 20 000 years ago to the present day. The mismatch in timing between these two processes is a novel phenomenon.

dimensions in other primates. Most mammals undergo puberty as their postnatal growth is tailing off [42]. In humans, reproductive competence is achieved early in the growth spurt in males and at a later stage in females [25] but in both genders it is underpinned by oestradiol. Although there is a small amount of linear growth after menarche, most cycles in the first year after menarche are anovulatory [48] and the interspinal pelvic dimensions only reach maximal size several years after menarche [49,50].

The pubertal growth spurt appeared late in hominin evolution [42] – ~500 000 years ago, in the same era that there was more rapid expansion of brain size relative to body size [51]. The most widely proposed explanation for the pubertal growth spurt is a life-history trade-off: to protect brain development in the human, investment in somatic growth is delayed until brain growth is essentially complete [42].

An alternative explanation is based partially on natural and partially on sexual, selection. Although many aspects of pelvic dimensions have been selected for nonobstetrical reasons, including locomotor [52], there is particularly close matching between foetal head size and the pelvic canal dimensions in humans relative to other apes [49]. Pelvic inlet size and height are closely correlated [50] and thus taller women will be more likely to give birth to larger babies, or babies with larger heads. The latter was important for the evolution of the large-brained ape, *H. sapiens*; the former to maternal and infant survival. Thus, there would have been positive selection in the upright hominid for taller females, who would have been more likely to survive childbirth and to produce offspring with bigger brains. However, as Darwin first suggested [53], most secondary sexual characteristics arise from sexual selection and this is a possible origin of the pubertal growth spurt, albeit for gender-specific

reasons. A taller male might be positively selected, both by being dominant and appearing stronger, and a taller female might similarly have been selected based on taller women being regarded as more reproductively healthy. It might be that processes such as these contributed to the origin of the human pubertal growth spurt.

Final comments

We have suggested that an evolutionary perspective is useful in understanding various components of contemporary human puberty. This perspective argues for more careful use of the term 'precocious puberty'. This term implies pathology and, although there are important organic causes of central and gonadal precocious puberty and onset of puberty at a particularly young age is clearly pathological, the vast majority of young women undergoing menarche at increasingly younger ages have normal physiology and progression of puberty; their physiology has been simply determined by their distant ancestors.

However, the mismatch between early biological puberty and later psychosocial maturation is a recent phenomenon. It has only appeared in the past 100 years of our 200 000 year history as a species. Our social structures are largely based on the belief that biological puberty is matched to psychosocial maturation. This is no longer the case, and might never be so again. The challenge is for society to adjust its structures to this biology; adjusting the biology to society is impracticable, unethical and potentially dangerous.

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