

REVIEW

Puberty as a life history transition

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Background: James Tanner's landmark publication, *Growth at Adolescence*, was not only the first and most comprehensive treatise on the subject of human pubertal development of its time, its core insights have held up remarkably well over time.

Review: This review connects Tanner's contributions to contemporary understanding of puberty as a process fundamentally driven by neuroendocrine maturation. It introduces the concepts of the 'hour-glass of puberty' and 'somatic strategy' as heuristic constructs. The 'hour-glass of puberty' describes the converging pathways of information flow influencing the timing of the neuroendocrine events of puberty and its ramifying consequences throughout the body. Somatic strategy refers to the pattern of sex-specific, adult body morphology that develops at puberty as the individual undergoes a life history transition from juvenile to adult.

Keywords: Puberty, adolescence, growth

GROWTH AT ADOLESCENCE

Growth at Adolescence, first published 2 years after Tanner received his PhD (Tanner 1955) and then revised in 1962 (Tanner 1962), probably remains Tanner's most important and enduring work. Several of the images and figures that appear in that work have become nearly synonymous with Tanner's name. 'Tanner stages' of pubertal development are still used around the world to assess pubertal development in boys and girls. The graph of the secular decline in menarcheal age is equally famous, even if some of the early data have now been revised. Also, the graphs depicting pubertal growth spurts in non-human primates continue to fuel current academic debates (Bogin and Smith 1996; Hamada and Udono 2002; Gluckman and Hanson 2006; Bogin 2009). Each of these iconic images derives from intellectual contributions original to Tanner, but they all point to the central theme that is woven through the text of *Growth at Adolescence*: the synchrony between physical

growth and reproductive maturation at the threshold of adulthood.

The first chapter of *Growth at Adolescence* is given to a documentation of the adolescent growth spurt, a phenomenon that, as Tanner points out, is not merely a matter of growth in height. 'Every muscular and skeletal dimension of the body seems to take part in the adolescent growth spurt' (Tanner 1962:10). Muscles like the heart undergo a spurt. Bones in the face, jaw, cranium and pelvis all undergo spurts that Tanner chronicles.

However, at the same time that this general acceleration and deceleration in tissue growth occurs, the reproductive system matures, which is the message of Chapter 2 of *Growth at Adolescence*. 'The adolescent spurt in skeletal and muscular dimensions is closely related to the spectacular development of the reproductive system which takes place at that time' (Tanner 1962:28). In this chapter we find a thorough documentation of the growth of the internal reproductive organs—ovaries, uterus, testes, prostate—as well as a presentation of the now famous 'Tanner stages' of breast, genital and pubic hair development. However, also in this chapter are presented data on the close correlation in timing between the growth spurt in height and the stages of pubertal maturation: correlations in the range of 0.84–0.89 between the beginning and end of the growth spurts in height, penis length and testis volume; a correlation of 0.93 between age at menarche and age at peak height velocity.

The causes of the synchrony between adolescent growth and reproductive maturation, Tanner argues, lie in hormonal mechanisms. At the time *Growth at Adolescence* was published, endocrinology was itself an immature field, just at the threshold its own growth spurt brought on by the development of radioimmunoassay techniques by Berson and Yalow (1961). In the data available to Tanner, sex steroids were still measured with bioassays or metabolites were measured in urine with chemical assays of low sensitivity and specificity. Hypothalamic peptides were

unknown. Growth hormone was known, but insulin-like growth factors (or somatomedins as they were first known to Tanner) were not. Yet, despite these limitations, Tanner was able to infer a remarkably accurate picture of the endocrinology of adolescence, including the key role of sex steroids in the acceleration of linear growth and eventual closure of the epiphyses and the simultaneous development of secondary sexual characteristics. The upstream causes of reproductive maturation were wrapped in mystery, yet Tanner was able to conclude that there was 'very convincing evidence that it is the hypothalamus, and not the pituitary, which has to mature before adolescence begins' (Tanner 1962:197).

During his lifetime Tanner saw many of the gaps in our knowledge surrounding pubertal growth and maturation filled in, occasionally requiring him to revise his views, but more often confirming them even as additional detail was uncovered. In this paper we attempt to provide a brief summary of current understanding and then use that understanding to return to one area of controversy with roots in *Growth at Adolescence*. However, we would also like to introduce a framing concept unavailable to Tanner in 1962, but one that we believe aligns well with his own: puberty as a life history transition.

LIFE HISTORY TRANSITIONS

Life history theory or the theory of the evolution of mortality and fertility schedules, was in its infancy at the time that *Growth at Adolescence* appeared. It, too, began a growth spurt of sorts with the publication in 1970 of Gadgil and Bossert's (1970) influential paper, 'Life historical consequences of natural selection'. In this paper, the authors introduce the model of an organism 'choosing' how to allocate energy and other scarce resources among competing physiological domains. They identify growth, maintenance and reproduction as three major allocation alternatives. This simple model remains a dominant framework in the field today. Life history stages are recognized by dominant patterns of energy allocation. For example, in mammals, infancy is the stage in which energy inputs come primarily from maternal milk, juvenility is typified by investment in growth and maintenance and adulthood is typified by investment in maintenance and reproduction. Charnov and Berrigan (1993) have noted that, across mammalian taxa, there is a close equivalence between the rate of energy investment in growth during the juvenile period and investment in reproduction in the adult period. Puberty marks the transition between these two life history stages, the period when growth is completed and reproductive function initiated, a critical 'switching point' in the pattern of investment of metabolic energy.

The transition from juvenile to adult involves changes in anatomy and behaviour, as well as changes in physiology. The adult mammalian soma must be prepared to take on new roles and challenges, to gestate, lactate and give birth in females, to attract mates and contend with intra-sexual competition in males. In humans there is also a transition to

adult responsibilities in many realms. In formative human history, the assumption of an adult role in foraging was likely a part of the transition. Initiation into adult roles remains an important part of pubertal rituals in many societies.

Although Tanner did not use the framework of evolutionary ecology and life history theory to organize his thinking or his description of adolescence, recognizing puberty as the life history transition between juvenility and adulthood in terms of both somatic morphology and reproductive potential summarizes his key insights and contributions quite nicely.

THE 'HOUR-GLASS' OF PUBERTY

The phenomenology of pubertal growth and maturation remains very true to Tanner's description and, as noted, 'Tanner stages' are still used in the clinical assessment of pubertal progression. Our understanding of the endocrine mechanisms underlying the pubertal transition has advanced a great deal, however, since *Growth at Adolescence* appeared. Not only do we understand much more about the onset of reproductive maturation, we understand more clearly the linkages between reproductive maturation and physical growth. Current understanding of puberty can be summarized in an hour-glass shaped diagram such as that presented in Figure 1.

At the centre of the hour-glass is the hypothalamic-pituitary-gonadal (HPG) endocrine axis, the axis that controls adult reproductive physiology. The upper end of this axis is constituted by ~ 2000 neurons dispersed in the mediobasal hypothalamus capable of secreting the decapeptide, gonadotropin-releasing hormone or GnRH (Ebling and Cronin 2000). Elegant work in the 1980s (Knobil et al. 1980; Wildt et al. 1981; Knobil 1992) showed that pulsatile release of GnRH from the median eminence of the hypothalamus into the tiny vascular system connecting to the anterior pituitary at a rate of about once every 90 minutes was both necessary and sufficient to stimulate adult patterns of gonadotropin secretion from the pituitary in rhesus monkeys, which would in turn stimulate adult level responses from the gonads. Although similar experiments could not be performed in humans, successful interventions to treat premature or delayed puberty were developed for humans based on exogenously providing or disrupting pulsatile patterns of GnRH to the pituitary (Crowley and Jameson 1992). Perhaps the most compelling evidence comes from the treatment of patients with Kallman's syndrome, in which the GnRH neurons fail to migrate into the hypothalamus during embryogenesis and no spontaneous puberty occurs. By providing a regular, 90-minute pulse of GnRH through an exogenous pump system, these patients can be induced to enter and complete puberty (Crowley and McArthur 1980; Crowley and Whitcomb 1990; Seminara et al. 1998; Balasubramanian et al. 2010).

As inferred by Tanner, the pubertal transition begins in the hypothalamus with the appearance of a pulsatile pattern of GnRH secretion (Plant and Barker-Gibb 2004). This is

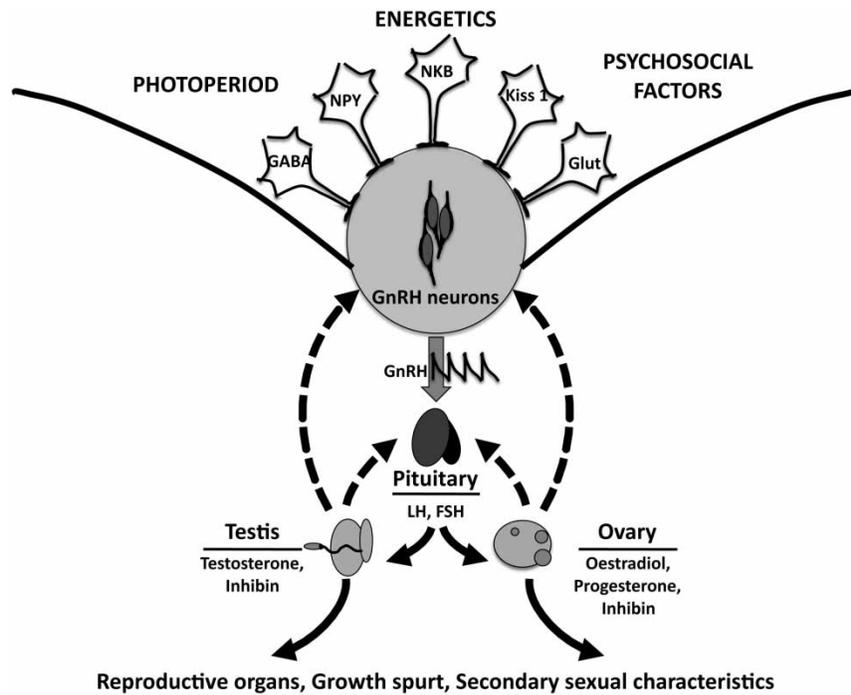


Figure 1. The hour-glass of puberty. Reproductive function in both sexes is controlled by the hypothalamic-pituitary-gonadal (HPG) axis. This axis begins a transformation to mature function at the onset of puberty with the re-emergence of pulsatile gonadotropin-releasing hormone (GnRH) secretion by a small population of hypothalamic neurons. The timing of this re-emergence is modulated by upstream inputs to the GnRH neurons. Direct modulation includes inhibiting signals from gamma aminobutyric acid (GABA) secreting neurons and neuropeptide Y (NPY) secreting neurons, as well as stimulating signals from glutamine (Glut), kisspeptin (Kiss 1) and neurokinin B (NKB) secreting neurons. These signals in turn are affected by more distal signals related to environmental inputs relating to energy availability, photoperiod (in many mammals) and psychosocial stressors. The output of the HPG axis is gamete and steroid production. Steroids have wide effects on the body, including the transformation of the immature soma into that of an adult and the development of a sex-specific somatic strategy. Thus, a broad range of factors follow converging pathways to influence the timing of puberty and the onset of mature HPG activity, while the consequences of that activity have broad, co-ordinated effects on the soma.

now recognized to be a resumption of pulsatile secretion, a pattern that also occurs during foetal life and immediately after birth (Terasawa and Fernandez 2001; DiVall and Radovick 2008). GnRH neurons from animal models will even secrete GnRH in a pulsatile pattern *in vitro* (Ronnekleiv and Resko 1990; Wetsel et al. 1992). For reasons that are not well understood, pulsatile GnRH secretion subsides over the first post-natal months in humans and then is reinitiated at puberty. The resumption of pulsatile GnRH secretion occurs spontaneously in gonadectomized rhesus monkeys and humans suffering from gonadal dysgenesis, so changes in gonadal feedback are not involved (Conte et al. 1975; Plant 1985; Pohl et al. 1995). Once this 'on/off switch' for the HPG has been thrown, however, the rest of the axis begins to function. Increasing circulating levels of the gonadotropins, luteinizing hormone (LH) and follicle-stimulating hormone (FSH), stimulate both the growth of the gonads to their mature size and the progressive maturation of gonadal function in the production of both gametes and hormones. Some of the gonadal hormones, such as inhibin, regulate the HPG axis itself. However, the major gonadal steroid hormones, the androgens, oestrogens and progestagens secreted by testes and ovaries, have far-reaching effects on anatomy, physiology and behaviour. (The physiology of the HPG axis is reviewed in more detail in many sources, including Ellison (2001).) So we can think of the neck of the

hour-glass as constituted by the HPG axis, with pulsatile GnRH secretion at the top and sex steroid secretion at the bottom.

PROXIMATE UPSTREAM REGULATORS

The small population of GnRH-secreting neurons integrates a wide variety of upstream neuronal and neuromodulatory inputs (Plant and Barker-Gibb 2004). Some of the signals that converge on these neurons are transmitted by rather generic inhibitory (e.g. gamma-aminobutyric acid or GABA) and stimulatory (e.g. glutamate) mediators. However, two recently identified neuropeptides, kisspeptin and neurokinin B, appear to have special roles in the control of GnRH secretion (Castellano et al. 2006; Plant 2006; Topaloglu 2010; Navarro et al. 2011).

Kisspeptin refers to a family of closely-related peptides coded by the *KISS1* gene that have variable affinity for the kisspeptin receptor (Oakley et al. 2009). Inactivating mutations in the *KISS1* gene or the gene for the kisspeptin receptor, *GPR54*, are associated with idiopathic hypogonadotropic hypogonadism (IHH, abnormally low gonadotropin and gonadal steroid levels) and failure to undergo spontaneous puberty in both humans and animal models (Oakley et al. 2009). In animal models, including both rodents and primates, *KISS1* transcriptional activity

significantly increases just before puberty and *GPR54* expression has been localized on GnRH secreting neurons (Gottsch et al. 2004; Ramaswamy et al. 2008). *KISS1* and *GPR54* knockout models have demonstrated that kisspeptin signalling is necessary for the normal pubertal activation of pulsatile GnRH secretion in animal models, while centrally administered kisspeptin has been shown to acutely stimulate pulsatile GnRH secretion (Kauffman et al. 2007).

Neurokinin B (NKB), a distinct peptide that is co-secreted by some kisspeptin secreting neurons, also appears to provide a necessary signal for normal puberty. Mutations in the gene coding for the peptide (*TAC3*) and its receptor (*TAC3R*) have been associated with IHH in humans and NKB neuronal projections have been traced to GnRH neurons in ultrastructure studies (Topaloglu 2010).

Kisspeptin and NKB neurons in the arcuate nucleus of the hypothalamus express oestrogen receptors and their activity appears to be subject to steroid feedback. Kisspeptin neurons are sexually dimorphic in their abundance in the hypothalamus of rodent models and it has been proposed that the organizing effects that steroids have on the brain early in development may involve organizing effects on kisspeptin and NKB neurons (Navarro et al. 2011).

Neuropeptide Y (NPY) is another neuropeptide that may help to regulate GnRH secretion. Its effects are primarily inhibitory. Central administration of NPY inhibits pulsatile GnRH secretion in gonadectomized rhesus monkeys. However, it is unclear whether NPY acts directly on GnRH neurons or through the mediation of other signalling pathways (Shahab et al. 2003; Pralong 2010).

FURTHER UPSTREAM

Upstream of the peptides and neuromodulators that have one- or two-step connections to the hypothalamic GnRH neurons are numerous inputs from a variety of sources and modalities that affect the timing of puberty in humans or animal models. Prominent among these are energetic factors and psychosocial stress.

The influence of energetics on the timing of human puberty was first explicitly proposed by Frisch and various colleagues in the 1970s. A series of related hypotheses were proffered linking the attainment of either an average or a minimum weight or percentage body fat to the age of menarche in girls (Frisch and Revelle 1970, 1971; Frisch and McArthur 1974). Although heavily and often justifiably criticized (Johnston et al. 1971; Billewicz et al. 1976; Trussell 1978; Ellison 1982), the Frisch hypotheses also explicitly incorporated an evolutionary argument, suggesting that attempts at reproduction in an under-nourished state would lower, rather than increase, individual fitness and that the linkage between energy status and ovarian function had evolved to reduce the likelihood of pregnancies with low chance of success. One difficulty with the Frisch hypotheses is that an acceleration in adolescent fat gain in girls is a consequence of pubertal maturation, not an antecedent, a consequence stimulated in part by rising levels of oestrogen from the ovary.

A clearer relationship can be drawn between the pace of childhood growth and the timing of puberty. Within populations, children of both sexes who are taller and heavier in middle childhood are more likely to enter puberty earlier. Also, the secular trend toward faster childhood growth through time has been associated with a simultaneous trend toward earlier puberty in both sexes (Ellison 1982; Eveleth and Tanner 1991; Cole 2000; Karlberg 2002). It is important to note, however, that the relationship between adiposity and gonadal function is actually U-shaped in both sexes, with obesity associated with gonadal dysfunction. In girls, dysfunction can arise due to excessive hypothalamic feedback from adrenal steroids and extragonadal oestrogens produced in fat tissue and from excessive insulin stimulation of ovarian androgen production. In boys, obesity can lead to excessive production of extragonadal oestrogen in adipose tissue and sex-hormone-binding globulin in the liver (Nelson and Fleming 2007; Zain and Norman 2008; Eskandar et al. 2012).

There are a number of converging pathways by which information on energy balance, energy expenditure and energy status may reach the hypothalamus. Levels of circulating oxidizable substrates, such as glucose and ketone bodies, may be directly monitored by the hypothalamus (Kinoshita et al. 2003). Circulating insulin levels, which correlate with positive energy balance and low energy expenditure, may also be registered by the hypothalamus (Strack et al. 1995; Oppert et al. 1997; Obici and Rossetti 2003). Insulin receptor is expressed in the mouse hypothalamus and in immortalized GnRH neuron cell lines (Bruning et al. 2000; Salvi et al. 2006). Suppression of insulin receptor expression in the mouse leads to a reduction in GnRH pulsatility (Bruning et al. 2000).

Leptin, secreted by adipose cells and stimulated by insulin, also carries information about energy status and energy balance (Rosenbaum et al. 1997; Campostano et al. 1998; Wadden et al. 1998; Hilton and Loucks 2000; Franks et al. 2003). Although leptin receptor is not expressed by GnRH-secreting neurons (Burcelin et al. 2003), it is expressed by kisspeptin-secreting neurons, among others, and so may influence GnRH neurons indirectly (Castellano et al. 2006; Oakley et al. 2009). In animal models exogenous leptin administration can advance reproductive maturation (Ahima et al. 1997). Evidence in humans and non-human primates is less convincing, however (Plant and Durrant 1997; Rogol 1998; Andreelli et al. 2000). Additionally, as with adipose tissue itself, increases in circulating leptin levels follow, rather than precede, pubertal maturation, especially in girls where oestrogen increases the output of leptin relative to fat mass (Kennedy et al. 1997; Rogol 1998; Rosenbaum and Leibel 1999).

Psychosocial stress appears to affect pubertal timing in humans, although the effect is complex. For example, early studies documented later puberty in children institutionalized in orphanages compared to non-institutionalized peers, while more recent studies have associated similar histories of institutionalization with early puberty (Boas 1940; Charmandari et al. 2003; Teilmann et al. 2006; Wise

et al. 2009). One reason for the difference may be that the more recent studies focus on children who changed environment from an institutionalized to a family situation, while Boas' subjects remained institutionalized through childhood.

A number of studies (but not all) of girls raised in 'father absent' conditions find that this condition is related to earlier menarcheal age (Belsky et al. 1991; Grainger 2004; Maestripieri et al. 2004; Bogaert 2008; Tither and Ellis 2008). The effect size in these studies is often small, especially when controlling for genetic factors. The causal pathways are also not well understood, although some effect on the hypothalamic-pituitary-adrenal axis and its interaction with the HPG axis is usually assumed (Abbott et al. 1997; Gotz et al. 2008). Some researchers appeal to the evolutionary logic of life history theory, noting that increases in exogenous, adult mortality rates should select for earlier maturation (Belsky et al. 1991; Hoier 2002; Coall and Chisholm 2003; Chisholm et al. 2005). Developmental plasticity in response to cues regarding adult mortality, it is argued, might have evolved to adjust developmental timing in similarly adaptive ways.

Energetic and psychosocial factors reflect more complex webs of causality that include subsistence and disease ecology, social organization and kinship and the broader influence of culture. All of these factors can be thought of as feeding into a progressively converging set of pathways, ultimately neural pathways, ultimately impinging on the GnRH secreting neurons of the hypothalamus.

DOWNSTREAM CONSEQUENCES

As noted above, the major outputs of the HPG axis, in addition to gametes, are steroid hormones. These steroids act within the axis itself, to provide regulatory feedback to the pituitary and hypothalamus and positive drive to gamete maturation within the gonad. However, they also circulate throughout the body, exerting powerful effects on many tissues.

Among the targets of steroid action at puberty are the external reproductive organs. Genitals grow and pubic hair develops in the groin in both sexes and breasts grow and develop in females under the influence of increasing titers of gonadal steroids in the blood. These visible changes are, of course, the classic basis of the Tanner ratings of pubertal development. However, they are, in a stricter sense, reflections of steroid exposure. Early development of genitals, pubic hair and breasts can occur due to pathological steroid exposure, as in adrenal hyperplasia or due to high levels of aromatization of normal adrenal androgen levels, as in childhood obesity (Himes 2006; Viswanathan and Eugster 2009; Oberfield et al. 2011; Wagner et al. 2012). In these cases they do not represent true puberty, since the resumption of pulsatile GnRH secretion may not be involved.

There are many pubertal changes outside the reproductive system, *sensu strictu*, that are driven by changing steroid profiles. Some of these affect libido and behaviour,

influencing what is referred to as the *behavioural reproductive strategy* of each sex, a topic we will not pursue further here (see Daly and Wilson 1983; Cronk 1991; Voland 1998 for examples of this literature). Many other pubertal changes contribute to the transformation of the soma into adult form and most also lead to the emergence or enhancement of somatic sexual dimorphism. Each sex may be said to pursue a *somatic strategy* that will enhance individual fitness. These somatic strategies involve differential accumulation of body mass leading to dimorphism in strength and body composition, differential skeletal growth leading to a wider birth canal in the female pelvis and broader shoulders relative to hips in males and greater stature in both sexes. Other changes in craniofacial robustness, subcutaneous fat, axial and facial hair and apocrine secretions can all be understood as resulting from sexual selection acting on somatic strategies and are also induced by gonadal steroid exposure. Tanner (1962) covers all of these aspects of somatic strategy and their emergence at puberty in *Growth and Adolescence*.

SOMATIC STRATEGIES AND PRIMATE PUBERTY

We have seen that modern research has largely supported the view of pubertal growth and development put forth by Tanner in his seminal *Growth at Adolescence*. However, the final chapter of that remarkable book puts forth a thesis that has proven more controversial, particularly in recent years (Bogin and Smith 1996; Hamada et al. 1996b; Leigh 1996; Gluckman and Hanson 2006; Bogin 2009). This thesis is also Tanner's only explicitly evolutionary claim in the book, although he is appropriately cautious in making it: 'It seems that the adolescent spurt proper is an evolutionary step taken by the primates' (Tanner 1962:236). In making this claim, Tanner notes:

Other animals manage their reproductive affairs differently, and with considerably more dispatch. This is a very sweeping statement; exceptions may well exist, and the data are anyway too flimsy to support such a generalization with certitude. Nevertheless the evidence, briefly reviewed in this chapter, certainly points in this direction (p. 223).

Tanner marshals the evidence available to demonstrate that 'other animals', principally mice, rats, rabbits, sheep and cattle, demonstrate what some have called 'one-cycle growth'; that is, growth rates (usually measured in mass gain per unit time) that accelerate from birth until puberty or just before and then subside to near zero. This contrasts with the 'two-cycle' pattern of primates, with growth rates (also usually measured in mass gain per unit time) that decelerate from soon after birth and then accelerate again before puberty, subsiding to near zero after (Hamada and Udono 2002).

Tanner also notes that primate growth spurts are often associated with the development of pronounced sexual dimorphism and what we have termed somatic strategy. Regarding rhesus macaques he writes:

Though the sex difference in weight spurt is bigger in this species than in man, the trunk length spurt in both sexes is relatively smaller. No more detailed measurements are yet available on Rhesus, but this seems to argue that the male spurt is largely one of shoulder breadth and, above all, of muscle mass. The muscles evidently develop more at puberty in the male Rhesus than they do in the male human. From an evolutionary point of view the differences in type of secondary sexual characters developing at adolescence are of much interest, but data bearing upon them are still very scarce. Even within the primate group there is great variability; the weight of the adult female compared with the male, which in man is about 85% and in the chimpanzee 90%, is as low as 50% in the gorilla and orang-utan and as high as 100% in the spider monkey, gibbon and marmoset (Schultz 1960). It seems probable, though not certain, that most of this variability depends on species differences in the development of secondary sex characters at adolescence. Sex differences in the lengths of the limb bones have also been documented by Schultz (1937) though again it is not clear to what extent they arise at adolescence (Tanner 1962:235–236).

The data on pubertal growth in primates have improved since the publication of *Growth at Adolescence*, though they are largely still restricted to data on body mass. Longitudinal data on the growth of individual animals remain scarce and longitudinal data on specific aspects of growth rather than simply body mass are rarer still. However, those data that do exist largely support the speculations Tanner put forth in 1962.

Leigh (1996) has produced excellent analyses of largely cross-sectional data available from captive species of primates. These data clearly show variability between species in the degree to which pubertal growth spurts in mass are apparent and also show that, where they exist, growth spurts are often sexually dimorphic, with males typically having larger spurts in body mass than females. It is also true that the species with pronounced pubertal spurts in one or both sexes are species that demonstrate adult sexual dimorphism in body size. These sex differences in somatic strategy have long been associated with mating strategies and species ecology, as demonstrated by the reduced to non-existent spurts and reduced to non-existent dimorphism of the pair-bonded species like gibbons and marmosets and the pronounced spurts and dimorphism in the species with harem mating like gorillas and hamadryas baboons.

Longitudinal growth data, where they exist, also reveal inflections in pubertal growth, including of the long bones, in rhesus macaques and chimpanzees, although the degree of inflection varies between individuals and never approaches the magnitude seen in humans (Hamada et al. 1996a; Hamada and Udono 2002). The data also show a clear relationship between pubertal somatic growth and reproductive maturation (Bernstein et al. 2007; Gesquiere et al. 2005; Hamada et al. 1999; Hamada et al. 1996a). In many species, including humans, mandibular and maxillary

growth displays a pubertal growth spurt in males associated with the emergence of dimorphic male canines (Coquerelle et al. 2011; Fukase 2011).

IS THE HUMAN PUBERTAL GROWTH SPURT UNIQUE?

Despite the fact that available evidence supports Tanner's suggestion that pubertal growth spurts are a general, although not universal, primate characteristic, claims that appear to contest this understanding continue to appear. Perhaps the most prominent of these is the proposal, first put forth by Bogin and Smith (1996), that adolescence is a unique human life history stage, with the human pubertal height spurt identified as one of its defining characteristics. This claim seems weak on a number of grounds. Most importantly, other primate species, including rhesus macaques and chimpanzees, do show inflections and often accelerations in linear growth at puberty where careful longitudinal measurements are taken, patterns that are consistent with underlying two-cycle growth. The difference between these species and humans in linear growth spurts at puberty seems to be one of degree, not kind.

In addition, focusing on the height spurt in particular, to the exclusion of evidence of growth spurts in other physical dimensions involved in the development of adult somatic strategies, in humans and other primates, seems arbitrary and 'species-centric'. Certainly we would demure if a mandrill scientist were to assert that a pronounced snout spurt was the defining characteristic of adolescence and thereby concluded that humans did not display evidence of an adolescent life history stage. In fact, the feature of the human skeleton that undergoes the most dramatic pubertal growth spurt is not the trunk or leg bones, but the diameters of the birth canal of the female pelvis (Tanner 1962; Moerman 1982).

The fact that humans display a particularly pronounced height spurt is likely related to our bipedal gait. The cost of transport for humans is a function of leg length and decreases significantly with linear growth (Rogers et al. 1995; DeJaeger et al. 2001; Steudel-Numbers and Tilkens 2004). Human foragers have a day range that is significantly larger than chimpanzees and adult foraging responsibilities are taken on at adulthood (Pontzer and Wrangham 2006; Pontzer et al. 2009). The human linear growth spurt can, thus, easily be understood as part of the general transformation of the soma into adult form, preparing it for adult tasks related to reproduction, including co-operative foraging and the provisioning of dependent offspring.

In our view, Tanner's original thesis remains largely supported by the accumulating evidence: pubertal growth spurts are a general primate growth pattern, part of the process of life history transition from juvenile to adult soma. This transition may not be as dramatic as insect metamorphosis, but like metamorphosis it represents a shift in somatic strategy to serve adult reproductive ends. The differences in pubertal growth spurts, between primate species, between sexes within species, between different

tissues and dimensions within sexes, reflect differences in adult somatic strategies, not differences in kind between the existence or non-existence of unique life history stages. The transformation of the soma that is one of the hallmarks of puberty is itself a downstream consequence of HPG activation that appears broadly distributed in primates, the broad base of the pubertal hour-glass.

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