

Puberty

<http://en.wikipedia.org/wiki/Puberty>

Puberty refers to the process of physical changes by which a child's body becomes an adult body capable of reproduction. Puberty is initiated by hormone signals from the brain to the gonads (the ovaries and testes).

In response, the gonads produce a variety of hormones that stimulate the growth, function, or transformation of brain, bones, muscle, skin, breasts, and reproductive organs.

Growth accelerates in the first half of puberty and stops at the completion of puberty. Before puberty, body differences between boys and girls are almost entirely restricted to the genitalia.

During puberty, major differences of size, shape, composition, and function develop in many body structures and systems. The most obvious of these are referred to as secondary sex characteristics.

In a strict sense, the term puberty (and this article) refers to the bodily changes of sexual maturation rather than the psychosocial and cultural aspects of adolescent development.

Adolescence is the period of psychological and social transition between childhood and adulthood. Adolescence largely overlaps the period of puberty, but its boundaries are less precisely defined and it refers as much to the psychosocial and cultural characteristics of development during the teen years as to the physical changes of puberty.

Two of the most significant differences between puberty in girls and puberty in boys are the age at which it begins, and the major sex steroids involved.

Approximate outline of development periods in child and teenager development. Puberty is marked in green at right.

The normal age for girls begin the process of puberty about 1–2 years earlier than boys (with average ages of 10 to 14 for girls and 12 to 14 for boys),[1]

Typically reach completion in a shorter time,[2] with girls usually having completed puberty by age 16 or 17.[3][4] For girls, any increase in height beyond these ages is uncommon.[4] Boys have usually completed puberty by 17 or 18.

Girls attain reproductive maturity about 4 years after the first physical changes of puberty appear.[4] In contrast, boys accelerate more slowly but continue to grow for about 6 years after the first visible pubertal changes.[5]

In males, testosterone, an androgen, is the principal sex steroid. While testosterone produces all the male changes characterized as virilization, a substantial product of testosterone metabolism in males is estradiol, though levels rise later and more slowly than in girls. The male growth spurt also begins later, accelerates more slowly, and lasts longer before the epiphyses fuse.

Although boys are 2 cm shorter than girls before puberty begins, adult men are on average about 13 cm (5.2 inches) taller than women. Most of this sex difference in adult heights is attributable to a later onset of the growth spurt and a slower progression to completion,[6] a direct result of the later rise and lower adult male levels of estradiol.

The hormone that dominates female development is estradiol, an estrogen. While estradiol promotes growth of breasts and uterus, it is also the principal hormone driving the pubertal growth spurt and epiphyseal maturation and closure.[7] Estradiol levels rise earlier and reach higher levels in women than in men.

Puberty onset

Onset is associated with high GnRH pulsing, which precedes the rise in sex hormones, LH and FSH.[8] Exogenous GnRH pulses cause the onset of puberty.[9] Brain tumors which increase GnRH output may also lead to premature puberty.[10]

The cause of this GnRH rise is contentious. Puberty begins consistently at around 47 kg for girls and 55 kg for boys[citation needed]. This dependence on bodyweight makes leptin a good candidate for causing GnRH rise. It is known that leptin has receptors in the hypothalamus which synthesises GnRH.[11]

Furthermore, individuals who are deficient in the leptin pathway fail to initiate puberty.[12] The levels of leptin change in line with the onset of puberty, and then decline to adult levels. However, the onset of puberty might also be caused by genetics.

A study carried out by Cukurova University in Turkey discovered that a mutation in genes encoding both Neurokinin B as well as the Neurokinin B receptor can alter the timing of puberty. To accomplish this, authors hypothesize that Neurokinin B might play a role in regulating the secretion of Kisspeptin, a compound responsible for triggering direct release of Gonadotropin-releasing hormone as well as indirect release of Luteinizing Hormone and Follicle Stimulating Hormone.[13]

Physical changes in males – Testicular size, function, and fertility

In boys, testicular enlargement is the first physical manifestation of puberty (and is termed gonadarche).[14] Testes in prepubertal boys change little in size from about 1 year of age to the onset of puberty, averaging about 2–3 cc in volume and about 1.5–2 cm in length.

Testicular size continues to increase throughout puberty, reaching maximal adult size about 6 years later.[15] While 18–20 cc is reportedly an average adult size, there is wide variation in the normal population.[16]

The testes have two primary functions: to produce hormones and to produce sperm. The Leydig cells produce testosterone (as described below), which in turn produces most of the changes of male sexual maturation and maintains libido. However, most of the increasing bulk of testicular tissue is spermatogenic tissue (primarily Sertoli and interstitial cells).

The development of sperm production and fertility in males is not as well documented. Sperm can be detected in the morning urine of most boys after the first year of pubertal changes (and occasionally earlier). Potential fertility is reached at about 13 years old in boys, but full fertility will not be gained until 14–16 years of age, although some go through the process faster, reaching it only 1 year later.

Pubic hair

Pubic hair often appears on a boy shortly after the genitalia begin to grow. As in girls, the first appearance of pubic hair is termed pubarche. The pubic hairs are usually first visible at the dorsal (abdominal) base of the penis.

The first few hairs are described as stage 2. Stage 3 is usually reached within another 6–12 months, when the hairs are too many to count. By stage 4, the pubic hairs densely fill the "pubic triangle."

Stage 5 refers to spread of pubic hair to the thighs and upward towards the navel as part of the developing abdominal hair.

Body and facial hair – Facial hair of a male that has been shaved

In the months and years following the appearance of pubic hair, other areas of skin which respond to androgens develop heavier hair (androgenic hair) in roughly the following sequence: underarm (axillary) hair, perianal hair, upper lip hair, sideburn (preauricular) hair, periareolar hair, and the rest of the beard area.

Arm, leg, chest, abdominal, and back hair become heavier more gradually. There is a large range in amount of body hair among adult men, and significant differences in timing and quantity of hair growth among different ethnic groups.[1]

Facial hair in males normally appears in a specific order during puberty: The first facial hair to appear tends to grow at the corners of the upper lip, typically between 14 to 16 years of age.[17][18] It then spreads to form a moustache over the entire upper lip. This is followed by the appearance of hair on the upper part of the cheeks, and the area under the lower lip.[17]

The hair eventually spreads to the sides and lower border of the chin, and the rest of the lower face to form a full beard.[17] As with most human biological processes, this specific order may vary among some individuals.

Facial hair is often present in late adolescence, around ages 17 and 18, but may not appear until significantly later.[18][19] Some men do not develop full facial hair for 10 years after puberty.[18] Facial hair will continue to get coarser, darker and thicker for another 2–4 years after puberty.[18]

Chest hair may appear during puberty or years after.[1] Not all men have chest hair.

Voice change

Under the influence of androgens, the voice box, or larynx, grows in both sexes. This growth is far more prominent in boys, causing the male voice to drop and deepen, sometimes abruptly but rarely "over night," about one octave, because the longer and thicker vocal folds have a lower fundamental frequency. Before puberty, the larynx of boys and girls is about equally small.[20]

Occasionally, voice change is accompanied by unsteadiness of vocalization in the early stages of untrained voices. Most of the voice change happens during stage 3–4 of male puberty around the time of peak growth. Full adult pitch is attained at an average age of about 15 years. However, it usually precedes the development of significant facial hair by several months to years.

Male musculature and body shape

By the end of puberty, adult men have heavier bones and nearly twice as much skeletal muscle. Some of the bone growth (e.g., shoulder width and jaw) is disproportionately greater, resulting in noticeably different male and female skeletal shapes. The average adult male has about 150% of the lean body mass of an average female, and about 50% of the body fat.

This muscle develops mainly during the later stages of puberty, and muscle growth can continue even after a male is biologically adult. The peak of the so-called "strength spurt," the rate of muscle growth, is attained about one year after a male experiences his peak growth rate.

Body odor and acne

Rising levels of androgens can change the fatty acid composition of perspiration, resulting in a more "adult" body odor. As in girls, another androgen effect is increased secretion of oil (sebum) from the skin and the resultant variable amounts of acne. Acne can not be prevented or diminished easily, but it typically fully diminishes at the end of puberty.

However, it is not unusual for a fully grown adult to suffer the occasional bout of acne, though it is normally less severe than in adolescents. Some may need prescription topical creams or ointments to keep acne from getting worse, or even oral medication. Acne may also cause scarring.

Physical changes in females

Breast development

The first physical sign of puberty in females is usually a firm, tender lump under the center of the areola(e) of one or both breasts, occurring on average at about 10.5 years of age.[21] This is referred to as thelarche.

By the widely used Tanner staging of puberty, this is stage 2 of breast development (stage 1 is a flat, prepubertal breast). Within six to 12 months, the swelling has clearly begun in both sides, softened, and can be felt and seen extending beyond the edges of the areolae. This is stage 3 of breast development.

By another 12 months (stage 4), the breasts are approaching mature size and shape, with areolae and papillae forming a secondary mound. In most young women, this mound disappears into the contour of the mature breast (stage 5), although there is so much variation in sizes and shapes of adult breasts that stages 4 and 5 are not always separately identifiable.[22]

Pubic hair

Pubic hair is often the second unequivocal change of puberty noticed, usually within a few months of thelarche.[23] It is referred to as pubarche and the pubic hairs are usually visible first along the labia.

The first few hairs are described as Tanner stage 2.[22] Stage 3 is usually reached within another 6–12 months, when the hairs are too numerous to count and appear on the pubic mound as well. By stage 4, the pubic hairs densely fill the "pubic triangle."

Stage 5 refers to spread of pubic hair to the thighs and sometimes as abdominal hair upward towards the navel. In about 15% of girls, the earliest pubic hair appears before breast development begins.[23]

Vagina, uterus, ovaries

The mucosal surface of the vagina also changes in response to increasing levels of estrogen, becoming thicker and a duller pink in color (in contrast to the brighter red of the prepubertal vaginal mucosa).[24]

Whitish secretions (physiologic leukorrhea) are a normal effect of estrogen as well.[21] In the next 2 years following thelarche, the uterus and ovaries increase in size, and follicles in the ovaries reach larger sizes.[25] The ovaries usually contain small follicular cysts visible by ultrasound.[26][27]

Menstruation and Fertility

The first menstrual bleeding is referred to as menarche, and typically occurs about 2 years after thelarche.[23] The average age of menarche in American girls is about 11.75 years.[23] Menses (menstrual periods) are not always regular and monthly in the first 2 years after menarche.[28] Ovulation is necessary for fertility, but may or may not accompany the earliest menses.[29]

In postmenarchal girls, about 80% of the cycles were anovulatory in the first year after menarche, 50% in the third and 10% in the sixth year.[28] However, initiation of ovulation after menarche is not inevitable, and a high proportion of girls with continued irregularity several years from menarche will continue to have prolonged irregularity and anovulation, and are at higher risk for reduced fertility.[30] The word nubility is used commonly in the social sciences to designate achievement of fertility.

Body shape, fat distribution, and body composition

During this period, also in response to rising levels of estrogen, the lower half of the pelvis and thus hips widen (providing a larger birth ca-

nal).[22][31] Fat tissue increases to a greater percentage of the body composition than in males, especially in the typical female distribution of breasts, hips, buttocks, thighs, upper arms, and pubis.

Progressive differences in fat distribution as well as sex differences in local skeletal growth contribute to the typical female body shape by the end of puberty. At age 10 years, the average girl has 6% more body fat than the average boy, but by the end of puberty the average difference is nearly 50%.[32]

Body odor and acne

Rising levels of androgens can change the fatty acid composition of perspiration, resulting in a more "adult" body odor. This often precedes thelarche and pubarche by 1 or more years. Another androgen effect is increased secretion of oil (sebum) from the skin. This change increases the susceptibility to acne, a characteristic affliction of puberty greatly variable in its severity.[33]

Timing of onset

The definition of onset depends on perspective (e.g., hormonal versus physical) and purpose (establishing population normal standards, clinical care of early or late pubescent individuals, or a variety of other social purposes).

The most commonly used definition of onset for both social and medical purposes is the appearance of the first physical changes. These physical changes are the first outward signs of preceding neural, hormonal, and gonadal function changes that are usually impossible or impractical to detect.

The age at which puberty begins varies between individuals and between populations. Age of puberty is affected by both genetic factors and by environmental factors such as nutritional state or social circumstances.[1]

Ethnic/racial differences have been recognized for centuries. For example, the average age of menarche in various populations surveyed in the last several decades has ranged from 12 to 18 years. The earliest mean is reported for African-American girls and the oldest for high altitude subsistence populations in Asia.

However, it is clear that much of the higher age averages reflect nutritional limitations more than genetic differences and can change within a few generations with a substantial change in diet. The median age of menarche for a population may be an index of the proportion of undernourished girls in the population, and the width of the spread may reflect unevenness of wealth and food distribution in a population.

Researchers have identified an earlier age of the onset of puberty. However, they have based their conclusions on a comparison of data from 1999 with data from 1969. In the earlier example, the sample population was based on a small sample of white girls (200, from Britain). The later study identified as puberty as occurring in 48% of African-American girls by age nine, and 12% of white girls by that age.[34]

Historical shift

The age at which puberty occurs has dropped significantly since the 1840s.[35][36][37] Researchers refer to this drop as the 'secular trend'. From 1840 through 1950, in each decade there was a drop of four months in the average age of menarche among Western European female samples.

In Norway, girls born in 1840 had their menarche at average 17 years. In France in 1840 the average was 15.3 years. In England the 1840 average was 16.5 years for girls. In Japan the decline happened later and was then more rapid: from 1945 to 1975 in Japan there was a drop of 11 months per decade.

Genetic influence and environmental factors

Various studies have found direct genetic effects to account for at least 46% of the variation of timing of puberty in well-nourished populations.[38][39][40][41] The genetic association of timing is strongest between mothers and daughters. The specific genes affecting timing are not defined yet.[38] Among the candidates is an androgen receptor gene.[42]

Researchers has postulated that early puberty onset may be caused by certain types of hair care products (containing estrogen or placenta), and by certain kinds of chemicals, namely, phthalates, which are used in many cosmetics, toys, and plastic food containers.[43]

If genetic factors account for half of the variation of pubertal timing, environment factors are clearly important as well. One of the earliest observed environmental effects is that puberty occurs later in children raised at higher altitudes.

The most important of the environmental influences is clearly nutrition, but a number of others have been identified, all which affect timing of female puberty and menarche more clearly than male puberty.

Hormones and steroids

There is theoretical concern, and animal evidence, that environmental hormones and chemicals may affect aspects of prenatal or postnatal sexual development in humans.[44]

Large amounts of incompletely metabolized estrogens and progestagens from pharmaceutical products are excreted into the sewage systems of large cities, and are sometimes detectable in the environment.

Sex steroids are sometimes used in cattle farming but have been banned in chicken meat production for 40 years. Although agricultural laws regulate use to minimize accidental human consumption, the rules are largely self-enforced in the United States.

Significant exposure of a child to hormones or other substances that activate estrogen or androgen receptors could produce some or all of the changes of puberty.

Harder to detect as an influence on puberty are the more diffusely distributed environmental chemicals like PCBs (polychlorinated biphenyl), which can bind and trigger estrogen receptors.

More obvious degrees of partial puberty from direct exposure of young children to small but significant amounts of pharmaceutical sex steroids from exposure at home may be detected during medical evaluation for precocious puberty, but mild effects and the other potential exposures outlined above would not.

Bisphenol A (BPA) is a chemical used to make plastics, and is frequently used to make baby bottles, water bottles, sports equipment, medical devices, and as a coating in food and beverage cans. Scientists are concerned about BPA's behavioral effects on fetuses, infants, and children at current exposure levels because it can effect the prostate gland, mammary gland, and lead to early puberty in girls.

BPA mimics and interferes with the action of estrogen—an important reproduction and development regulator. It leaches out of plastic into liquids and foods, and the Centers for Disease Control and Prevention (CDC) found measurable amounts of BPA in the bodies of more than 90 percent of the U.S. population studied.

The highest estimated daily intakes of BPA occur in infants and children. Many plastic baby bottles contain BPA, and BPA is more likely to leach out of plastic when its temperature is increased, as when one warms a baby bottle or warms up food in the microwave.[45]

Nutritional influence

Nutritional factors are the strongest and most obvious environmental factors affecting timing of puberty.[38] Girls are especially sensitive to nutritional regulation because they must contribute all

of the nutritional support to a growing fetus. Surplus calories (beyond growth and activity requirements) are reflected in the amount of body fat, which signals to the brain the availability of resources for initiation of puberty and fertility.

Much evidence suggests that for most of the last few centuries, nutritional differences accounted for majority of variation of pubertal timing in different populations, and even among social classes in the same population.

Recent worldwide increased consumption of animal protein, other changes in nutrition, and increases in childhood obesity have resulted in falling ages of puberty, mainly in those populations with the higher previous ages. In many populations the amount of variation attributable to nutrition is shrinking.

Although available dietary energy (simple calories) is the most important dietary influence on timing of puberty, quality of the diet plays a role as well. Lower protein intakes and higher dietary fiber intakes, as occur with typical vegetarian diets, are associated with later onset and slower progression of female puberty.

Studies have shown that calcium deficiency is a cause of late puberty, irregular and painful cramping during menstruation with excessive blood loss, and lowered immune response to infections in young girls. This could be from a deficient diet or lack of vitamin D from too little sun exposure. This lack of calcium could predispose them to osteoporosis later in life.

Obesity influence and exercise

Scientific researchers have linked early obesity with a drop of puberty onset in girls. They have cited obesity as a cause of breast development before nine years and menarche before twelve years.[46] Early puberty in girls can be a harbinger of later health problems.[47]

The average level of daily physical activity has also been shown to affect timing of puberty, especially female. A high level of exercise, whether for athletic or body image purposes, or for daily subsistence, reduces energy calories available for reproduction and slows puberty. The exercise effect is often amplified by a lower body fat mass and cholesterol.

Physical and mental illness

Chronic diseases can delay puberty in both boys and girls. Those that involve chronic inflammation or interfere with nutrition have the strongest effect. In the western world, inflammatory bowel disease and tuberculosis have been notorious for such an effect in the last century, while in

areas of the underdeveloped world, chronic parasite infections are widespread.

Mental illnesses occur in puberty. The brain undergoes significant development by hormones which can contribute to mood disorders such as Major depressive disorder, bipolar disorder, dysthymia and schizophrenia. Girls aged between 15 and 19 make up 40% of anorexia nervosa cases.[48]

Stress and social factors

Some of the least understood environmental influences on timing of puberty are social and psychological. In comparison with the effects of genetics, nutrition, and general health, social influences are small, shifting timing by a few months rather than years. Mechanisms of these social effects are unknown, though a variety of physiological processes, including pheromones, have been suggested based on animal research.

The most important part of a child's psychosocial environment is the family, and most of the social influence research has investigated features of family structure and function in relation to earlier or later female puberty.

Most of the studies have reported that menarche may occur a few months earlier in girls in high-stress households, whose fathers are absent during their early childhood, who have a stepfather in the home, who are subjected to prolonged sexual abuse in childhood, or who are adopted from a developing country at a young age. Conversely, menarche may be slightly later when a girl grows up in a large family with a biological father present.

More extreme degrees of environmental stress, such as wartime refugee status with threat to physical survival, have been found to be associated with delay of maturation, an effect that may be compounded by dietary inadequacy.

Most of these reported social effects are small and our understanding is incomplete. Most of these "effects" are statistical associations revealed by epidemiologic surveys. Statistical associations are not necessarily causal, and a variety of covariables and alternative explanations can be imagined.

Effects of such small size can never be confirmed or refuted for any individual child. Furthermore, interpretations of the data are politically controversial because of the ease with which this type of research can be used for political advocacy. Accusations of bias based on political agenda sometimes accompany scientific criticism.

Another limitation of the social research is that nearly all of it has concerned girls, partly because female puberty requires greater physiologic resources and partly because it involves a unique event (menarche) that makes survey research into female puberty much simpler than male. More detail is provided in the menarche article.

Variations of sequence

The sequence of events of pubertal development can occasionally vary. For example, in about 15% of boys and girls, pubarche (the first pubic hairs) can precede, respectively, gonadarche and thelarche by a few months. Rarely, menarche can occur before other signs of puberty in a few girls. These variations deserve medical evaluation because they can occasionally signal a disease.

Conclusion

In a general sense, the conclusion of puberty is reproductive maturity. Criteria for defining the conclusion may differ for different purposes: attainment of the ability to reproduce, achievement of maximal adult height, maximal gonadal size, or adult sex hormone levels.

Maximal adult height is achieved at an average age of 15 years for an average girl and 18 years for an average boy. Potential fertility (sometimes termed nubility) usually precedes completion of growth by 1–2 years in girls and 3–4 years in boys. Stage 5 in the tables above[clarification needed] typically represents maximal gonadal growth and attainment of adult hormone levels.

Neurohormonal process

The endocrine reproductive system consists of the hypothalamus, the pituitary, the gonads, and the adrenal glands, with input and regulation from many other body systems. True puberty is often termed "central puberty" because it begins as a process of the central nervous system. A simple description of hormonal puberty is as follows:

1. The brain's hypothalamus begins to release pulses of GnRH.
2. Cells in the anterior pituitary respond by secreting LH and FSH into the circulation.
3. The ovaries or testes respond to the rising amounts of LH and FSH by growing and beginning to produce estradiol and testosterone.
4. Rising levels of estradiol and testosterone produce the body changes of female and male puberty.

The onset of this neurohormonal process may precede the first visible body changes by 1–2 years.

Components of the endocrine reproductive system

The arcuate nucleus of the hypothalamus is the driver of the reproductive system. It has neurons which generate and release pulses of GnRH into the portal venous system of the pituitary gland. The arcuate nucleus is affected and controlled by neuronal input from other areas of the brain and hormonal input from the gonads, adipose tissue and a variety of other systems.

The pituitary gland responds to the pulsed GnRH signals by releasing LH and FSH into the blood of the general circulation, also in a pulsatile pattern.

The gonads (testes and ovaries) respond to rising levels of LH and FSH by producing the steroid sex hormones, testosterone and estradiol.

The adrenal glands are a second source for steroid hormones. Adrenal maturation, termed adrenarche, typically precedes gonadarche in mid-childhood.

Major hormones

- Neurokinin B (a tachykinin peptide) and kisspeptin (a G protein-coupled receptor), both present in the same hypothalamic neurons, are critical parts of the control system that switches on the release of GnRH at the start of puberty.[49]
- GnRH (gonadotropin-releasing hormone) is a peptide hormone released from the hypothalamus which stimulates gonadotrope cells of the anterior pituitary.
- LH (luteinizing hormone) is a larger protein hormone secreted into the general circulation by gonadotrope cells of the anterior pituitary gland. The main target cells of LH are the Leydig cells of testes and the theca cells of the ovaries. LH secretion changes more dramatically with the initiation of puberty than FSH, as LH levels increase about 25-fold with the onset of puberty, compared with the 2.5-fold increase of FSH.
- FSH (follicle stimulating hormone) is another protein hormone secreted into the general circulation by the gonadotrope cells of the anterior pituitary. The main target cells of FSH are the ovarian follicles and the Sertoli cells and spermatogenic tissue of the testes.
- Testosterone is a steroid hormone produced primarily by the Leydig cells of the testes, and in lesser amounts by the theca cells of the ovaries and the adrenal cortex. Testosterone is the primary mammalian androgen and the "original" anabolic steroid. It acts on androgen receptors in responsive tissue throughout the body.

- Estradiol is a steroid hormone produced by aromatization of testosterone. Estradiol is the principal human estrogen and acts on estrogen receptors throughout the body. The largest amounts of estradiol are produced by the granulosa cells of the ovaries, but lesser amounts are derived from testicular and adrenal testosterone.
- Adrenal androgens are steroids produced by the zona reticulosa of the adrenal cortex in both sexes. The major adrenal androgens are dehydroepiandrosterone, androstenedione (which are precursors of testosterone), and dehydroepiandrosterone sulfate which is present in large amounts in the blood. Adrenal androgens contribute to the androgenic events of early puberty in girls.
- IGF1 (insulin-like growth factor 1) rises substantially during puberty in response to rising levels of growth hormone and may be the principal mediator of the pubertal growth spurt.
- Leptin is a protein hormone produced by adipose tissue. Its primary target organ is the hypothalamus. The leptin level seems to provide the brain a rough indicator of adipose mass for purposes of regulation of appetite and energy metabolism. It also plays a permissive role in female puberty, which usually will not proceed until an adequate body mass has been achieved.

Endocrine perspective

The endocrine reproductive system becomes functional by the end of the first trimester of fetal life. The testes and ovaries become briefly inactive around the time of birth but resume hormonal activity until several months after birth, when incompletely understood mechanisms in the brain begin to suppress the activity of the arcuate nucleus.

This has been referred to as maturation of the prepubertal "gonadostat," which becomes sensitive to negative feedback by sex steroids. The period of hormonal activity until several months after birth, followed by suppression of activity, may correspond to the period of infant sexuality, followed by a latency stage, which Sigmund Freud described.[50]

Gonadotropin and sex steroid levels fall to low levels (nearly undetectable by current clinical assays) for approximately another 8 to 10 years of childhood. Evidence is accumulating that the reproductive system is not totally inactive during the childhood years. Subtle increases in gonadotropin pulses occur, and ovarian follicles surrounding germ cells (future eggs) double in number.

Normal puberty is initiated in the hypothalamus, with de-inhibition of the pulse generator in the arcuate nucleus. This inhibition of the arcuate nucleus is an ongoing active suppression by other areas of the brain. The signal and mechanism releasing the arcuate nucleus from inhibition have been the subject of investigation for decades and remain incompletely understood.

Leptin levels rise throughout childhood and play a part in allowing the arcuate nucleus to resume operation. If the childhood inhibition of the arcuate nucleus is interrupted prematurely by injury to the brain, it may resume pulsatile gonadotropin release and puberty will begin at an early age.

Neurons of the arcuate nucleus secrete gonadotropin releasing hormone (GnRH) into the blood of the pituitary portal system. An American physiologist, Ernst Knobil, found that the GnRH signals from the hypothalamus induce pulsed secretion of LH (and to a lesser degree, FSH) at roughly 1-2 hour intervals.

The LH pulses are the consequence of pulsatile GnRH secretion by the arcuate nucleus that, in turn, is the result of an oscillator or signal generator in the central nervous system ("GnRH pulse generator").[51]

In the years preceding physical puberty, Robert M. Boyar discovered that the gonadotropin pulses occur only during sleep, but as puberty progresses they can be detected during the day.[52] By the end of puberty, there is little day-night difference in the amplitude and frequency of gonadotropin pulses.

Some investigators have attributed the onset of puberty to a resonance of oscillators in the brain.[53][54][55][56] By this mechanism, the gonadotropin pulses that occur primarily at night just before puberty represent beats.[57][58][59]

An array of "autoamplification processes" increases the production of all of the pubertal hormones of the hypothalamus, pituitary, and gonads.

Regulation of adrenarche and its relationship to maturation of the hypothalamic-gonadal axis is not fully understood, and some evidence suggests it is a parallel but largely independent process coincident with or even preceding central puberty.

Rising levels of adrenal androgens (termed adrenarche) can usually be detected between 6 and 11 years of age, even before the increasing gonadotropin pulses of hypothalamic puberty.

Adrenal androgens contribute to the development of pubic hair (pubarche), adult body odor, and other androgenic changes in both sexes.

The primary clinical significance of the distinction between adrenarche and gonadarche is that pubic hair and body odor changes by themselves do not prove that central puberty is underway for an individual child.

Hormonal changes in boys

Early stages of male hypothalamic maturation seem to be very similar to the early stages of female puberty, though occurring about 1-2 years later.

LH stimulates the Leydig cells of the testes to make testosterone and blood levels begin to rise. For much of puberty, nighttime levels of testosterone are higher than daytime. Regularity of frequency and amplitude of gonadotropin pulses seems to be less necessary for progression of male than female puberty.

However, a significant portion of testosterone in adolescent boys is converted to estradiol. Estradiol mediates the growth spurt, bone maturation, and epiphyseal closure in boys just as in girls. Estradiol also induces at least modest development of breast tissue (gynecomastia) in a large proportion of boys.

Boys who develop mild gynecomastia or even developing swellings under nipples during puberty are told the effects are temporary in some male teenagers due to high levels of Estradiol.

Another hormonal change in males takes place during the teenage years for most young men. At this point in a males life the testosterone levels slowly rise, and most of the effects are mediated through the androgen receptors by way of conversion dihydrotestosterone in target organs (especially that of the bowels).

Hormonal changes in girls

As the amplitude of LH pulses increases, the theca cells of the ovaries begin to produce testosterone and smaller amounts of progesterone. Much of the testosterone moves into nearby cells called granulosa cells.

Smaller increases of FSH induce an increase in the aromatase activity of these granulosa cells, which converts most of the testosterone to estradiol for secretion into the circulation.

Rising levels of estradiol produce the characteristic estrogenic body changes of female puberty: growth spurt, acceleration of bone maturation and closure, breast growth, increased fat composition, growth of the uterus, increased thickness of the

endometrium and the vaginal mucosa, and widening of the lower pelvis.

As the estradiol levels gradually rise and the other autoamplification processes occur, a point of maturation is reached when the feedback sensitivity of the hypothalamic "gonadostat" becomes positive. This attainment of positive feedback is the hallmark of female sexual maturity, as it allows the mid cycle LH surge necessary for ovulation.

Levels of adrenal androgens and testosterone also increase during puberty, producing the typical androgenic changes of female puberty: pubic hair, other androgenic hair as outlined above, body odor, acne.

Growth hormone levels rise steadily throughout puberty. IGF1 levels rise and then decline as puberty ends. Growth finishes and adult height is attained as the estradiol levels complete closure of the epiphyses.

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Onset of puberty, menstrual frequency, and body fat in elite rhythmic gymnasts compared with normal controls

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Conclusion: Delayed menarche, menstrual irregularities, and low body fat are common in elite rhythmic gymnasts. Premenarcheal gymnasts train more often and for longer, and have a lower body mass index and less body fat, than menarcheal gymnasts. Prospective studies are needed to explore further these and other factors associated with delayed menarche and menstrual irregularities in female athletes.

Publications

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Weight Status in Young Girls and the Onset of Puberty

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