Growth and pubertal development in children and adolescents: effects of diet and physical activity

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ABSTRACT The longitudinal growth of an individual child is a dynamic statement of the general health of that child. Measurements should be performed often and accurately to detect alterations from physiologic growth. Although any single point on the growth chart is not very informative, when several growth points are plotted over time, it should become apparent whether that individual’s growth is average, a variant of the norm, or pathologic. Somatic growth and maturation are influenced by several factors that act independently or in concert to modify an individual’s genetic growth potential. Linear growth within the first 2 y of life generally decelerates but then remains relatively constant throughout childhood until the onset of the pubertal growth spurt. Because of the wide variation among individuals in the timing of the pubertal growth spurt, there is a wide range of physiologic variations in normal growth. Nutritional status and heavy exercise training are only 2 of the major influences on the linear growth of children. In the United States, nutritional deficits result from self-induced restriction of energy intake. That single factor, added to the marked energy expenditure of training and competition for some sports, and in concert with the self-selection of certain body types, makes it difficult to identify the individual factors responsible for the slow linear growth of some adolescent athletes, for example, those who participate in gymnastics, dance, or wrestling.

GROWTH MEASUREMENTS

The growth of children should be measured periodically and accurately. Two common devices are adequate for such measurements and were described by Rogol and Lawton (2).

INTRODUCTION

A child’s growth can be compared with that of his or her peers by referring to the norm on an appropriate growth chart. More important, the longitudinal measurements of a child’s growth are a dynamic statement of his or her general condition or health. Tanner (1) has proposed that children be measured accurately to identify individuals or groups of individuals within a community who require special care, to identify illnesses that influence growth, or to determine an ill child’s response to therapy. The linear growth of a child-adolescent athlete may also reflect the adequacy of energy intake for a particular training regimen. Measurement of growth may also be used as an index of the general health and nutrition of a population or subpopulation of children.

By definition, normal (physiologic) growth encompasses the 95% CI for a specific population. Most children and adolescents who have a normal growth pattern but who remain below the lower 2.5 percentile (approximately −2.0 SD) are otherwise normal. The farther an individual’s growth falls below the −2.0 SD mark, the more likely he or she is to have a condition that is keeping him or her from reaching the genetically determined height potential.

Cross-sectional data are derived from the measurements of many children at various ages and are generally used to derive standard growth charts. However, individual children do not necessarily grow according to these standard curves. Longitudinal growth charts derived from growth points of the same child over time more accurately describe the growth pattern of an individual. In adolescence, there may be quite large deviations from the derived percentile lines, depending on the timing and tempo of the pubertal growth spurt. An average pubertal growth pattern is built into the percentiles derived from cross-sectional data, but virtually no one individual adheres strictly to that pattern.

GROWTH MEASUREMENTS

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Neonates and infants

Small inaccuracies in length measurement can easily affect a child’s percentiles on growth curve charts. Infants should be placed with the top of the head against the fixed headboard of the measurement device and with the eye-ear plane perpendicular to the base of the device (Figure 1). The child’s knees must be flat against the table and the footboard moved until the soles of the feet are against it, with the toes pointing up.
Children and adolescents

Growth in children older than 2 y is measured with the child standing. A diurnal variation of <0.7 cm in height may occur in these children, whose height is greatest upon arising. Children should be measured without shoes while standing against the vertical plane to which the measuring tape is attached. The child’s heels, buttocks, shoulders, and back of the head should be touching the wall (Figure 2). The eye-ear plane should be perpendicular to the wall and the feet, including the heels, should be flat on the floor. With the child in this position, the right-angle device is lowered until it touches the top of the head, and the height is recorded on the appropriate physical growth curve.

An important parameter of growth is height velocity, which can be derived from measurements taken every 3–4 mo in infants and every 6 mo in older children. Because children often have growth spurts, yearly growth velocities are usually more accurately determined by taking yearly measurements rather than by “annualizing” the growth velocity from intervals shorter than 1 y. Growth velocity in children has a wide normal range, according to the percentile along which a child is growing. Children growing along the third percentile average 5.1 cm of growth per year, whereas those growing along the 97th percentile average 6.4 cm/y for boys and 7.1 cm/y for girls during childhood to maintain growth along one of the percentiles on the growth curve (3). To maintain growth along the 10th percentile for height, a child must grow at the 40th percentile for velocity, whereas to maintain growth along the 90th percentile for height, a velocity at the 60th percentile is required. This implies that a child who persistently grows at the 10th percentile for velocity will progressively cross percentiles downward on the standard height curve. Some children have a small increase in growth velocity at approximately 6–7 y (midgrowth spurt), but this is not a consistent finding, and the gain in height is generally of small magnitude (4). Seasonal variations in growth have been noted in some children. Linear growth tends to be greater in the spring than in the fall, but weight gain is greater in the fall months. These trends emphasize the need for repeated measurements during a year to accurately assess a child’s growth pattern.

Growth assessment

Linear growth and physical maturation are dynamic processes encompassing molecular, cellular, somatic, and organismal changes. Traditionally, stature has been primarily used for growth assessment, but changes in body proportion and composition are also essential elements of growth, especially of maturation. Growth standards have been derived for several populations and parameters within a population and are often codified into a series of growth charts. The following discussion emphasizes the genetic, nutritional, hormonal, and physical activity factors that might alter the growth process.

PHYSIOLOGIC GROWTH PATTERNS

Although any single point on a growth chart is not very informative, when several growth points are plotted over time, it becomes apparent whether an individual’s growth is average, a variant of the norm, or pathologic (growth failure). The point at which an individual is placed at any given time can be related to the height age, or the age at which that child’s height would be at the 50th percentile. This point indicates the mean age of children of that measured height in the normal population. The height age is determined from the growth chart by drawing a line parallel to the chronologic age axis from the child’s plotted point to the 50th percentile and then a perpendicular line to the horizontal axis. The intersection of the latter line with the age axis is the height age.

Growth in several dimensions shows a significant family resemblance. Adult stature, tempo of growth, timing and rate of sexual development, skeletal maturation, and dental development are all significantly influenced by genetic factors (5) and estimates of genetic transmissibility range from 41% to 71% (4). Adult stature is best correlated with calculations of midparental height (the difference in the mean adult heights of the parents), but the polygenic mode of inheritance of height results in greater variation in the size of children born to parents of disparate heights than in children of parents who are both of medium height (6). Mature height can be predicted on the basis of midparental height. The adjusted midparental height (target height) is calculated by adding 13 cm (the difference between the 50th percentiles for adult men and women) to the mother’s height (for boys) or subtracting 13 cm from the father’s height (for girls).
and then taking the mean of the height of the same-sex parent and the adjusted height of the opposite-sex parent. Adding 8.5 cm above and below the midparent target height will approximate the target height range of the 3rd to 97th percentile for the anticipated adult height for that child adjusted for his or her midparental stature (genetic potential). Other methods also exist for predicting the adult stature of an individual on the basis of mathematical formulations derived from the growth history of that child or from the attained height and bone age of the child as calculated from specific tables.

The overall contribution of heredity to adult size and shape varies with environmental circumstances, and the 2 continuously interact throughout the entire growth period. Children with similar genotypes, who would reach the same adult height under optimal conditions, may be differentially affected by adverse circumstances. Thus, the interaction between genetic makeup and the environment is complex and nonadditive. The genetic control of the tempo of growth appears to be independent of that for body size and shape, and environmentally induced changes in tempo do not seem to significantly alter adult height or shape (4).

FACTORS INFLUENCING SOMATIC GROWTH

Somatic growth and maturation are influenced by several factors that act independently or in concert to modify an individual’s genetic potential. For example, at birth, an infant’s size is more dependent on maternal nutrition and intrauterine and placental factors than on genetic makeup. The correlation coefficient for adult height is only 0.25 at birth but is 0.80 by 2 y of age (7). There is also evidence that not all genes are actively expressed at the time of birth, which probably accounts for the observation that the correlation between the sizes of the parents and the child is weak during the first year of life but increases to the adult value of 0.5 by ≈18 mo of age (4).

Differences in growth and development also vary as a function of sex and ethnic origin. Sex-specific patterns in the tempo of growth, the timing of the adolescent growth spurt, overall size, and the age of skeletal maturity are well known, but differences between the sexes are apparent from the time of fetal life. At birth, the skeletal maturation of females is 4–6 wk more advanced than that of males, and this trend continues throughout childhood and adolescence. Growth velocity is slightly slower in females at birth, becomes equal at ≈7 mo of age, and is then somewhat faster until age 4 y. Thereafter, children of both sexes grow at approximately the same rate until the adolescent growth spurt. On average, females enter puberty 2 y earlier than males but have a lesser peak height velocity (9 cm compared with 10.3 cm) and adult stature (8, 9). Overall size and rate of development vary significantly among ethnic populations. Black infants tend to be smaller at birth but experience an acceleration of linear growth that results in greater height than in white children during the first few years of life. Skeletal maturity in black children, especially girls, also tends to be more advanced and the age at peak height velocity earlier (10, 11). Black girls also tend to be taller and heavier than white girls during puberty and have a tendency toward greater body mass index and greater skinfold-thickness measurements.

Growth in the first 2 y of life

Growth during the first 2 y of life is characterized by a gradual deceleration in both linear growth velocity and rate of weight gain, both of which level off at 2–3 y of age. It is during this period that infants exhibit the pattern of growth consistent with their genetic backgrounds. Two-thirds of all infants cross percentiles on the growth curve, either upward (catch-up growth) or downward (lag-down growth) (6). Catch-up growth typically begins within the first 3 mo and is complete by 12–18 mo, whereas lag-down growth commences a little later and may not be complete until 18–24 mo (6). With the exception of puberty, the crossing of growth percentiles at any other time is cause for concern and further evaluation.

Prepubertal growth

Growth during childhood is a relatively stable process. The infancy shifts in the growth pattern are complete and the child follows the trajectory attained previously. Until about the age of 4 y, girls grow slightly faster than boys and both sexes then average a rate of 5–6 cm/y and 2.5 kg/y until the onset of puberty (4). A general rule of thumb is that a child grows 10 cm (25 inches) in the first year of life, half that (12–13 cm (5 inches)) in the second year, and then 5–6 cm (2.5 inches) each year until puberty. Assuming an average birth length of 51 cm (20 inches), an average 1-y-old is 76 cm (30 inches) long, a 2 y-old is 89 cm (35 inches), a 4-y-old is 102 cm (40 inches), and an 8-y-old is 127 cm (50 inches).

Pubertal growth

Puberty is a dynamic period of development marked by rapid changes in body size, shape, and composition, all of which are sexually dimorphic. The onset of puberty corresponds to a skeletal (biological) age of ≈11 y in girls and 13 y in boys (12). On average, girls enter and complete each stage of pubertal earlier than do boys. The timing and tempo of puberty vary widely, even among healthy children. In determining the appropriateness of a particular growth velocity, the child’s degree of biological maturation must be considered. Skeletal or pubertal maturation may be used to determine the child’s degree of biological development. The bone age is determined as the mean of the skeletal ages of several of the small bones of the hand and wrist. Pubertal maturation status is based on the development of breasts and pubic hair in girls and of pubic hair and genitals in boys. This range of normal variability is expanded to an even greater degree by alterations in energy intake and expenditure. Although moderate activity is associated with cardiovascular benefits and favorable changes in body composition, excessive physical activity during childhood and adolescence may negatively affect growth and adolescent development. Sports that emphasize strict weight control and high energy output—for example, scholastic wrestling, gymnastics, and dancing—are of particular concern for growth disorders, although selection criteria for certain body types make selection bias a confounding variable in assessing the effect of training on growth and adolescent development. One must consider that some of these change are transient, at least in wrestlers. The same markers of growth and body composition that are slowed during training (in season) accelerate after the season, which permits a catch-up process to control growth and cause no permanent growth reductions (see the section “Constitutional delay of growth,” below).

One of the hallmarks of puberty is the adolescent growth spurt. As puberty approaches, growth velocity slows to a nadir (“preadolescent dip”) before its sudden acceleration during midpuberty. The timing of the pubertal growth spurt in girls is typically at Tanner breast stage 3 and does not reach the magnitude
of that in boys. Girls average a peak height velocity of 9 cm/y at age 12 and a total gain in height of 25 cm during the pubertal growth period (13). Boys, on average, attain a peak height velocity of 10.3 cm/y 2 y later than girls, during Tanner genital stage 4, and gain 28 cm in height (9, 13). The longer duration of prepubertal growth in boys, combined with a greater peak height velocity, results in an average adult height difference of 13 cm between men and women. After a period of decelerating height velocity, growth virtually ceases because of epiphyseal fusion, typically at a skeletal age of 15 y in girls and 17 y in boys (4).

Puberty is also a time of significant weight gain; 50% of adult body weight is gained during adolescence. In boys, peak weight velocity occurs at about the same time as peak height velocity and averages 9 kg/y. In girls, peak weight gain lags behind peak height velocity by ~6 mo and reaches 8.3 kg/y at ~12.5 y of age (4). The rate of weight gain decelerates in a manner similar to height velocity during the later stages of pubertal development.

Marked changes in body composition, including alterations in the relative proportions of water, muscle, fat, and bone, are a hallmark of pubertal maturation and result in typical female-male differences. Under the influence of the gonadal steroid hormones and growth hormone (GH), increases in bone mineral content and muscle mass occur and the deposition of fat is maximally sexually dimorphic. The changes in the distribution of body fat (central compared with peripheral, subcutaneous compared with visceral, and upper compared with lower body) results in the typical android and gynoid patterns of fat distribution of the older adolescent and adult (14).

Under the influence of testosterone, boys have a significant increase in growth of bone and muscle and a simultaneous loss of fat in the limbs (4). The maximal loss of fat and increase in muscle mass in the upper arms corresponds to the time of peak height velocity. In boys, the significant increase in lean body mass exceeds the total gain in weight because of the concomitant loss of adipose tissue. As height velocity declines, fat accumulation resumes in both sexes but is twice as rapid in girls. As adults, males have 150% of the lean body mass of the average female and twice the number of muscle cells (15). The increase in skeletal size and muscle mass leads to increased strength in males. Both androgens and estrogens promote deposition of bone mineral, and >90% of peak skeletal mass is present by age 18 y in adolescents who have undergone normal pubertal development at the usual time. In girls, nearly one-third of total skeletal mineral is accumulated in the 3–4-y period immediately after the onset of puberty (16, 17). Adolescents with delayed puberty or secondary amenorrhea may fail to accrue bone mineral normally and have reduced bone mineral density as adults.

During pubertal development, interactions between GH and the sex steroid hormones are striking and pervasive. Studies of adolescent boys showed that the rising concentrations of testosterone during puberty play a pivotal role in augmenting spontaneous secretion of GH and production of insulin-like growth factor I (IGF-I). The ability of testosterone to stimulate pituitary GH secretion, however, appears to be transient and expressed only peripubertally; GH and IGF-I concentrations decrease significantly during late puberty and into adulthood, despite continued high concentrations of gonadal steroid hormones (18). In contrast with testosterone, estrogen modulates GH secretory activity in a disparate manner; low doses of estrogen stimulate IGF-I production through enhanced GH secretion, but higher doses inhibit IGF-I production at the hepatic level (19).

**VARIATIONS OF NORMAL GROWTH**

Normal variants of growth were found in 82% of children whose height decreased at the third percentile (~2 SD) but in only 50% of those whose height decreased at the first percentile (~3 SD) of the mean for age (20). Assessment of skeletal maturation is perhaps the best indicator of biological age or maturity status, because its development spans the entire period of growth. Several methods exist for determining the former (21–23). Each uses a single radiograph of the left hand and wrist and makes comparisons with children of normal stature by using an atlas and scoring system. Because girls are more developmentally mature than boys at any given chronologic age, separate standards exist for females and males.

**Familial short stature**

On average, children of smaller parents will eventually attain lesser height than children of taller parents. Because bone age approximates chronologic age, these children usually grow at an appropriate rate during childhood and attain sexual maturation and pubertal growth spur at the usual ages.

**Constitutional delay of growth**

A constitutional growth delay is considered to be a delay in the tempo of growth. In this case, each calendar year is not accompanied by a full year of growth and skeletal development, so the individual requires more time to complete the growth process. Most of these children will eventually have delayed adolescence as well as delayed attainment of adult stature. Birth history and birth length are generally normal, but the growth pattern shifts downward to the lower percentiles, so that the lowest values for growth velocity are obtained at ~3–5 y of age. Thereafter, this pattern is characterized by steady progression of growth. Because the bone age does not advance 1 y for each calendar year, it progressively deviates from the chronologic age. The height age is usually approximately the same as the bone age, and if true, the mature height will be well within the normal range for the appropriate population. Like familial short stature, this pattern is often familial, and because both are relatively common, some children will have elements of both.

**NUTRITION AND GROWTH**

Worldwide, the single most common cause of growth retardation is poverty-related malnutrition. In the United States, nutritional growth retardation (also known as nutritional dwarfism) and delayed pubertal development among suburban upper-middle- and upper-class adolescents more often result from self-induced restriction of nutrient (energy) intake. In addition to effects on overall growth, malnutrition secondary to avoidance of certain foods or malabsorption can lead to serious disorders, such as osteopenia, anemia, and syndromes related to deficiencies of vitamins, minerals, essential fatty acids and amino acids, and trace elements. Nutritional status also has a significant modulating effect on the timing of adolescent sexual development. Undernutrition is associated with later age at menarche (as well as secondary amenorrhea), whereas a moderate degree of obesity is associated with early sexual maturation (24, 25). The growth curves for length and weight may at first be indistinguishable from those of children and adolescents with constitutional delay of growth (see “Constitutional delay of growth,” above) or, more rarely, from those of children with familial short stature.
The diagnostic criteria for nutritional growth failure follow those of the Wellcome Trust classification. The weight for chronologic age is low, although there may often be a minimal deficit in weight-for-height, as occurs in constitutional delay of growth and adolescence or even familial short stature. Although the specific behaviors required for the diagnosis of anorexia nervosa or bulimia nervosa are absent, there is deteriorating linear growth or delay in adolescent development associated with inadequate weight gain. It appears that a preoccupation with slimness and striving for weight control, fueled by current health beliefs, cause the retarded growth. Single growth points of underweight-for-height are not nearly as important as longitudinal data, because individuals with constitutional thinness may have a weight that is > 2 percentile lines below their height.

Nutritional growth retardation must be differentiated from the variations of normal growth noted above and also from some of the forms of inflammatory and other bowel diseases in which growth failure, often noted by deviation from a previously defined length and weight channel, may be well above the lowest percentiles. Rehabilitation of nutritional growth failure or relief of the inflammation may promote catch-up growth.

The theoretical weight, the weight deficit for that theoretical weight, and the weight-for-height deficit should be defined (Figure 3). This is because children with familial short stature and mild constitutional delay of growth and adolescence most often continue to gain weight. This weight gain is either along an established percentile or slightly below but parallel to the lowest percentile on the chart. The hallmark of nutritional growth delay is that weight progressively deviates from the previous channel, an observation that underscores the importance of gathering longitudinal data.

Children with nutritional growth retardation may have reached a new energy equilibrium phase between their genetically determined growth potential and the present energy intake, because growth deceleration is the adaptive response to suboptimal energy intake. This growth deceleration has limits; for example, energy intake (and often protein) may be inadequate for such a prolonged period of time that energy malnutrition becomes evident. Acutely, suboptimal intake due to illness or heavy exercise load (see the following section) may temporarily delay growth, but this will be followed quickly by catch-up growth. This process must be properly distinguished from other causes of organic and nonorganic growth retardation.

The adaptive response is marked by a decrement in basal (and resting) metabolic rate and a decrease in protein synthesis, the latter being an energy-intensive process. In addition, there may be deficiencies in minerals, particularly zinc and iron, and vitamins. All may lead to a decrease in physical activity, which is an attempt to decrease ongoing energy losses.

**EFFECTS OF PHYSICAL ACTIVITY AND TRAINING ON GROWTH AND ADOLESCENT DEVELOPMENT**

Does physical activity, sport training, or both affect linear growth and pubertal maturation? The literature is replete with reports that the effects of athletic training on growth and pubertal development are salutary, deleterious, or nonexistent [for a review, see Malina (27)]. However, careful appraisal of these reports often reveals severe methodologic faults, such as lack of consideration of interindividual variation in biological maturity status and subject selection. Certain sports show advantages for
the early maturer, especially for males, and others, especially gymnastics and dance, favor the later-developing female. Thus, there is concern about the potential effects of training on the timing and progression (tempo) of puberty “caused” by participation in training and sports. Critical analysis with the biological indicators of bone age or peak height velocity in longitudinal study designs is required to tease out the effects of such training on pubertal development and adult height.

**Females**

Delay in growth and sexual maturation is well documented among certain groups of elite female athletes, most notably gymnasts, dancers, and long-distance runners (28). The underlying mechanisms, however, are not entirely clear, in part because of few longitudinal data in girls. Control of growth and age at menarche involve the complex interaction of many factors, including the physical and metabolic demands of intensive athletic training and competition.

Investigations of growth parameters in adolescent female gymnasts consistently find these girls to be shorter and lighter and to have a significantly lower percentage of body fat than do age-matched control girls or athletes participating in less strenuous sports, such as swimming. Girls participating in the latter types of sports are generally taller and mature earlier than normal (28–31). Theintz et al (29) followed a cohort of adolescent gymnasts and swimmers over an interval of 2–3 y. Training periods averaged 22 h/wk for the gymnasts and 8 h/wk for the swimmers. The gymnasts had significantly lower growth velocities from skeletal age 11–13 y, showing a peak height velocity of only $5.48 \pm 0.32$ cm/y compared with $8.0 \pm 0.50$ cm/y for the swimmers. Over time, height SD scores decreased significantly in the gymnasts without a change in the ratio of chronologic age to bone age. Consequently, predicted heights of the gymnasts decreased with time, but those of the swimmers did not change.

Lindholm et al (32) also observed slower growth velocities among a group of adolescent female gymnasts. These girls did not display the distinct growth spurt seen in the control group of inactive girls, and 27% had adult heights that were less than expected based on midparental height. Bernadot and Czerwinski (33) studied 2 groups of female gymnasts, one aged 7–10 y and the other aged 11–14 y. Weight-for-age and height-for-age decreased from the 48th percentile in the younger group to the 20th percentile in the older gymnasts. Body fat did not differ significantly between the age groups, and at all ages the gymnasts had significantly more muscle mass for their size than did the control group.

Several investigations have compared age at menarche among female athletes participating in different sports with that of the general population. Claessens et al (34) found the median age at menarche to be $15.6 \pm 2.1$ y among a group of gymnasts and $13.2 \pm 1.2$ y among the control population. Theintz et al (29) observed that among a group of gymnasts and swimmers aged $12.7 \pm 1.1$ y, only $7.4\%$ of the gymnasts had experienced menarche, in contrast with $50\%$ of the age-matched swimmers. The gymnasts in this study, however, had a significant delay in skeletal age ($-1.42 \pm 0.99$ y), but the swimmers had comparable chronologic and skeletal ages. This report emphasized the importance of the interaction between somatic growth and sexual maturation and the interpretation of physiologically versus pathologically delayed puberty. Baxter-Jones et al (35) reported the mean ages at menarche of adolescents being intensively trained in gymnastics, swimming, and tennis to be $14.3$, $13.3$, and $13.2$ y, respectively, with a population reference value of $13.0$ y. Significant delay was again noted only among the group of gymnasts. The data for gymnasts are replicated to a lesser degree in dancers and runners. Sports such as swimming, speed skating, and tennis appear to have minimal effects on growth or age at menarche (27, 28, 35).

Although these data suggest a relation between intense athletic training and growth and pubertal development in female gymnasts, they are not conclusive. In interpreting growth and development data of athletes, a host of other variables, including the intensity of training, must also be considered. An individual’s general state of health is critical to normal growth and development, but this is assumed in adolescents who meet the great physical demands of long-term training. Genetic predisposition also plays an important role; the short stature of gymnasts is often familial (28) and a positive correlation has been found between menarcheal age in mothers and daughters (35). Historically, socioeconomic class and family size have been influential; menarche occurs earlier in the higher socioeconomic classes and in families with fewer siblings (4). Psychologic and emotional stressors associated with years-long training, frequent competition, maintenance of low body weight, altered peer relations, and demands of coaches may also influence growth and pubertal timing (28).

Nutrition, especially dieting behavior, can be a major factor for disordered growth, particularly in sports that emphasize strict weight control. Although the principle of a critical percentage of body fat is no longer considered valid, the issue of energy balance is crucial to growth and development. Intake of energy, as well as of vital nutrients such as calcium, which is necessary for bone mineral accrual, may be suboptimal in athletes who restrict dietary intake during a time of increased metabolic demand. Nutter (36) found that the desire to be thin may influence dietary patterns of female athletes even more than do changes in exercise training.

Several investigators, for example, Warren (37), have stressed the importance of strenuous physical training before menarche that might cause disordered growth and adolescent development. Younger children may be especially susceptible to the energy demands of strenuous exercise. Although similar trends depending on type of sport are apparent, menarche is more delayed in gymnasts than in swimmers or tennis players who began training at a comparable age. Prior menstrual irregularity appears to be an important risk factor for oligomenorrhea or amenorrhea in adolescents who begin training after menarche.

Alterations in growth and pubertal maturation are not common among young women engaging in recreational exercise or in adolescents who train <15 h/wk (38). The incidence of oligomenorrhea or amenorrhea and secondary amenorrhea has been cited as 10–40% among athletes and 2–5% among the general population. The distinction between elite and nonelite athletes is important because it pertains to training time and intensity. Olympic athletes have been shown to have significantly later menarche than high school, college, and club-level athletes (27). The different demands of various sports also dictate the amount of time spent in strenuous physical activity; gymnasts and dancers far exceed swimmers and tennis players in the available studies. Catch-up growth has been reported in gymnasts when their training is temporarily reduced or stopped (30).

However, one of the most important variables (perhaps the single most important variable) to take into account is that of selection bias. Body types that are most successful are selected for particular sports. Several studies have reported gymnasts to be smaller than their peers from a young age (27, 28). Delayed
menarche favors the continuation of sports such as gymnastics, which suggests that elite gymnasts are selected in part for this attribute. Continued participation in turn leads to more intense training and blurring of cause and effect.

The implications of delayed menarche are directly relevant to the accrual of bone mineral. Because >90% of the total adult bone mass is established during the pubertal years, failure to accrue bone mineral at a normal rate during this time may result in permanent deficits. Bone mineralization is a complex process influenced by nutrition (especially calcium intake), weight-bearing activity, and sex steroid hormones. Hypoestrogenism because of pubertal delay or secondary amenorrhea can lead to low bone mineral density despite adequate weight-bearing exercise. In a group of female runners, Louis et al (39) found decreases in bone mineral density in all subjects with oligomenorrhea or amenorrhea, whereas runners with regular menses had values within the normal range. A low rate of bone mineral accrual has been suggested as one factor contributing to skeletal injuries in gymnasts.

Males

In general, boys who participate in sports have normal growth rates and are normal or advanced for their state of skeletal and sexual maturation (27). The advanced states of maturation in male athletes may be attributed to the power and performance advantages associated with maturation (40).

However, for sports that may create an energy drain, the effects on growth and maturation remain inconclusive. Seefeldt et al (41) reported that the height velocity of elite male distance runners was equal to nonrunning control subjects during 1 y of training. Other investigations have reported the linear growth of male distance runners to be either slowed or advanced relative to reference data. Unfortunately, the maturity levels of the runners, the reference data, or both were not given for the 2 former studies, so few conclusions can be made with regard to the influence of distance running on growth velocity.

The growth of scholastic wrestlers has also been the concern of several investigations. American wrestlers begin losing weight to certify for lower competitive weight classes as young as 8 y of age. The weight is lost through dieting, severe exercise, dehydration, and various other methods (42, 43), which has produced enough concern to warrant both the American College of Sports Medicine and the American Medical Association to publish position statements calling for the limitation of this practice. In fact, several authors have speculated that the growth of peripubescent wrestlers may be slowed during the sport season. As a group, high school wrestlers are usually shorter than average for their age (27), although this too is probably a self-selection process for wrestling.

In a cross-sectional study, the growth patterns of 477 high school wrestlers were compared with those of a representative sample of adolescent males (44). The wrestler and reference groups were not different at any age for body weight, but the slope value for the gain in body weight was significantly greater for the reference sample. The reference group was significantly taller than the wrestlers after age 16.4 y, but the slope values for gain in height were not statistically different. Slope values were also compared for 13 other anthropometric variables, with few notable group differences. The investigators concluded that wrestling does not slow growth and maturation (44). However, the study did not address whether the growth rate during the sport season was slowed and, if so, whether there was catch-up growth during the nontraining season.

As expected, many investigators have reported reductions in weight, fat mass, and percentage of body fat during the wrestling season (45, 46). However, the fat-free mass is more conserved; most investigators report nonsignificant reductions (45, 46). Still, the fat-free mass does not increase as one would expect for normal pubescent males. Because arm and leg strength diminish (45), one might suggest that statistically insignificant reductions in these variables may be biologically relevant. After the sport season, wrestlers experience accelerated incremental gains in weight, fat mass, and fat-free mass (45, 47). The postseason gains in weight may be above the 99th percentile for age. Accelerated postseason gains in weight, fat mass, and fat-free mass suggest soft-tissue catch-up growth in the wrestlers. During the sport season, changes in anthropometric measures of lean tissue, such as mid-arm girth and lean limb cross-sectional areas (obtained from skinfold corrected girths), also provide evidence that despite heavy bouts of training, wrestlers can fail to accrue lean tissue during the sport season and show an accelerated accrual postseason (45).

SUMMARY

A few compelling data implicate training or competition as causai in the shorter stature and decreased body mass of some pubertal athletes in specific sports. It appears likely that activities such as gymnastics and dance in girls or wrestling in boys select for those participants with desirable genetic anthropometric traits. Added to this process are the interactions among diminished nutrition and the energy drain of training. Preliminary hormonal studies cannot distinguish between constitutionally delayed puberty and a syndrome caused by sport participation. However, studies designed to make this distinction probably cannot be done in adolescents. Investigations in adult women show that some amenorrheic athletes have altered pulsatile gonadotropin release, but it has not yet been possible to separate the effect of the training itself from nutritional and stress factors (48).

REFERENCES


