Chapter 1
Growth and the Young Female Athlete

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Introduction

The body goes through remarkable changes from birth through childhood and adolescence into adulthood. Biological growth and maturation, along with behavioral development, are important aspects "growing up." These three processes, growth, maturation, and development, dominate the daily lives of children and adolescents for approximately the first two decades of life. This chapter will focus predominantly on growth and maturation, and how timing and tempo of these may affect sports involvement and injury risk in young female athletes.

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C.J. Stein et al. (eds.), *The Young Female Athlete*, Contemporary Pediatric and Adolescent Sports Medicine, DOI 10.1007/978-3-319-21632-4_1

Definitions

Growth: the increase in the size of the body as a whole and of its parts. As children grow, height and body mass increase. The latter includes changes in skeletal, muscle and fat mass, as well as in bodily organs. Different segments of the body grow at different rates and at different times, resulting in altered body proportions.

Maturation: the progress towards biological maturity. Maturation is a process, while maturity is a state. Maturation occurs in all bodily organs and systems, but maturity varies with each biological system considered. Sexual maturity is fully functional reproductive capability. Skeletal maturity is a fully ossified adult skeleton. Maturation of the nervous and endocrine systems is a major factor underlying sexual, skeletal, and somatic growth and maturation.

Timing and tempo of growth and maturation: Timing refers to the chronological age at which specific events or milestones in growth and maturation occur, while tempo refers to the rate at which the processes of growth and maturation progress. Both timing and tempo vary considerably among individuals.

Development: a psychological, emotional, and behavioral process often subsumed in the term socialization, which is specific to a culture.

Indicators and Patterns of Growth

The study of growth and maturation is based on standardized measurements and observations, which reflect underlying biological and neuroendocrine changes. Time between birth and adulthood has been formally subdivided by Karlberg into phases of normal growth: infancy, childhood, and puberty [1, 2]. During infancy (birth to 6–12 months), growth is rapid but progresses at a decelerating rate. Growth curves often cross percentile lines as the infant moves farther away from the excesses or constraints of the intrauterine environment and toward her genetic potential. The next phase is childhood, during which growth and development are primarily dependent on adequate nutrition, an appropriate psychosocial environment, and the absence of disease. In the context of these environmental factors, one must have sufficient levels of thyroid hormone and insulin-like growth factor 1 (IGF-1), the stable pharmacodynamic marker of growth hormone (GH), to grow normally. Growth rate continues to decelerate until around age 3 years, and then continues at a relatively constant velocity of 4.5–7.0 cm/year and 2.5 kg/year for girls and boys. Growth rate often slows slightly just prior to adolescence [1, 2].

The hypothalamic–pituitary–gonadal (HPG) axis is virtually quiescent until late childhood, when activation from the secretion of estrogen from the ovaries in girls (testosterone from the testes in boys) occurs, well before the outward signs of pubertal development become apparent. The pubertal phase is characterized by a growth spurt of 8–14 cm/year because of the synergistic effects of increasing gonadal steroids and GH secretion [1, 2]. The upper limit of middle childhood is arbitrary given variations in the onset of puberty; some girls as young as 9–10 years old are already in the early stage of puberty/adolescence. The termination of adolescence is also quite variable. Biologically, some girls are sexually mature by 12–13 years of age (i.e., they are biologically adult), even though socially and behaviorally, they remain adolescents. Although chronological age is a commonly used reference point, and children are often divided into age groups, there is great variability among individuals of the same chronological age, especially around the time of puberty [3].

Body Size and Composition

Height and weight are the body dimensions most commonly used to monitor growth status and growth rate. Length is commonly used in place of height during the first 2–3 years of life.

Height

Height in most children is measured yearly, and growth charts represent "smoothed" growth data. However, linear growth is actually episodic with stepwise jumps, as noted by daily measurements over time in both newborns and adolescents [4]. Although less common in girls than in boys, some children show a minor growth spurt between ages 6 and 8. By 9–10 years of age, the rate of height growth accelerates in girls, marking the beginning of the adolescent spurt. From the onset of this growth spurt, the rate of growth in height increases until it reaches its peak [peak height velocity (PHV)], which generally occurs at about 12 years of age in girls [5] and 14 years of age in boys [5].

Children continue to grow in height through adolescence, but after the growth spurt, the rate of growth gradually decreases and eventually reaches zero as individuals attain their adult stature. The timing of each of these events—the growth spurt, PHV, and termination of growth—is variable among individuals.

Weight

Weight, like height, increases throughout childhood, but the rate of increase changes during different phases of growth, decelerating from birth through the second year of life, and then accelerating as the child ages. A growth spurt is also observed in body weight, which generally begins slightly later than that of height. Average age of peak weight velocity in girls is about half a year after PHV. Weight continues to increase into the late teens and early 20s, and unlike height, can continue beyond skeletal maturity [5].

Body Composition

Body weight is a composite of all bodily tissues, but it is often described in terms of its lean (fat-free) and fat components. Thus, body weight = fat-free mass (FFM) + fat mass (FM). Major components of FFM are skeletal muscle and bone mineral. FFM has a growth pattern similar to that of height and weight and demonstrates a clear adolescent spurt. FM increases more gradually during childhood and adolescence. Most other body dimensions (sitting height, leg length, limb circumferences, skeletal breadths) and many major organs follow growth patterns similar to height and weight, although growth rates and the timing of adolescent spurts vary relative to age at PHV [5]. Differential growth rates in specific dimensions influence body proportions and regional distribution of body fat [5, 6].

Body composition changes throughout life, but striking transformations are evident at puberty, resulting from sex-dependent changes in hormones and cytokines, including increases in GH, IGF-1, and reproductive hormone concentrations. GH secretion increases by a similar magnitude in both boys and girls during puberty, but IGF-1 levels are consistently higher in girls, in pre-pubertal and pubertal phases [7, 8]. While reproductive hormones act directly on growth plates of bones, independent of GH, a majority of the sex-related differences in growth and body composition are likely mediated by the effects of reproductive hormones on the GH–IGF-1 axis [8, 9]. For example, in boys, the interactions of testosterone, GH, and IGF-1 together enhance the increase in muscle and loss of fat, leading to the more muscular configuration of the young adult male. In contrast, in pubertal girls, the increase in estrogen attenuates GH secretion and IGF-1, leading to a slowing in the accrual of muscle and an enhancement of fat gain, especially in the gynoid distribution [8].

Bone mineral density (BMD) increases steadily from childhood to adolescence, with a decrease in bone mass relative to bone size before PHV. Peak velocity of bone mineral content occurs about 6 months later than PHV. A majority of bone mass and size are acquired by late adolescence, followed by small increases thereafter [10]. See Chapter 5 for additional details.

Physical Performance

Physical performance is generally measured with standard tests of strength, speed, power, agility, endurance and flexibility, and in the context of sport, with sport-specific skills tests. Sex differences in performances are relatively small during childhood, but are magnified during adolescence as performances of girls typically reach a plateau or improve only slightly. Isometric (static) strength, for example, generally increases in a linear fashion through childhood for both boys and girls. Around age 13, males experience an adolescent spurt and an increase in the rate of strength development, while females continue to experience a linear increase until around age 15. Nevertheless, limited longitudinal data for girls indicate a growth spurt in static strength that is about one-half the magnitude of that noted for boys. The spurt generally occurs after PHV [3].

Performance on flexibility tasks tends to decline from childhood through mid-adolescence and then increases. Flexibility in girls usually remains stable or decreases slightly during childhood, increases during adolescence, and plateaus at 14–15 years of age [3, 5, 11, 12]. In general, flexibility tends to be greater in girls than boys.

An adolescent spurt occurs in absolute aerobic power, VO_2max or peak VO_2, in both boys and girls. Peak VO_2 generally increases in boys from childhood through adolescence, but reaches a plateau in girls by 13–14 years. VO_2max starts to increase several years before PHV and continues to increase after. However, due to changes in height and mass, VO_2max per unit body mass actually begins to decline 1 year before PHV and continues after [3].

Indicators and Measurements of Maturation

Maturity Status

Maturity status and progress are traditionally monitored in the skeleton and secondary sex characteristics. Radiographs of the hand and wrist can be used to estimate skeletal maturity using the standards of Greulich and Pyle [13]. This assessment of skeletal maturation, along with current height, can be used to predict adult or mature height. Sexual maturation is based on the development of secondary sex characteristics; in girls, breast bud development (thelarche) is typically the first physically apparent sign of sexual maturation, followed by the appearance of pubic hair (pubarche).

Tanner described five stages of maturation [14]. Stage 1 (pre-pubertal) indicates lack of overt manifestation of the development of breasts and pubic hair; however, pre-pubertal children of the same chronological age can vary in skeletal age by 4 years or more [5]. Stage 2 marks the onset of puberty, typically starting around a bone age of 11 years in girls, while advancement in breast development and pubic hair distribution in stages 3 and 4 mark progress in puberty. Stage 5 is the mature state [15]. It is important to note that stages have limitations; they provide no information on when the stage was reached (timing) or how long the child has been in the stage (tempo).

Maturity Timing

Ages at PHV and the beginning of menstrual periods (menarche) are indicators of maturity timing. Menarche typically occurs after PHV and on average, 2.6 years after the onset of puberty [16]. The average age of menarche in the United States has declined over the last century, and is now around 12.3 years of age [17]. There are various influences on timing of puberty, including nutrition, race, ethnicity, geography, and other factors.

Measurement of Maturation

Measuring maturation can be challenging. Established indicators used in growth studies and in clinics have limitations: skeletal age determination with X-rays exposes children to low-dose radiation and is dependent on experienced individuals to interpret the films; directly observing secondary sex characteristics is invasive of personal privacy; age at PHV is an "after-the-fact" indicator; and surveys questioning age at menarche, breast bud development, and pubic hair growth are affected by recall accuracy. Other non-invasive estimates using percentage of predicted adult stature at the time of observation and predicted maturity offset (time before PHV) have also been suggested to estimate maturity status and timing, respectively, in youth athletes [18, 19]. However, these too have limitations.

Growth in the Athlete

It is obvious when observing athletes in a variety of sports that different physical factors offer competitive advantages. In some cases, such as taller average height in basketball players, these differences are clearly the result of selection, due to the fact that certain athletes enter or succeed in the sport while others either do not participate or do not progress. However, in other cases, like shorter average height in gymnasts, it has been more difficult to tease out the impact of selection and training. Even when patterns are recognized, causality may be difficult to establish, especially if data are cross-sectional rather than prospective. Despite these limitations, current research indicates that sports training and participation do not appear to affect adult stature or overall rate of growth [3, 20, 21]. In addition, sports training alone does not likely affect skeletal or sexual maturation [3].

Growth data for young female athletes are limited largely to height and weight [21–24]. Although there is variation among sports, female athletes in most sports tend to have heights similar to or greater than the median of U.S. reference data from childhood through adolescence, and weights that are appropriate for their height [3]. Notable exceptions include artistic gymnasts and figure skaters, who are generally shorter than their peers [21, 25]. Young female athletes also tend to be leaner, with lower weight-for-height, compared to non-athletes [3]. Ballet dancers and distance runners tend to have lower weight-for-height, while participants in field events and higher weight categories in weightlifting often have excess weight-for-height [3, 26].

The available height and weight data for young female athletes span decades. There is a need to consider changes in sport rules, training, selection, and other factors when examining small subsets of athletes at different time points. For example, between the 1960s and 1997, the minimum age for female artistic gymnastics to compete internationally was increased from 13 to 16 years. While the average age of participants has increased, heights and weights have changed little from 1987 through 2008 [21], suggesting preferential selection for shorter, leaner athletes. In a study of body size in elite junior rowers (ages 15–18 years) competing at the World Championships in 1997 versus 2007, female rowers in 2007 were on average 2.1 cm taller [27]. This may simply reflect recruitment of taller athletes to a sport in which height is a competitive advantage.

The BMI is currently the most commonly used index of weight-for-height. Although a great deal of focus is on overweight and obesity in the non-athlete population, low BMI often gets more attention among female athletes. Three grades of low weight-for-height (labeled mild, moderate, and severe thinness) among children and adolescents have been described. The term thinness was selected to avoid confusion with other labels commonly used to define low weight-for-height (e.g., wasting and underweight) in children. Three standard growth curves have been designed to pass through a BMI of 16, 17, and 18.5 at age 18. These points are, respectively, the cut-offs used to define severe, moderate, and mild thinness in adults. Thus the specific BMI cut-off at each age prior to 18 years depends on the standard curves for each grade of thinness [28]. In a sample of over 1000 female artistic gymnasts, figure skaters, divers, distance runners and ballet dancers spanning childhood through young adulthood, mild thinness was reasonably common among female athletes, but prevalence varied by sport and level of competition [29].

On average, female gymnasts are of lower weight than a reference population; however, their weight is appropriate for their diminished height. Some gymnasts are indeed at low weight-for-height. In an evaluation of ages, heights, and weights as reported in the official program of 60 participants 15–20 years of age at the 2008 Beijing Olympic Games, 23 (38 %) were classified as mildly thin and 6 (10 %) were classified as moderately thin; none were severely thin [29]. Allowing for the limited data, some female artistic gymnasts may be at risk for mild or moderate thinness, but the risk is related in part to later maturation. Later maturing girls, on average, tend to have less weight-for-height [5].

Maturation in the Young Female Athlete

There is great interest in understanding how the stages of growth and maturation can be best utilized in sport development programs. However, the available data are extremely limited, and additional studies are needed. The popular Long Term Athlete Development model, a concept of planned, systematic, and progressive training of an athlete from childhood onward, calls for the determination of the time of PHV as a potentially sensitive period for training [30]. As noted earlier, the anthropometric protocol for predicting maturity offset and age at PHV has major limitations.

For male athletes in most sports, there appears to be a competitive advantage to being advanced or at least average in maturity status [3, 31]. For female athletes, the picture is less clear and varies by sport. Girls involved in sports such as basketball, volleyball, rowing, swimming and track, except for some distance runners, generally have a pattern of average growth and maturity [32, 33]. Female athletes typically reach menarche within the normal age range seen in the general population.

However, within this range, gymnasts, ballet dancers, and figure skaters tend to start menstruating later than other athletes and non-athletes [3].

Both male and female gymnasts have been noted to have later ages at PHV, and female gymnasts have been noted to have later maturation in terms of breast and pubic hair development, compared to age-matched controls [3, 34]. In our recent review of the literature, we found that despite the fact that gymnasts are generally shorter than their peers, as well as later and slower in growth, gymnastics training does not appear to affect adult stature [21] nor does such training impact growth rate, timing, or tempo. Data in male gymnasts suggest that observed shorter stature is a result of selection rather than training [20].

Girls who trained in rowing, track, or swimming for approximately 12 h per week for an average of 4 years during puberty and the growth spurt were found to have a slightly later PHV and age of menarche than girls inactive in sport, but the differences were not significant. The interval between PHV and menarche, PHV (cm/year), ages at attaining pubic hair and Tanner breast stages 3, 4 and 5, as well as estimated intervals between adjacent stages were also not different in the athletes versus non-athletes [24]. Studies in gymnasts and ballet dancers have found a 2- to 3-year delay in menarche, as well as oligomenorrhea and secondary amenorrhea, likely secondary to undernutrition disturbing neuroendocrine function [35, 36]. Thus, there is a need for more well-designed studies, accounting for energy availability, using validated non-invasive indicators of maturity status and timing, to better determine the effects of sport involvement alone on maturity. It is also essential to account for the selectivity of sport, specifically differential persistence and dropout, either voluntary or systematic, as in getting "cut" from the team. Data for artistic gymnasts indicate size and maturity differences between those who persist and do not persist in the sport [21].

Body Composition in the Young Athlete

Methods for assessing body composition of athletes in general [37] and of youth athletes [38] are diverse and include densitometry, whole body potassium counting (TBK), total body water (isotope dilution, hydrometry), dual energy X-ray absorptiometry, bioelectrical impedance analysis, magnetic resonance imaging, and skinfold thickness, among others [26, 39].

Allowing for variation associated with sampling and methodology, several trends are apparent in the relative fatness of youth female athletes in several sports. Young female athletes tend to have a lower %Fat than non-athletes of the same chronological age, and overlap among samples of athletes in different sports is considerable. Most estimates based on skinfolds are below the reference range, but a number are at and/or above the reference range compared to estimates based on densitometry and hydrometry. Elevated %Fat is more common in athletic field events (specifically throwers) and several samples of team sport athletes [38, 39].

Training Effects on Body Composition

Studies of the influence of athletic training on body composition have traditionally focused on changes in FFM and FM [40], while more recent studies have focused on bone mineral content (BMC), BMD, and bone microarchitecture [41, 42]. Studies of youth are confounded by difficulties in isolating those changes associated with normal growth and maturation, especially the adolescent spurt and sexual maturation, from those attributed to training [43].

There is a need to study changes in the body composition of youth athletes during the course of a competitive season to better understand the potential impact of these changes on performance; however, few data are available. Studies of changes in FFM and/or %Fat in late adolescence and young adulthood provide some insights. The studies generally compare pre- and post-training means, while duration, intensity, and frequency of training are often variable. One review examined studies of female athletes between 18 and 22 years of age and found that the differences between pre- and post-training means ranged from −1.7 to +1.5 kg for FFM (overall mean +0.3 kg), and from −2.1 to +3.1 % for %Fat (overall mean −0.4 %) [44]. These observed changes are relatively small and may fall within the range of measurement variability. Moreover, it is not clear if these changes associated with training persist, because this information is not typically reported.

Currently, there is considerable interest in the influence of regular sport training on bone health in young athletes. BMD typically doubles between the onset of puberty and young adulthood [45]. While genetic factors greatly affect bone mass, other factors such as nutrition, types of exercise, diseases, medicines, age of menarche, and menstrual regularity also significantly influence bone accrual [46]. Beneficial effects of weight-bearing sports training have been observed in cortical thickness, BMC, and BMD [47–49]. Potential confounding factors are selection for sport and limited control of biological maturity status. For example, athletes and controls are often described as pre-pubertal, although skeletal age can vary by as much as 4 years in pre-pubertal children [5]. BMC and BMD are, on average, greater in weight-bearing female athletes compared to the general population of female youth [48]. Limited longitudinal data indicate training-associated increases in BMC and/or BMD in youth athletes. The long-term effect of sport training on bone is especially apparent in the dominant versus non-dominant arms of racquet sport athletes, highlighting localized increases in bone mineral accretion [50, 51]. Effects of decreased energy availability, as seen in weight-restricted athletes and those with eating disorders, can negate the beneficial effect of weight-bearing [42, 52] (see Chapter 5). It is also interesting to note that nonimpact sports such as swimming, water polo, and cycling during youth and young adulthood are not associated with enhanced BMC or BMD, and swimming may even negatively influence hip bone geometry [53].

Effects of Puberty on Sports Training and Performance

It is not yet well understood how the various changes associated with growth, maturation, and development affect athletic performance, but it is clear that puberty is accompanied by many challenges for the young female athlete, including changes in body size and composition, increased rates of injury, and emotional changes surrounding puberty. These changes, compiled with societal pressures, may contribute to the higher drop-out rate from athletics seen in girls during middle school and high school [54, 55]. During puberty, girls may find it difficult to adapt to their changing bodies and the alterations in their athletic abilities. They may become frustrated by the normal fat gain, breast development, hip widening, and height increases, and they may see at least a temporary decline in performance. These adjustments may be particularly evident for those aesthetic sports such as dance, figure skating, gymnastics, and diving, and can derail a young athlete's confidence [56].

As discussed earlier, performance on various standardized tests typically improves throughout adolescence in boys. However, in girls, performance generally improves until about ages 13–14 years (slightly later in some motor tasks), with little subsequent improvement. Whereas males typically experience an increase in the rate of strength development starting around age 13, females generally demonstrate only a linear increase until around the age of 15. Nevertheless, some girls and particularly athletes often continue to improve in performance through adolescence. This improvement is due to regular activity and probably sport-specific training. Historically, research focused on the performance of adolescent female athletes has not received the attention given to male athletes, though the situation is changing [57, 58].

In addition to the physiologic and emotional challenges that affect young athletes during adolescence, injuries also increase during this time. One study of peripubertal gymnasts demonstrated increased injury rates during periods of rapid growth [59]. Anterior cruciate ligament (ACL) injury rates increase dramatically around ages 12–13 in girls, and female athletes 15–20 years of age account for the largest number of ACL injuries reported [60, 61]. More common in females than males, the difference in ACL injury rates becomes apparent around the time of the growth spurt, peaks during adolescence, and declines in young adulthood [61–63]. This discrepancy is likely multifactorial: during PHV, rapid lengthening of the tibia and femur leads to greater torque at the knee, increasing height results in a higher center of gravity, which requires more challenging muscular control, and increases in weight cause more joint force that is difficult to control during high velocity movements [61, 62]. Unlike boys, whose pubertal increase in testosterone aids in accumulating muscle mass, pubertal girls have difficulty building muscle, making joint control even more difficult [62]. See Chapter 9 for additional information on ACL injuries.

Around PHV, adolescents are more prone to injury because of imbalances of strength and flexibility and changes in biochemical properties of bone [64]. There is a dissociation during puberty between peak BMC and bone mineral area; bones increase in size more rapidly than they fill in with mineral. BMD decreases before and rebounds after the period of PHV. Thus there is a time of relative skeletal weakness during adolescent growth, with a temporary increase in fracture rate [10]. Also of concern are growth plate injuries. Tendons and ligaments of the growing athlete are relatively stronger than the epiphyseal growth plate. Thus, during sports trauma, such as an ankle inversion, the epiphysis is more likely to be damaged than the ligamentous complex (e.g. fibular growth plate injury rather than a ligamentous tear).

Summary and Conclusions

The human body goes through remarkable transformations throughout the life stages. As young athletes grow and develop, they must also adapt. For sports and medical professionals, awareness of general patterns of growth and maturation allows for improved recognition of the advantages, risks, and challenges that accompany these changes. We can then better support the young athlete as she matures. With girls and young women increasingly involved in intense sports training and participation, additional prospective studies are needed to enhance our understanding of normal variations in growth and maturation and the ways in which they influence and are impacted by sports training and performance.

References

1. Karlberg J, et al. Analysis of linear growth using a mathematical model. I. From birth to 3 years. Acta Paediatr Scand. 1987;76(3):478–88.
2. Karlberg J. A biologically-oriented mathematical model (ICP) for human growth. Acta Paediatr Scand Suppl. 1989;350:70–94.
3. Beunen G, Malina RM. Growth and biologic maturation: relevance to athletic performance. In: Hebestreit H, Bar-Or O, editors. The young athlete. Oxford, UK: Blackwell Publishing Ltd; 2007.
4. Lampl M, Veldhuis JD, Johnson ML. Saltation and stasis: a model of human growth. Science. 1992;258(5083):801–3.
5. Malina R, Bouchard C, Bar-Or O. Growth, maturation and physical activity. 2nd ed. Champaign, IL: Human Kinetics Press; 2004.
6. Malina RM. Variations in body composition associated with sex and ethnicity. In: Heymsfield SB et al., editors. Human body composition. Champaign, IL: Human Kinetics; 2005. p. 271–98.
7. Lofqvist C, et al. Reference values for insulin-like growth factor-binding protein-3 (IGFBP-3) and the ratio of insulin-like growth factor-I to IGFBP-3 throughout childhood and adolescence. J Clin Endocrinol Metab. 2005;90(3):1420–7.
8. Casazza K, Hanks LJ, Alvarez JA. Role of various cytokines and growth factors in pubertal development. Med Sport Sci. 2010;55:14–31.
9. Meinhardt UJ, Ho KK. Regulation of growth hormone action by gonadal steroids. Endocrinol Metab Clin North Am. 2007;36(1):57–73.
10. Faulkner RA, et al. Size-corrected BMD decreases during peak linear growth: implications for fracture incidence during adolescence. J Bone Miner Res. 2006;21(12):1864–70.

11. Haubenstricker JL, Seefeldt VD. Acquisition of motor skills during childhood. In: Seefeldt V, editor. Physical activity and well-being. Reston, VA: AAHPERD; 1986. p. 41–102.
12. Beunen GP, Simons J. Physical growth, maturation and performance. In: Simons J et al., editors. Growth and fitness of Flemish girls: the Leuven growth study. Champaign, IL: Human Kinetics; 1990. p. 69–118.
13. Greulich WW, Pyle SI, Brush Foundation Cleveland. [from old catalog], Radiographic atlas of skeletal development of the hand and wrist. Stanford: Stanford University Press; 1950. p. xiii. 190 p.
14. Tanner JM. Growth at adolescence, with a general consideration of the effects of hereditary and environmental factors upon growth and maturation from birth to maturity. 2nd ed. Oxford: Blackwell Scientific Publications; 1962. 325 p.
15. Tanner JM, et al. Prediction of adult height from height, bone age, and occurrence of menarche, at ages 4 to 16 with allowance for midparent height. Arch Dis Child. 1975;50(1): 14–26.
16. Biro FM, et al. Pubertal correlates in black and white girls. J Pediatr. 2006;148(2):234–40.
17. Anderson SE, Must A. Interpreting the continued decline in the average age at menarche: results from two nationally representative surveys of U.S. girls studied 10 years apart. J Pediatr. 2005;147(6):753–60.
18. Mirwald RL, et al. An assessment of maturity from anthropometric measurements. Med Sci Sports Exerc. 2002;34(4):689–94.
19. Malina RM, et al. Maturity status of youth football players: a noninvasive estimate. Med Sci Sports Exerc. 2005;37(6):1044–52.
20. Daly RM, et al. Short stature in competitive prepubertal and early pubertal male gymnasts: the result of selection bias or intense training? J Pediatr. 2000;137(4):510–6.
21. Malina RM, et al. Role of intensive training in the growth and maturation of artistic gymnasts. Sports Med. 2013;43(9):783–802.
22. Malina RM. Physical growth and biological maturation of young athletes. Exerc Sport Sci Rev. 1994;22:389–433.
23. Eisenmann JC, Malina RM. Growth status and estimated growth rate of young distance runners. Int J Sports Med. 2002;23(3):168–73.
24. Geithner CA, Woynarowska B, Malina RM. The adolescent spurt and sexual maturation in girls active and not active in sport. Ann Hum Biol. 1998;25(5):415–23.
25. Leone M, Lariviere G, Comtois AS. Discriminant analysis of anthropometric and biomotor variables among elite adolescent female athletes in four sports. J Sports Sci. 2002;20(6): 443–9.
26. Malina RM. Growth and maturation of child and adolescent track and field athletes: Final report, Monaco; 2004.
27. Rakovac M, et al. Body size changes in elite junior rowers: 1997 to 2007. Coll Antropol. 2011;35(1):127–31.
28. Cole TJ, et al. Body mass index cut offs to define thinness in children and adolescents: international survey. BMJ. 2007;335(7612):194.
29. Malina RM, Rogol AD. Sport training and the growth and pubertal maturation of young athletes. Pediatr Endocrinol Rev. 2011;9(1):441–55.
30. Balyi I, Way R, Higgs C. Long-term athlete development. Champaign, IL: Human Kinetics; 2013.
31. Malina RM. Skeletal age and age verification in youth sport. Sports Med. 2011;41(11): 925–47.
32. Malina RM. Physical activity and training: effects on stature and the adolescent growth spurt. Med Sci Sports Exerc. 1994;26(6):759–66.
33. Malina RM, Bielicki T. Retrospective longitudinal growth study of boys and girls active in sport. Acta Paediatr. 1996;85(5):570–6.
34. Bernink MJ, et al. Height, body composition, biological maturation and training in relation to socio-economic status in girl gymnasts, swimmers, and controls. Growth. 1983;47(1):1–12.
35. Georgopoulos NA, et al. Growth, pubertal development, skeletal maturation and bone mass acquisition in athletes. Hormones (Athens). 2004;3(4):233–43.

36. Georgopoulos NA, et al. The influence of intensive physical training on growth and pubertal development in athletes. Ann N Y Acad Sci. 2010;1205:39–44.
37. Malina RM. Body composition in athletes: assessment and estimated fatness. Clin Sports Med. 2007;26(1):37–68.
38. Malina RM, Geithner CA. Body composition of young athletes. Am J Lifestyle Med. 2011;5:262–78.
39. Wells JC, Fewtrell MS. Measuring body composition. Arch Dis Child. 2006;91(7):612–7.
40. Peltenburg AL, et al. Biological maturation, body composition, and growth of female gymnasts and control groups of schoolgirls and girl swimmers, aged 8 to 14 years: a cross-sectional survey of 1064 girls. Int J Sports Med. 1984;5(1):36–42.
41. Christo K, et al. Bone metabolism in adolescent athletes with amenorrhea, athletes with eumenorrhea, and control subjects. Pediatrics. 2008;121(6):1127–36.
42. Ackerman KE, et al. Bone microarchitecture is impaired in adolescent amenorrheic athletes compared with eumenorrheic athletes and nonathletic controls. J Clin Endocrinol Metab. 2011;96(10):3123–33.
43. Iuliano-Burns S, Mirwald RL, Bailey DA. Timing and magnitude of peak height velocity and peak tissue velocities for early, average, and late maturing boys and girls. Am J Hum Biol. 2001;13(1):1–8.
44. Wilmore JH. Body composition in sport and exercise: directions for future research. Med Sci Sports Exerc. 1983;15(1):21–31.
45. Katzman DK, et al. Clinical and anthropometric correlates of bone mineral acquisition in healthy adolescent girls. J Clin Endocrinol Metab. 1991;73(6):1332–9.
46. Fehily AM, et al. Factors affecting bone density in young adults. Am J Clin Nutr. 1992;56(3):579–86.
47. Ferry B, et al. Bone health during late adolescence: effects of an 8-month training program on bone geometry in female athletes. Joint Bone Spine. 2013;80(1):57–63.
48. Maimoun L, et al. Peripubertal female athletes in high-impact sports show improved bone mass acquisition and bone geometry. Metabolism. 2013;62(8):1088–98.
49. Burt LA, et al. Skeletal adaptations associated with pre-pubertal gymnastics participation as determined by DXA and pQCT: a systematic review and meta-analysis. J Sci Med Sport. 2013;16(3):231–9.
50. Ducher G, et al. Effects of repetitive loading on the growth-induced changes in bone mass and cortical bone geometry: a 12-month study in pre/peri- and postmenarcheal tennis players. J Bone Miner Res. 2011;26(6):1321–9.
51. Kontulainen S, et al. Good maintenance of exercise-induced bone gain with decreased training of female tennis and squash players: a prospective 5-year follow-up study of young and old starters and controls. J Bone Miner Res. 2001;16(2):195–201.
52. Nattiv A, et al. American College of Sports Medicine position stand. The female athlete triad. Med Sci Sports Exerc. 2007;39(10):1867–82.
53. Tenforde AS, Fredericson M. Influence of sports participation on bone health in the young athlete: a review of the literature. PM R. 2011;3(9):861–7.
54. Kimm SY, et al. Decline in physical activity in black girls and white girls during adolescence. N Engl J Med. 2002;347(10):709–15.
55. Dumith SC, et al. Physical activity change during adolescence: a systematic review and a pooled analysis. Int J Epidemiol. 2011;40(3):685–98.
56. Naughton G, et al. Physiological issues surrounding the performance of adolescent athletes. Sports Med. 2000;30(5):309–25.
57. Vescovi JD, et al. Physical performance characteristics of high-level female soccer players 12–21 years of age. Scand J Med Sci Sports. 2011;21(5):670–8.
58. Martinez-Lagunas V, Niessen M, Hartmann U. Women's football: player characteristics and demands of the game. J Sport Health Sci. 2014;3:258–72.
59. Caine D, et al. An epidemiologic investigation of injuries affecting young competitive female gymnasts. Am J Sports Med. 1989;17(6):811–20.

60. Granan LP, et al. The Scandinavian ACL registries 2004–2007: baseline epidemiology. Acta Orthop. 2009;80(5):563–7.
61. Renstrom P, et al. Non-contact ACL injuries in female athletes: an International Olympic Committee current concepts statement. Br J Sports Med. 2008;42(6):394–412.
62. LaBella CR, et al. Anterior cruciate ligament injuries: diagnosis, treatment, and prevention. Pediatrics. 2014;133(5):e1437–50.
63. Stracciolini A, et al. Anterior cruciate ligament injuries in pediatric athletes presenting to sports medicine clinic. A comparison of males and females through growth and development. Sports Health. 2015. doi:10.1177/1941738114554768.
64. Maffulli N, et al. Aetiology and prevention of injuries in elite young athletes. Med Sport Sci. 2011;56:187–200.