LEADING ARTICLE

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Physiological Issues Surrounding the Performance of Adolescent Athletes

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Abstract

More than ever, many young athletes are being encouraged to train intensely for sporting competitions from an early age. Compared with studies in adults, less is known about the physiological trainability of adolescents. The velocity of physical growth during the adolescent years makes research with a group of young athletes particularly difficult. The purpose of this review is to discuss a number of physiological issues that surround the performances of the adolescent athlete. Research has highlighted the role of growth hormone (GH) in the abrupt acceleration of linear growth that occurs during adolescence. In addition, GH has been shown to be sensitive to exercise following short term intervention studies. The reduced anaerobic power of the adolescent athlete compared with that of an adult athlete has been attributed to the intrinsic properties of the muscle that are yet to be fully understood. Resistance training studies in male adolescents, and to a lesser extent female adolescents, highlight the substantial relative strength gains that can be obtained. Aerobic trainability in young boys appears to improve markedly during the adolescent years. One of the most plausible explanations for this observation is the 'trigger hypothesis' which links increased aerobic improvements in adolescence with hormonal changes and substantial growth of the cardiorespiratory and musculoskeletal systems. Studies of aerobic trainability in adolescent girls are too scarce to be conclusive. An understanding of the impact of long term intensive training on adolescent athletes is difficult to ascertain because physical stresses vary both between and within sports. There is, however, limited evidence to suggest that 'intense' training does not impair normal growth, development or maturation. Adolescent athletes who experience rapid growth as well as large increases in training volumes may be vulnerable to overuse injuries.

Adolescents with exceptional sporting ability generate a great deal of public interest. This is exemplified in the international achievements of young athletes such as Martina Hingis (Switzerland) in tennis and Ian Thorpe (Australia) in swimming. However, there are concerns about the physiological limitations associated with sporting performances of adolescents. Compared with training studies in adults, relatively less is known about the trainability of adolescents. Yet adolescence is a stage of development characterised by unprecedented physiological changes in the musculoskeletal, cardiorespiratory and reproductive systems of the body. To date, there is uncertainty surrounding the effects that aerobic and anaerobic performances have on the growth and sexual maturation of young athletes in different sports. Changes in maturational status during the adolescent period make the interpretation of research with this group of individuals particularly difficult. The purpose of this article is to review some of the physiological issues that influence research on the performance of adolescent athletes.

1. Puberty

1.1 Physical Changes at Puberty

Puberty is denoted by the development of secondary sex characteristics, the marked acceleration in linear growth and alterations in body composition.[1] Secondary characteristics appear at a mean age of 10.5 years in girls and 11.5 to 12 years in boys. Puberty is considered precocious if these changes are noted before 8 years of age in girls and 9 years of age in boys, and is considered delayed when such changes do not occur before 13 year of age in the former group and 14 years of age in the latter group.[2]

Five stages of puberty from childhood to full maturity (P1 to P5) have been described by Tanner.[3] In girls, these stages reflect the progressive modification of the breasts under the control of estrogens which are secreted by the ovaries, and the growth of pubic and axillary hair secondary to androgen secretion by the adrenal cortex. Genital organs develop progressively until stage P4, when the first menstruation (menarche) occurs, at a mean age of 12.5 to 13 years. In boys, growth of the testes is commonly the first sign of puberty. The growth and maturation of the penis usually correlate with pubic hair development under the control of androgen. Other major physical changes in boys include facial, body and axillary hair and lowering of the voice pitch.

The year of greatest height gain occurs at an earlier stage of puberty in girls (breast stage 2 to 3) than boys (testes stage 3 to 4). The relatively earlier growth in pubescent females than males is supported by a recent report on bone mass accretion during pubertal stages. Cheng et al.[4] reported that bone mass accretion was higher in girls who were at a less advanced stage of puberty than their female peers. In contrast, bone mass accretion was higher in boys who were more advanced in their pubertal status. Peak height velocity occurs at approximately 11 and 13 years of age, respectively, for girls and boys,[5] and mean peak height gains for boys and girls average 9.5 and 8.3 cm/year, respectively. Abbassi[5] reported that linear height growth at puberty contributes to approximately 17 to 18% of the final height.

In both sexes, but more so in boys, there is a substantial gain in lean body mass during puberty. Muscle mass, estimated by creatinine excretion, accounts for as much as 42% of body composition in boys at the age of 5 years and increases to approximately 54% by the age of 17 years. Over the same age range, the muscle mass of girls increases from approximately 40 to 45% of the total body mass. The metabolic consequences of greater muscle mass benefit the adolescent male. The advantage of having greater active muscle mass to recruit during exercise is potentially useful in providing an enhanced functional capacity and possibly metabolic rate in the exercise performance of adolescent boys compared with girls.[6]

The greatest change in body composition of the adolescent girl occurs in fat mass. In general, the body is composed of approximately 14% fat in girls at 6 years of age which increases to 25% by 17 years of age. At 6 years of age, boys have approximately 11% body fat which increases to around 15% by 17 years of age.[7] In pubescent males fat deposition of the extremities declines while truncal fat slowly increases. In pubescent females, extremity and truncal fat increase concomitantly. This gain in fat distribution is faster in girls than in boys.[7]

In adult females, it is generally understood that additional adipose tissue may be detrimental to weight-bearing exercise because of the increased mass that needs to be carried and subsequently the decreased mechanical efficiency. The metabolic consequences of the increase in adipose tissue of the adolescent female athlete as her body changes during puberty are yet to be thoroughly investigated.

Body composition changes during puberty are not explicable by the actions of any one factor. Body fat distribution during puberty may be best perceived as a complex and interactive result of biological inheritance, sex hormones, growth hormone (GH), leptin, nutritional status and energy expenditure.

The influence of gender and hormones on fat distribution in adolescents was examined in a study which included 335 participants aged 4 to 35 years.[8] Using dual energy x-ray absorptiometry (DEXA), Cowell et al.[8] postulated that the predominance of androgenic hormones in boys during the cirumpubertal years contributed to lipolysis and inhibited the fat-storing capacity of lipoprotein lipase. In contrast, the increased secretion of estrogen in pubescent girls was described as promoting lipogenesis through its inability to inhibit lipoprotein lipase. It was also observed that the most substantial changes in fat distribution occurred after puberty. In support of previous reports,[6] fat distribution in the pubescent male favoured central deposition and was independent of total body fat; in pubescent females, increasing abdominal fat frequently occurred with increasing total body fat.[7]

In addition to its role in energy balance within the body, the adipocyte hormone leptin is also believed to be related to the body composition changes observed in puberty.[9-11] Sexual dimorphism in serum leptin levels has been demonstrated in some studies.[9,11,12] However, differences between studies in the methodologies used for body fat mass and distribution measurements have meant that these observations are not conclusive.[13] There is growing evidence that serum leptin levels increase throughout puberty in girls and peak at breast stage 5. This increase is linked with both total subcutaneous fat mass and the stimulatory effects of estradiol. An inverse relationship has been reported between serum leptin levels and the age of menarche in a 4-year longitudinal study in 343 healthy caucasian girls.[14] In boys, serum leptin levels begin to increase before puberty and peak at genital stage 2. Later in the pubertal development of boys, when subcutaneous fat mass declines, leptin levels are inversely related to the activity of the gonadal axis which may indicate that androgen production has a suppressive effect on leptin.[11-12] Even when data are normalised for differences in body fat, gender-related differences remain and demonstrate higher serum leptin levels in females than males. Leptin, especially in girls, may take on a ‘permissive’ role in the timing of puberty by signalling the CNS that energy stores are adequate for pubertal development.

Body composition changes in puberty have also been examined in relation to changes in insulin resistance. In adults, insulin resistance is strongly related to the body mass index (BMI), some skinfold measures, waist circumference and intra-abdominal fat mass. Anthropometric changes in puberty do not fully account for the observed changes in insulin resistance.[15] Insulin resistance increases during the pubertal stages of P1 to P2, plateaus between P2 and P4 and returns almost to prepubescent values by P5. Although insulin resistance is greater in girls than in boys at all stages of puberty, significant differences between the 2 genders are only apparent at stage P4. However, gender-related differences in insulin resistance are not apparent in adolescents with a BMI >27 kg/m^2.[15] In general, increases in insulin resistance are not fully explained by BMI and adiposity in pubertal males and females with a BMI <27 kg/m^2.

Malina and Bouchard[7] demonstrated that the relative difference in body fat was greater in girls than in boys when athletic populations were compared with their less active contemporaries (i.e. the more physically trained girls had lower fat mass than their sedentary peers). In contrast, boys tended to show a decrease in body fat throughout their pubertal years compared with girls regardless of their training status.

The early developing pubescent male is advantaged by having greater lean muscle mass than his later developing male peers. This benefit in early developing males may be most evident during physical pursuits requiring strength, speed and power.[16] For girls who experience puberty earlier than their female peers, the additional fat gain may have the potential to 'socialise' them away from sporting aspirations. This may be particularly evident in the weight-bearing aesthetic events such as gymnastics, diving and figure skating. In these sports, success is often presented with images of lean and petite competitors. Therefore, it is prudent for prepubescent girls to develop perceived physical competence in a broad range of skills so that early or late puberty does not lead to cessation of all physical activity. Using increased physical activity to manage the gain in bodyweight which occurs in puberty may be particularly advantageous to the health of girls during puberty and, although speculative, later on in adulthood.

1.2 Hormonal Changes at Puberty

Puberty is the period of transition between childhood and adulthood that takes place in several sequential steps that are controlled by complex neuroendocrine factors. While the understanding of the process remains incomplete, various sequences and links associated with pubertal development have been established. In adults, reproductive function is mediated by 2 hormones produced by the pituitary gland, luteinising hormone (LH) and follicle-stimulating hormone (FSH), after stimulation by the luteinising hormone-releasing hormone (LHRH) [also known as gonadotropin-releasing hormone (GnRH)] from the hypothalamus. LH and FSH are responsible for ovulation and secretion of estrogen by the ovary in the female and for sperm production and secretion of testosterone by the testes in the male. Estrogen and testosterone induce the development of sexual characteristics. During adolescence, the entire endocrine system is altered, and growth, thyroid and adrenal hormones are all involved in this maturational process. However, it is essentially the activation of the hypothalamic-pituitary-gonadal axis that induces and enhances the progressive secretion of the ovarian and testicular sex hormones that are responsible for the profound biological, morphological and psychological changes that occur during puberty.[17]

In prepubertal children, LH, FSH and gonadal sex hormones levels are low. Changes in the CNS and increased frequency and amplitude of LHRH at puberty initiate increases in the secretion of LH and FSH and gonadal sex hormones. Estrogen exerts different effects from those of testosterone, and many actions of testosterone on skeletal development are the result of its aromatisation to estrogen.[18] There is also a progressive increase in the plasma levels of adrenal androgens, dehydroepiandrosterone (DHES) and the sulphate of DHES (DHEAS), which begins before the age of 8 years and continues throughout early adulthood. The period in which these changes occur is known as adrenarche.

GH increases growth at puberty via stimulation of insulin-like growth factor-1 (IGF-1; somatomedin-1) production. There is a strong association between the increased amplitude of GH secretion and peak height velocity. GH secretion returns to prepubertal amplitude following this peak in linear growth. An important synergism between the gonadal axis and the GH–IGF-1 axis occurs during puberty. Gonadal sex hormones have 2 effects on pubertal growth: enhancement of GH secretion and thus IGF-1 production; and a direct effect on cartilage and bone by stimulating production of local factors such as IGF-1.[1] The aromatisation of testosterone to estradiol mainly accounts for the effects of testosterone on GH secretion.[18]

Administering estradiol to boys and girls at different stages of puberty resulted in a greater GH amplitude response following exercise in late pubescent children when compared with their prepubescent peers.[19] Therefore, puberty is a stage of development when GH secretion is most sensitive to the stimuli created by a number of factors including the secretion of gonadal sex hormones. Other factors regulating GH release include hypothalamic GHRH and GH-inhibiting hormone (somatostatin), brain neurotransmitters and neuropeptides, IGF-1 and its binding proteins, GH and GH binding protein (GHBP), exercise and nutritional factors.[20] The precise mechanisms, sequences and progressions underlying this period of explosive growth remain uncertain; however, the growth is unequivocally linked to dramatic changes in the hormonal milieu.

Insulin is a major anabolic hormone which regulates the metabolism and storage of ingested metabolic fuels. As previously stated, serum fasting insulin levels during puberty increase 2- to 3-fold at peak height velocity, and increased insulin secretion after glucose administration suggests a degree of insulin resistance.[21] It has been suggested that the peaking of insulin resistance in puberty is selective for glucose metabolism and therefore both spares and supports the role of protein and fat metabolism during pubescent growth. Specifically, hyperinsulinaemia may regulate the GH–IGF-1 system by suppressing circulating levels of the insulin-like growth factor binding protein-1 (IGFBP-1) which may subsequently increase the availability of IGF-1 for anabolic activity.[22] Therefore, the enhanced insulin response to glucose caused by the relative increase in resistance may facilitate the anabolic effects of insulin on protein synthesis during puberty.

2. Growth Hormone-Insulin-Like Growth Factor-1 Axis Response to Exercise in Adolescents

In adults, the sensitivity of GH to aerobic and anaerobic exercise as well as its role in training and recovery from exercise has been extensively reviewed.[23] Endurance activity is linked to increased free fatty acid release and an inhibition of insulin. Intensive exercise also appears to stimulate GH secretion,[23] specifically exercise-related events such as acute shortages of energy substrates and oxygen,[24] as well as an increase in core temperature.[25] In addition to these roles, GH has been reported to support left ventricular size and ejection fraction of the heart and increased anabolic activity during exercise recovery.[26]

The response of IGF-1 to training was investigated in an intensive 5-week programme of endurance training (1 hour per day for 5 days each week in 23 adolescent girls aged 15 to 17 years.[27] A 14% decrease in serum IGF-1 levels was reported in the trained group compared with the control group over the duration of the study. Eliakim et al.[27] suggested that short term training may have resulted in a substantial catabolic hormone response in this group of girls.

Data from 38 boys (mean age 16.7 years) from the same study[27] were published separately.[28] In agreement with the data from girls,[27] a 12% decrease in serum IGF-1 levels was observed following the 5 weeks of training. However, in contrast to the girls, IGF-1 levels did not correlate well with aerobic power or lean thigh volume in this group of adolescent boys. The authors postulated that in young boys there may be a fitness-associated attenuation of GH-receptor sensitivity. It was suggested that this characteristic combined with lower levels of GHBP in a feedback loop that ultimately resulted in higher levels of GH being secreted from the pituitary as a long term adaptation to increased aerobic fitness. In adolescent boys it was the reduced levels of circulating GHBP that correlated inversely with peak aerobic power and thigh volume. The authors suggested that investigations into the roles of testosterone and estrogen on GHBP may clarify these observed gender-related differences.[28] While training elicited a catabolic response in both genders in both studies,[27,28] as stated previously, in females but not males IGF-1 was positively correlated with training-induced increases in aerobic power and lean thigh volume.

The exercise-induced hormone responses of girls during their 5 stages of puberty have been examined.[29] Once a year, over a 3-year period, 34 girls (beginning at a mean age of 11.5 years) cycled for 20 minutes at 60% peak aerobic power ($\dot{V}O_{2peak}$). Pre- and postexercise blood samples were analysed for cortisol, insulin, GH, estradiol, progesterone and testosterone. Increases in cortisol, GH and estradiol levels and a decrease in insulin levels were observed following exercise at all stages of puberty. Pre-exercise GH levels were highest and cortisol levels were lowest at breast stage 3. Peaks in estradiol and testosterone levels were observed towards the end of puberty (breast stage 5) while progesterone responses became significant at breast stage 4. The peak in the levels of sex steroids towards the end of puberty is likely to correspond with menarche. Following menarche, the late follicular phase of the normal menstrual cycle has also been associated with a 2-fold increase in serum GH levels.[30] Therefore, it would appear that the potential biological utility of GH may peak in the mid pubescent years of girls and be subsequently enhanced through the normal menstrual processes.

In summary, the neuroendocrine mechanisms that regulate growth during the adolescent years are unique. The overall result for the active adolescent may be positive in terms of enhanced GH secretion through physical activity and exercise as well as through the endogenous mediators of GH. The qualitative alterations in the GH–IGF-1 feedback systems appear to be stage- and gender-dependent and highly complex in nature.

3. Trainability in Adolescence

3.1 Anaerobic Performances in Adolescence

The power generated per kilogram of bodyweight during high intensity anaerobic exercise is lower in adolescents than in adults.[31-33] Various physiological mechanisms have been postulated to explain the lower anaerobic power in younger populations. These include lower levels of phosphofructokinase (PFK) which is a rate-limiting enzyme of the glycolytic pathway,[34] lower sympathoadrendal activity,[35] maturational differences in muscle fibre distribution[36] and immature anabolic hormonal responses such as lower levels of testosterone.[31]

Increased anaerobic potential refers to enhanced rates of ATP release via ATP-PCr (phosphocreatine) catabolism or the breakdown of stored or transported carbohydrate in active tissue under high intensity exercise demands. A progressively increased anaerobic potential in boys during adolescence compared with preadolescence is supported by studies of increased enzymatic activity,[36] anaerobic performance (defined by postexercise oxygen consumption) and serum lactate levels.[37]

In previous decades, researchers of adult-based studies have been advancing their understanding of the energy demands of high intensity exercise by using invasive procedures such as muscle biopsies. Because of ethical and moral constraints, less is known about the metabolic mechanisms and limitations of exercise responses in healthy adolescents. More recently, phosphorus nuclear magnetic resonance spectroscopy (^{31}PMRS) has been used as a well tolerated, noninvasive measurement of intracellular inorganic phosphate (Pi), PCr, ATP and pH. Increases in the ratios of these variables can reflect accelerated anaerobic glycolysis. For example, there is an increase in the Pi to PCr ratio with progressive exercise demands. This ratio has been observed to be smaller in children than in adults.[38] More specifically, children were reported to achieve a postexercise Pi to PCr ratio that was only 27% of the adult value.[38]

Kuno et al.[39] reported PCr to PCr + Pi ratios that were similar between untrained and trained 12- to 17-year-old adolescents but these values were higher than those observed in adults. Cooper and Barstow[38] concluded that ‘inherent muscle properties’, which were yet to be identified, were most likely responsible for poorer anaerobic responses associated with younger populations compared with adults.

Despite the attractiveness of the ^{31}PMRS methodology, only 1 other study involving healthy adolescents has been published.[40] In this study, skeletal muscle metabolism during short term high intensity exercise was compared in prepubertal and pubertal female swimmers (n = 18). Intracellular pH and phosphorus responses during submaximal and supramaximal plantar flexion exercise were compared between the 2 groups of participants. No between-group differences were reported following submaximal exercise. However, a lower Pi to

PCr ratio was observed in the prepubertal group compared with the pubertal group following supramaximal exercise. This finding was linked to a larger area of cross-sectional muscle mass in the more mature girls than in the prepubertal group and was therefore perceived as a ‘size-’ rather than a maturity-dependent response. Limitations of the ^{31}PMRS methodology may preclude acceptable between-group comparisons because morphological differences are unable to be controlled. The authors therefore believed that their results could not support an augmented glycolytic contribution to energy expenditure during puberty.[40] However, there is a strong indication that future research using the ^{31}PMRS method in combination with other methods which help account for differences in size and fibre type will be useful in determining the short and long term skeletal muscle responses to exercise in both genders as they progress through puberty.

Bar-Or[31] contended that younger populations should be encouraged to pursue a number of different physical activities. Conversely, adolescence is thought to be the optimal time to specialise in physical activity. In adolescent boys, the gains in muscle mass and body size appear to enhance the potential for anaerobic trainability.

Fournier et al.[41] examined the skeletal muscle adaptations in 16 adolescent boys who had undergone either endurance or sprint training over a 3-month period. The activities of a glycolytic enzyme (PFK) and an aerobic enzyme (succinate dehydrogenase) were analysed following pre- and post-intervention biopsies from the vastus lateralis. The activity of PFK increased by 21% in association with training in the sprint trained group only. A subsequent biopsy 6 months after the study revealed that the activity of this anaerobic enzyme had returned to pretraining levels.

However, there are no anaerobic training studies comparing trainability cross-sectionally through the different pubertal stages. The inherent difficulties in anaerobic training studies include a lack of agreement on what denotes anaerobic training, the complex masking effects of hormonal factors with training responses during puberty and the difficulties in matching exercise and control groups.[42] The difficulties in matching exercise and control groups are due to problems in matching pubertal status, training status, training history and body composition between the 2 groups. In addition, in contrast to the studies where gender comparisons can be readily conducted with prepubertal groups, factors that need to be considered for gender comparisons of anaerobic trainability during adolescence become even more complex. Consequently, the responses of well-matched male and female adolescents to the short and long term stresses involved with anaerobic training is poorly described in the literature. Therefore, it is apparent from the limited data available that the understanding of anaerobic trainability during adolescence requires further research; however, the training outcomes appear to be specific and transient.

3.2 Resistance Training in Adolescence

Early studies conducted on resistance training in younger populations were not associated with improvements in strength. Often total training volume was relatively low in these early studies,[43-44] and researchers believed that these programmes were ineffective because of the lack of hypertrophy and relatively low absolute strength gains compared with the gains observed in adult populations.[45] The Position Statement on Youth Resistance Training published by National Strength and Conditioning Association from the US[46] upheld the more recent belief that properly supervised and well planned resistance training can be effective and safe in improving the strength of preadolescent and adolescent populations. Reliable research on strength training in young populations is limited, however.

Many weaknesses have been identified in resistance training studies conducted to date in younger populations.[47-48] Falk and Tenenbaum[47] have indicated the need for researchers to provide a pre-learning phase in the techniques and tests associated with resistance training so that tests reflect valid gains in strength rather than a familiarisation of the trainee with the tests. They[47] further noted that researchers in this area have frequently neglected to include a control group and/or randomly assign participants to groups. The authors[47] were also critical of the paucity of reliable data in resistance training studies in younger populations that included female participants. A similar criticism can be made of the literature including adolescents, with most of the early studies focusing on boys.[49] There are reports of substantial relative and absolute strength gains from a variety of resistance training modes, frequencies and programme duration in resistance training studies with adolescent boys.[45]

The gains in lean mass that more consistently accompany adolescent than preadolescent resistance training in boys are, however, smaller than the gains in strength.[50] Blimkie and Sale[50] contended that early maturing boys are stronger than those who are slower to mature; however, the maturity-related differences are less discernable by the completion of puberty. The velocity of strength gain in pubescent males appears to peak following peak height velocity. This observation is not as consistent in girls in that only around 50% peak in strength gain following their peak height velocity. Blimkie and Sale[50] suggested that many girls may peak in strength before or during peak height velocity. Despite the stage at which it occurs, the magnitude of the strength gain is consistently greater in boys than in girls.[50]

Resistance training studies in young female populations have more commonly assessed changes in bone mineral content and bone density as well as physical strength. Recently, Nichols et al.[51] compared strength and bone mineral density changes that occurred with a 5-month resistance training programme (30 to 45 minutes per day for 3 days each week). The researchers recruited 10 adolescent girls undergoing training (aged 14 to 17 years) and 10 sedentary (<2 hours of activity per week) age-matched girls. A 1-repetition maximum for the leg press and bench press increased strength by 18 and 20%, respectively, over the 5-month period in the group undergoing training. Total body bone mineral density increased to a greater extent in the resistance training group than in the control group. Changes in the lumbar spine and femoral neck bone mineral density, however, did not differ between groups.

A resistance training study of similar duration (26 weeks) was conducted by Blimkie et al.[52] who recruited postmenarcheal adolescent girls. Participants were required to train 3 times per week using hydraulic resistance machines. Compared with the control group, there were no differences in the total body bone mineral density or lumbar spine bone mineral density and bone mineral content after training. Significant changes were apparent between the pre- and postintervention percentage strength gains using manoeuvres that included bicep curls (21.4%), knee extension (25.1%), knee flexion (52.8%) and squat press (21.5%). However, no differences were reported in the pre- and postintervention anthropometric measurements of the upper arm girth or the cross-sectional area of the quadricep muscle using computerised tomography.

Therefore, hypertrophy and bone mineral changes may not consistently characterise the effects of resistance training in postmenarchal girls. However, marked changes in strength can be expected from well planned and carefully supervised programmes that are assessed with valid and specific testing regimens. Changes in bone mineral density are dependent on the nature of the mechanical load and the duration of the study. Longer term resistance training studies may provide more adequate time for a greater degree of bone remodelling to occur than the relatively short term studies described in the current literature.

The vast array of resistance training programmes used in the research available to date has lead to substantial inconsistencies between studies. This research also represented different genders and stages of maturation and varying levels of familiarisation with programme techniques. The inconsistencies consequently make prescribing exercise for resistance training extremely difficult. Safety remains the major issue in resistance training in adolescent populations. Appropriate teaching, planning, supervision and equipment are fundamental to a safe and positive framework for the implementation of resistance training programmes. It would be extremely valuable to have more documentation in the scientific literature of effective resistance programmes designed for, and implemented with, adolescents.

3.3 Aerobic Training in Adolescence

The cardiorespiratory benefits of aerobic exercise are evident early in life.[42,53] Pate and Ward[54] identified 3 physiological characteristics that denote increased endurance performance in children and youth. These characteristics are a high peak oxygen consumption ($\dot{V}O_{2peak}$), a delayed lactate threshold and an efficient economy of energy expenditure during submaximal performance. Pate and Ward[54] asserted that all 3 characteristics of endurance performance could be enhanced with training in younger populations.

Several reviews of submaximal energy expenditure during the developmental years appear elsewhere in the literature[42,55] These reviews highlight a spectrum of measurements through which submaximal performances by adolescents can be assessed including energy costs, cardiorespiratory responses and substrate and metabolite levels in the blood.

Comparisons of submaximal substrate utilisation have been conducted in some of the earliest research into the exercise responses of adolescents. No differences in plasma free fatty acid and glucose levels during prolonged exercise were reported when adolescent populations were compared with younger or older age groups.[56,57] In addition, similar plasma levels of free fatty acid and glucose were observed between well trained adolescent boys and girls (mean age 15.5 ± 0.3 years) when they participated in a laboratory simulated triathlon.[58] The validity and reliability of using plasma substrate samples is likely to become clearer when studies of adolescent responses to exercise are conducted using isotopic tracer protocols for substrates such as glycerol and leucine. The precision associated with isotopic tracer methodology is likely to reduce the large variance frequently reported in metabolic blood sampling.

Lactate is a blood borne metabolite and levels of this substance are lower in children than in adults following maximal exercise. Higher maximal and lower submaximal lactate levels may theoretically be expected in the adolescent period between childhood and adulthood. During adolescence energy expenditure becomes more efficient submaximally and results in lower plasma and blood lactate levels. Conversely, more powerful maximal performances by adolescents can generate higher plasma and serum lactate levels. A longitudinal study in 8 male runners examined submaximal and maximal responses to exercise over an 8-year period which began when the participants were 12 years of age. While submaximal blood lactate responses decreased over the period of the study in the well trained males, maximal lactate responses did not differ between the trained and sedentary groups.[59] The highest ‘steady state’ lactate responses were also investigated in 34 adolescent boys (15.4 ± 2.8 years of age). Blood lactate levels corresponding with the highest workload at which steady state could be maintained was independent of age and was more related to training status.[60] The absence of a difference in maximal plasma lactate levels among trained and untrained adolescents possibly reflects the insensitivity of blood lactate as a marker of physiological change following maximal exercise in the second decade of life. Therefore, the sensitivity of blood lactate levels to the training status of adolescents appears to be greater under submaximal rather than maximal exercise stimuli.

Energy efficiency can be defined as the energy expenditure requirements for a given amount of work. It has been reported to be similar in preadolescent and adolescent males during nonweight-bearing cycling.[61,62] In weight-bearing exercise such as treadmill running, the $\dot{V}O_2$ per kg of bodyweight can be expected to decline by about 1.0 ml/kg/min for each year between childhood and adolescence.[42] When data from treadmill studies were expressed as $\dot{V}O_2$ per stride, no differences were reported between preadolescent and adolescent populations.[63] However, there is a correlation between stride frequency and efficiency that highlights developmentally-related improvements in running mechanics.[64]

Adolescents, however, appear to be somewhat less efficient in energy expenditure than adults. A recent study on the energy cost of horizontal walking and running in 47 male and 35 female adolescents found that applying adult regression models to predict the energy costs of adolescents underestimated the true costs.[65] Thus, it appears that adolescents reflect age-related improvements in weight-bearing activity but may not demonstrate the economy of movement of which an adult is capable. During and after adolescence, it is likely that the potential for improving physical performance may be more dependent on training than on development.

Most endurance training studies in children and adolescents have focused on changes in $\dot{V}O_{2peak}$ rather than lactate and substrate responses during submaximal performance. The aerobic trainability, assessed using $\dot{V}O_{2peak}$, in preadolescent and adolescent populations has been the focus of several reviews and meta-analyses.[42,54,66,67] Results vary according to the inclusion criteria of the studies. Pate and Ward[54] restricted their review to studies that included control groups (random or pair-matched), physiological measures of training outcomes, clear descriptions of training protocols and publication in peer-reviewed journals. The authors[54] reported an average aerobic training improvement in $\dot{V}O_{2peak}$ in approximately 10% in children and adolescents. Because of the lack of data, the quantity of change in $\dot{V}O_{2peak}$ in aerobic trainability that occurs during, and as a result of, puberty remains uncertain. Although the authors acknowledged the very limited number of training studies including pre-, mid and postpubescent participants, they surmised that aerobic trainability appeared to be similar before and after puberty but not during it.[54]

One group of investigators[68] conducted a 10-week aerobic training programme (4 days per week) in twin boys aged 10, 13 and 16 years of age (n = 26). Of each twin pair, 1 twin trained and the other acted as the control. The authors observed a 13 and 15% improvement in $\dot{V}O_{2peak}$ in the trained pre- and postpubescent groups, respectively, but only a 10 to 11% improvement in the trained midpubescent group. Thus, the authors suggested that a decreased sensitivity to training occurred in the middle of the pubertal growth spurt when compared with the years surrounding it. The limited evidence for this hypothesis renders it difficult to support beyond the scope of the original study. The conclusions made by Pate and Ward[54] in the paragraph above appeared to be influenced by the present study.[68]

Not all researchers maintain this perspective. In a review[69] the $\dot{V}O_{2peak}$ during training of preadolescent populations was concluded to average 5% when the criteria for reviewing studies included only those with an acceptable effect size, well-matched control groups and a quantifiable estimate of the training intervention. Furthermore, it has been suggested that there were marked increases in the aerobic trainability of males following puberty.[70] The ‘trigger hypothesis’ was revisited from the original work of Katch[71] to help explain this observation. This hypothesis proposed that the marked increase in the potential for aerobic power in more mature males was associated with pubertal changes. Kobayashi et al.[72] monitored $\dot{V}O_{2peak}$ in boys aged between 9 to 10 and 15 to 16 years of age who were long distance runners. The researchers observed exponential increases $\dot{V}O_{2peak}$ around peak height velocity at approximately 14 years of age. The fact that the participants in the aforementioned study[72] were well trained highlights the need to interpret improvements within the context of the training background as well as with regards to growth. The growth-related improvements in aerobic trainability in well trained male adolescent athletes compared with well trained preadolescent male populations may be associated with the interactive effects of leaner body composition, proportionally higher muscle mass, higher blood oxygen carrying capacity and larger maximal cardiac output. These adaptations concomitantly relate to increased testosterone, GH and other hormone secretions that occur with maturation in males.[70,71] Thus, aerobic improvements in adolescent males may be related to maturation as well as training. The mechanisms through which both maturation and training improve aerobic performance in adolescent males are not well differentiated, however.

Inequality in the aerobic fitness of female compared with male populations is evident before puberty. Trends in $\dot{V}O_{2peak}$ (in ml/kg/min) in a sample of Polish girls (active and inactive) indicated that girls appeared to peak in their relative aerobic fitness 2 years before menarche and this then decreased almost linearly for approximately 3 years after menarche.[7] Although data are scarce, when girls engage in endurance-based sports during their adolescent years, $\dot{V}O_{2peak}$ scores appear to be similar to, or superior to, those published for prepubescent girls.

Baxter-Jones et al.[73] tracked $\dot{V}O_{2peak}$ performance in participants who were training intensively for soccer, gymnastics, tennis or swimming. The researchers conducted a mixed longitudinal research project using 5 age cohorts (8, 10, 12, 14 and 16 years) of approximately 450 young athletes over 3 years. Progressive increases in absolute aerobic power through the pre-, mid and late pubertal years were observed in boys. In contrast, the girls failed to demonstrate increased aerobic power during puberty. The $\dot{V}O_{2peak}$ of all the sporting groups entering the study, however, was higher than the mean values obtained for age-matched normative values for British children who were neither training nor sedentary. It is highly commendable that the active adolescent girls included in this study maintained their aerobic fitness despite substantial changes in fat mass and body image that would have occurred in most of the female participants. In addition, active young girls could in fact be rejecting or reversing the downward trend to decreased aerobic fitness that may occur in normative population profiles of adolescent girls. However, the aerobic trainability of young girls appears to be less researched than their male counterparts.

From one of the few aerobic training studies in adolescent girls (aged 15 to 17 years), Eliakim et al.[27] reported a 12% improvement in $\dot{V}O_{2peak}$ following 5 weeks of endurance training for 1 hour a day. In contrast, only a 7% difference was observed in another study in 14-year-old girls who trained for 40 minutes, 3 times a week for 10 weeks in whom average heart rates were greater than 160 beats per minute.[74] One possible explanation for the differences in results between the 2 studies may be that the volume of training was less in the study conducted by Armstrong.[74] It is also possible that there was a selection bias in that only adolescents with reasonable fitness volunteered for the training study by Armstrong[74] and therefore may have already been experiencing their peak aerobic fitness. However, both studies enrolled adolescent girls with a baseline $\dot{V}O_{2peak}$ of approximately 42 ml/kg/min. The aerobic trainability of adolescent girls requires further research.

As previously stated, a predominance of research has concentrated on fitness-based outcomes of exercise training where improvements were reported largely through increases in $\dot{V}O_{2peak}$.[75] A more holistic trend in exercise intervention studies in younger populations has been to include other health-related fitness measures such as blood lipid profiles, bone health and blood pressure. For example, a 16-week endurance study in 15 boys and 10 girls (aged 13 to 18 years) resulted in decreased total cholesterol levels and increased serum high density lipoprotein-cholesterol levels in the trained group compared with their pretraining values. No changes in either parameter were reported in the control group.[76] However, the training-related improvements in these 2 parameters observed in this study are not supported by other studies in adolescent participants.[77,78]

Improvements in markers of bone status were demonstrated following a 5-week intensive aerobic training programme at a summer school. Specifically, increases in blood borne markers of bone formation and decreases in markers of bone resorption were reported in a group of 38 boys (mean age 16.7 years) who were either in the exercise or the control groups.[79] In another study, a 1-semester programme of endurance training increased the aerobic fitness and decreased the systolic blood pressure in 44 adolescent girls who commenced the programme with blood pressure readings above the 67th percentile. In this randomised study the control group was assigned to the standard physical education programme.[80] Alternatively, a study examining the relationship between blood pressure and physical fitness in approximately 6000 Danish adolescents reported that fitness (assessed using $\dot{V}O_{2peak}$) was negatively related to blood pressure only up to the 50th percentile in boys and up to the 80th to 90th percentile in the girls.[81]

It appears from the literature that the health-related responses that accompany aerobic training in adolescence are limited, equivocal and clearly require further research. Studies that have researched aerobic trainability in adolescents have indicated increased $\dot{V}O_{2peak}$ values. The aerobic trainability of adolescents, as in adult intervention studies, most likely depends on the initial fitness of participants, their training history, compliance with the training programme and the quality and duration of the intervention.

4. Intensive Training During Adolescence

4.1 Intensive Training and its Potential Effects on Growth at Puberty

The question of how intensive training may affect pubertal growth has challenged researchers for decades. To address this question a British study profiled elite junior athletes from a number of sports annually over a 3-year period.[82] The starting age of participants in this study was sport-dependent and ranged from 8 to 16 years. The sporting groups studied (gymnastics, tennis, soccer and swimming) were training at different volumes and intensities. Participants were randomly selected from lists supplied by each of the 4 sporting associations and represented athletes who were undertaking the highest amounts of training expected of junior athletes in their respective sport. The authors[82] reported that the young participants largely remained on the same percentile for height and bodyweight over the 3-year period. For example, swimmers started and ended the study in the highest percentile rankings for height and bodyweight. In contrast, gymnasts started and ended the 3-year study with very low percentile scores for height and bodyweight. The absence of a downward shift in percentile rankings suggested that sport specific intensive training was not delaying growth during the years of measurement in this study.

While there is anecdotal evidence that aesthetic bodyweight-dependent sports, (gymnastics, figure skating, diving) appear to delay maturation, scientific reports of this observation are scarce. There are, however, a small number of reports of inappropriate decreases in standard deviation scores for height in young, active populations. This was implicit in the observation of stunted leg growth in a group of elite Swiss gymnasts (mean age 12.3 ± 0.3 years) who had been training an average of 22 hours per week.[83] The authors[83] postulated that prolonged and intensive training might have interfered with the normal activities of the hypothalamic-pituitary-gonadal axis.

Another group of gymnasts (aged 7 to 16 years) were assessed over a 5-year period and were reported to have a slower growth rate and delayed menarche compared with a control group.[84] There was also a report of decreased growth velocity in 16% of 97 young dancers.[85] However, growth velocity studies require careful analysis as training may not be the primary and single factor influencing growth.

Bass et al.[86] examined growth patterns of 83 active gymnasts (aged 5 to 15 years). Data were also analysed for the effects of the number of years of gymnastics training; participants had all been training for 0.5 to 10 years. Shorter leg length was observed in all gymnasts regardless of how long they had been training. Leg length velocity in the gymnasts increased at the same rate as in the control group. However, growth in the leg length of the gymnasts was tracked over a lower percentile. This indicated that gymnasts with constitutional delayed growth may be coincidently selected as they have attributes that are considered beneficial in gymnastics.

In the study by Pigeon et al.,[85] delayed onset of menarche was reported in young dancers compared with a control group. The incidence of delayed menarche, however, needs to be assessed in relation to the age of menarche in the mothers of the young athletes rather than in relation to a less active control group.[82] Malina et al.[87] contended that menarche needs to be considered as an interactive product of training, nutrition, familial numbers, and coach or family stress.

It is evident, therefore, that selection processes for sports such as gymnastics and dance may positively discriminate towards young athletes that have familial short stature or constitutional delayed growth. Peltenburg et al.[88] reported that elite gymnasts had a shorter stature from as early as 2 years of age, well before their gymnastics career began. In active adolescents for whom training is part of a balanced lifestyle, skeletal development through weight-bearing activity may in fact be enhanced when compared with less active young people at the same stage of development. Generally, normal levels of physical activity should not be associated with delayed sexual maturation or stunted height.[89]

4.2 Intensive Training and Injury Risks to Adolescent Athletes

More than ever, young athletes are being asked to participate in intensive and more repetitive sports training from an early age.[90] This has resulted in the popular notion that excessive training during adolescence may lead to inordinate physical stresses on the cardiorespiratory and musculoskeletal systems of the young athlete. Consequently, it is inferred that the impact of these stresses may impair normal function and result in long term implications for musculoskeletal function. However, the literature does not support these theories, and in fact there is much greater evidence of the apparent physiological benefits of long term training during the adolescent years.[91,92]

Reports of overuse injuries in children and adolescents, however, are increasingly common and should not be ignored.[89,93] Musculoskeletal injuries routinely occur at a similar rate in adults and younger populations;[94] however, the causes of these injuries may differ.[95] For example, an examination of the causes of ‘lower back pain’ in 100 adolescents and 100 adults who attended a sports medicine clinic and orthopaedic hospital, respectively, revealed the incidence of spondylolysis stress-related pain occurred in 47% of adolescents and 5% of adults.[95]

There are a number of musculoskeletal problems that are specific to children and adolescents because of the differences in the structure of the growing bone compared with adult bone.[96] Ligaments are 2 to 3 times stronger than bone, which results in epiphyseal fractures or avulsion injuries rather than ligamentous damage.[96] The growth plate or epiphysis is at risk of disruption, especially from shearing forces, and fractures of the growth plate are of particular concern because of the dangers of interruption to the growth process. It is acknowledged that the cartilage of the growth plate may be at its weakest during the most intensive phases of growth in adolescence.[97]

A review of the literature on the potential suppressive role of physical loading on radial growth in female gymnasts (n = 38, aged 10.5 to 17 years) investigated the vulnerability of female gymnasts to stress-related distal radius epiphyseal arrest. Given that the reported incidence of epiphyseal arrest ranged from 10 to 85% in gymnasts from 5 cross-sectional studies, the authors[98] believed that the evidence of vulnerability to injury was unconvincing and inconclusive.

The metaphysis of the long bones is more resilient and elastic in children and adolescents than in adults, thus greenstick fractures occur more readily in children and adolescents. The articular cartilage of growing bone is of greater depth than that of adult bone and can undergo remodelling. The impact of repetitive microtrauma in some adolescents is linked to subchondral stress fractures that can regress to osteochondrosis dissecans in the articular cartilage of the ankle, knee or elbow if they remain untreated. The cartilaginous apophysis provides a relatively weak attachment site for tendons to bone which if overused can result in apophysitis such as Osgood-Schlatters (tibial tubercle apophysitis) and Sever's Disease (calcaneal apophysitis). Hard rapid loading of the muscles, tendons and bone associated with explosive and intensive movements in exercise can lead to avulsion fracture of the apophysis.

Segesser et al.[99] reported that the ratio of the incidence of apophyseal lesions in adolescent boys compared with girls was 9 **:** 1. DiFiori[93] identified common factors contributing to the incidence of apophyseal injuries among adolescent populations such as weakened growth cartilages compared with the supporting muscle tendon unit, poor flexibility and increased tracking on the tendon insertion sites.

The American College of Sports Medicine contended that half of the overuse injuries diagnosed in adolescents were preventable.[100] To prevent injury, preparticipation screening and regular visits to sports medicine health professionals are strongly recommended in adolescent athletes who are serious about their training.[89,91,93]

In summary, it is acknowledged that adolescence is the recommended stage of development for specialising in sports. However, adolescent athletes participating in high volumes of training at the same time that marked changes in physical growth are occurring may be at greater risk of microtraumatic injury than athletes at any other stage of development. Therefore, there may be a need to carefully monitor the volume of training at the time of rapid growth and development.

5. Conclusion

The adolescent years present a unique challenge to the young athlete. Girls who remain active through their pubertal years may maintain superior aerobic fitness and remain leaner than their inactive peers. Active adolescent boys may improve their aerobic and anaerobic power through mechanisms that appear to be related to body composition, haemoglobin increases, hormonal changes and/or relatively high levels of habitual physical activity. In general, however, there is a dearth of research on the training responses of adolescent girls to aerobic, anaerobic and resistance intervention programmes.

Advances in technology have increased the understanding of exercise performances of adolescent populations using noninvasive procedures. Despite these improvements, the mechanisms behind the poorer anaerobic performances of young athletes compared with older athletes remain elusive. Because of the interactive effects of intensive training and growth during adolescence, intensive training could result in some adolescent athletes being particularly vulnerable to repetitive microtraumatic injuries.

In general, participation in sports during adolescence does not appear to be detrimental to maturational processes. The area of adolescent exercise provides useful and challenging information. It will, however, require an aggressive pursuit of knowledge if campaigns to increase physical activity and attract younger populations to participate are to succeed. Identifying the mechanisms responsible for changes in the physiology of adolescent athletes may facilitate the development of specific programmes for adolescents. The exercise prescription to produce the most desirable and long term benefits for adolescents requires dynamic and individual programming.

References

1. Rogal AD. Growth at puberty: interaction of androgens and growth hormone. Med Sci Sports Exerc 1994; 26 (6): 767-70
2. Ducharme JR, Forest MG. Normal pubertal development. In: Bertrand J, Rappaport R, Sizonenko PC, editors. Pediatric endocrinology. 2nd ed. Baltimore (MD): Williams & Wilkins, 1993: 372-86
3. Tanner JM. Foetus into man. Exeter: A. Wheaton & Co. Ltd, 1978
4. Cheng JCY, Maffulli N, Leung SSSF, et al. Axial and peripheral bone mineral acquisition: a 3-year longitudinal study in Chinese adolescents. Eur J Pediatr 1999; 158: 506-12
5. Abbassi V. Growth and normal puberty. Pediatrics 1998; 102 (2 Suppl.): 507-11
6. Clarkson P, Going S. Body composition and weight control: a perspective on females. In: Bar-Or O, Lamb D, Clarkson P, editors. Perspectives in exercise science and sports medicine. Exercise and the female: a life span approach. Carmel (IN): Cooper Publishing Co., 1996: 147-214
7. Malina R, Bouchard C. Growth, maturation and physical activity. Champaigne (IL): Human Kinetics, 1991
8. Cowell CT, Brady J, Lloyd-Jones S, et al. Fat distribution in children and adolescents: the influence of sex and hormones. Horm Res 1997; 48 (5 Suppl.): 93-100

9. Demerath E, Towne B, Wisemandle W, et al. Serum leptin concentration, body composition, and gonadal hormones during puberty. Int J Obes 1999; 23: 678-85
10. Kiess W, Reich A, Meyer K, et al. A role for leptin in sexual maturation and puberty? Horm Res 1999; 51 (3 Suppl.): 55-63
11. Roemmich JN, Clark P, Berr S, et al. Gender difference in leptin levels during puberty are related to the subcutaneous fat depot and sex steroids. Am J Physiol 1998; 275 (3 Pt 1): E543-51
12. Blum W, Englaro P, Hanitsch S, et al. Plasma leptin levels in healthy children and adolescents: dependence on body mass index, body fat mass, gender, pubertal stage and testosterone. J Clin Endocrinol Metab 1997; 82 (9): 2904-10
13. Arslanian S, Suprasongsin C, Kalhan S, et al. Plasma leptin in children relationship to puberty, gender, body composition and energy expenditure. Metabolism 1998; 47 (3): 309-12
14. Matkovic V, Ilich J, Skugor M, et al. Leptin is inversely related to age at menarche in human females. J Clin Endocrinol Metab 1997; 82 (10): 3239-45
15. Moran A, Jacobs D, Steinberger J, et al. Insulin resistance during puberty: results from clamp studies in 357 children. Diabetes 1999; 48 (10): 2039-44
16. Armstrong N, Welsman J. Young people and physical activity. Oxford: Oxford University Press, 1997
17. Grumbach MM, Styne DM. Puberty: ontogeny, neuroendocrinology, physiology and disorders. In: Wilson JD, Foster DW, Kronenberg HM, et al., editors. Williams textbook of endocrinology. 9th ed. Philadelphia (PA): WB Saunders, 1998: 1509-625
18. Metzger DL, Kerrigan JR. Estrogen receptor blockade with tamoxifen diminishes growth hormone secretion in boys: evidence for a stimulatory role of endogenous estrogen during male adolescence. J Clin Endocrinol Metab 1994; 79: 513-8
19. Marin G, Domene HM, Barnes KM, et al. The effects of estrogen priming and puberty on the growth hormone response to standardized treadmill exercise and arginine-insulin in normal girls and boys. J Clin Endorinol Metab 1994; 79 (2): 537-41
20. Rogal AD. Growth and growth hormone secretion at puberty in males. In: Blimkie CJR, Bar-Or O, editors. New horizons in pediatric exercise science. Champaigne (IL): Human Kinetics, 1995: 39-53
21. Amiel SA, Caprio S, Sherwin RS. Insulin resistance of puberty: a defect restricted to peripheral glucose metabolism. J Clin Endocrinol Metab 1991; 72: 277-82
22. Caprio S. Insulin: the other anabolic hormone of puberty. Acta Paediatr 1999; 433 Suppl.: 84-7
23. Roemmich JN, Rogal AD. Exercise and growth hormone: does one affect the other? J Pediatr 1997; 131 (1S Suppl.): 75S-80S
24. Felsing NE, Brasel J, Cooper DM. Effect of low- and high-intensity exercise on circulating growth hormone in men. J Endocrinol Metab 1992; 75: 157-62
25. Kappel M, Gyhrs A, Galbo H, et al. The response on glucoregulatory hormones of *in vivo* whole body hyperthermia. Int J Hyperthermia 1997; 13: 413-21
26. Borer K. The effects of exercise on growth. Sports Med 1995; 20: 375-97
27. Eliakim A, Brasel JA, Mohan S, et al. Physical fitness, endurance training and the growth hormone insulin-like growth factor I system in adolescent females. J Clin Endocrinol Metab 1996; 81: 3986-92
28. Eliakim A, Brasel JA, Barstow TJ, et al. Peak oxygen uptake, muscle volume, and the growth hormone-insulin-like growth factor-1 axis in adolescent males. Med Sci Sports Exerc 1998; 30: 512-7
29. Viru A, Laaneots L, Karelson K, et al. Exercise-induced hormone responses in girls at different stages of sexual maturation. Eur J Appl Physiol 1998; 77: 401-8
30. Veldhuis JD. The neuroendocrine regulation and implications of pulsitileGH secretion: gender effects. Endocrinologist 1995; 5 (3): 198-213
31. Bar-Or O. Pediatric sports medicine for the practitioner: from physiologic principles to clinical applications. New York: Springer-Verlag, 1983
32. Naughton G, Carlson J, Iuliano S, et al. Cardiorespiratory responses and circulating metabolite concentrations in male and female adolescents during a simulated duathlon. Int J Sports Med 1998; 19: 303-9
33. Van Praagh E. Developmental aspects of anaerobic function. In: Armstrong N, Kirby B, Welsman J, editors. Children and exercise XIX: promoting health and well-being: proceedings of the XIX International Symposium of the European Group of Pediatric Work Physiology; 1997 Sep 16-21. London: E & FN Spon, 1997: 267-90
34. Eriksson BO, Gollnick PD, Saltin B. Muscle metabolism and enzyme activities after training in boys 11-13 years old. Acta Physiol Scand 1973; 87: 485-97
35. Lehmann M, Keul J, Korsten-Reck U. The influence of graduated treadmill exercise on plasma catecholamines, aerobic and anaerobic capacity in boys and adults. Eur J Appl Physiol 1981; 47: 301-11
36. Berg A, Keul J. Biochemical changes during exercise in children. In: Malina RM, editor. Young athletes: a biological, psychological and educational perspective. Champaign (IL): Human Kinetics, 1988: 61-7
37. Paterson DH, Cunningham DA. Development of anaerobic capacity in early and late maturing boys. In: Binkhorst RA, Kemper, HCG, Saris WHM, editors. Children and exercise XI: proceedings of the Eleventh International Congress on Pediatric Work Physiology; 1983 Sep; Papendal, the Netherlands. Champaign (IL): Human Kinetics, 1985: 119-28
38. Cooper DM, Barstow TJ. Magnetic resonance imaging and spectroscopy in studying exercise in children. Exerc Sports Sci Rev 1996; 24: 475-99
39. Kuno S, Takahashi H, Fujimoto K, et al. Muscle metabolism during exercise using phosphorus^{31} nuclear magnetic resonance spectroscopy in adolescents. Eur J Appl Physiol 1995; 70: 301-4
40. Petersen S, Gaul C, Stanton M, et al. Skeletal muscle metabolism during short-term, high-intensity exercise in prepubertal and pubertal girls. J Appl Physiol 1999; 87 (6): 2151-6
41. Fournier M, Ricci J, Taylor AW, et al. Skeletal muscle adaptation in adolescent boys: sprint and endurance training and detraining. Med Sci Sports Exerc 1982; 14: 453-6
42. Rowland TW. Developmental exercise physiology. Champaign (IL): Human Kinetics, 1996
43. Docherty D, Gaul CA, Wenger HA. Exercise and the young female: maturational differences in the responsiveness to aerobic training. Ottawa (ON): Canadian Fitness and Lifestyle Research Institute, 1990. Technical report no.: 9107-2135
44. Kirsten G. Der einfluss isometrischen muskeltrainings auf die entwicklung der muskelkraft. Jugendlicher Internationale Zeischrift fur Angewandte Physiologie Einschliesslich. Argbeitsphysiologie 1963; 19: 387-402
45. Blimkie CJR, Bar-Or O. Trainability of muscle strength, power and endurance during childhood. In: Bar-Or O, editor. The child and adolescent athlete. Oxford: Blackwell Science, 1996: 113-29

46. National Strength and Conditioning Association. Youth resistance training: position statement paper and literature review. Natl Strength Cond Assoc J 1996; 18 (6): 62-75
47. Falk B, Tenenbaum G. The effectiveness of resistance training in children. Sports Med 1996; 22 (3): 176-86
48. Payne VG, Morrow JR, Johnson L, et al. Resistance training in children and youth. A meta-analysis. Res Q Exerc Sport 1997; 68 (1): 80-8
49. deKoning FL, Binkhorst RA, Vissers ACA, et al. The influence of static strength training on the force-velocity relationship of the arm flexors of 16-year-old boys. In: Ilmarinen J, Valimaki I, editors. Children and sport paediatric work physiology. New York (NY): Springer-Verlag, 1984: 201-5
50. Blimkie CJR, Sale DG. Strength development and trainability during childhood. In: Van Praagh E, editor. Pediatric anaerobic performance. Champaign (IL): Human Kinetics, 1998: 193-224
51. Nichols DL, Sandborn CF, Bonnick SL. The effects of resistance training on bone mineral density in adolescent females. J Bone Miner Res 1996; 11 (1 Suppl. Pt 2): S466
52. Blimkie CJR, Rice S, Webber CE, et al. Effects of resistance training on bone mineral content and density in adolescent females. Can J Physiol Pharm 1996; 74 (9): 1025-33
53. Rowland TW, Boyajian A. Aerobic response to endurance exercise training in children. Pediatrics 1995; 96 (4): 654-8
54. Pate R, Ward DS. Endurance trainability of children and youth. In: Bar-Or O, editor. The child and adolescent athlete. Oxford: Blackwell Science, 1996: 130-7
55. Turley KR. Cardiovascular responses to exercise in children. Sports Med 1997; 24 (4): 241-57
56. Eriksson B, Persson B, Thorell J. The effects of repeated prolonged exercise on plasma growth hormone, insulin, glucose, free fatty acids, glycerol, lactate, and beta-hydroxybutyric acid in 13 year old boys and in adults. Acta Paediatr Scand 1971; 217 Suppl.: 142-6
57. Wirth A, Trager E, Scheele K, et al. Cardiopulmonary adjustment and metabolic response to maximal and submaximal physical exercise of boys and girls at different stage of maturity. Eur J Appl Physiol 1978; 39: 229-40
58. Naughton GA, Carlson JS, Buttifant DC, et al. Accumulated oxygen deficit measurements during and after high-intensity exercise in trained male and female adolescents. Eur J Appl Physiol 1997; 76: 525-31
59. Sjodin B, Svedenhag J. Oxygen uptake during running as related to body mass in circumpubertal boys: a longitudinal study. Eur J Appl Physiol 1992; 65 (2): 150-7
60. Beneke R, Heck H, Schwarz V, et al. Maximal lactate steady state during the second decade of age. Med Sci Sports Exerc 1996; 28 (12): 1474-8
61. Cooper DM, Weiler-Ravell D, Whipp BJ, et al. Aerobic parameters of exercise as a function of body size during growth in children. J Appl Physiol 1984; 56: 628-34
62. Klausen K, Rasmussen B, Glensgaard LK, et al. Work efficiency of children during submaximal bicycle exercise. In: Binkhorst RA, Kemper HCG, Saris WHM, editors. Children and exercise XI: proceedings of the Eleventh International Congress on Pediatric Work Physiology; 1983 Sep; Papendal, the Netherlands. Champaign (IL): Human Kinetics, 1985: 210-7
63. Waters RL, Hislop HJ, Thomas L, et al. Energy cost of walking in normal children and teenagers. Dev Med Child Neurol 1983; 25: 184-8
64. Rowland TW, Cunningham LN. Oxygen uptake plateau during maximal treadmill exercise in children. Chest 1992; 101: 485-9
65. Walker JL, Murray TD, Jackson AS, et al. The energy cost of horizontal walking and running in adolescents. Med Sci Sports Exerc 1999; 31 (2): 311-22
66. Pate R, Ward DS. Endurance exercise trainability in children and youth. In: Grana WA, Lombardi JA, Sharkey BJ, et al., editors. Advances in sports medicine and fitness. Chicago: Year Book Medical, 1990; 37-55
67. Vaccaro P, Mahon A. Cardiorespiratory responses to endurance training in children. Sports Med 1987; 4: 352-6
68. Weber G, Kartodihardjo W, Klissouras V. Growth and physical training with reference to heredity. J Appl Physiol 1976; 40: 211-5
69. Payne VG, Morrow JR. The effect of physical training on prepubescent $\dot{V}O_{2max}$: a meta-analysis. Res Q Exerc Sport 1993; 64: 305-13
70. Rowland TW. The 'Trigger Hypothesis' for aerobic trainability: a 14-year follow up [editorial]. Ped Exerc Sci 1997; 9: 1-9
71. Katch VL. Physical conditioning of children. J Adol Health Care 1983; 3: 241-6
72. Kobayashi K, Kitamura K, Miura M, et al. Aerobic power as related to body growth and training in Japanese boys: a longitudinal study. J Appl Physiol 1978; 44: 666-72
73. Baxter-Jones ADG, Goldstein H, Helms PJ. The development of aerobic power in young athletes. J Appl Physiol 1993; 75 (3): 1160-7
74. Armstrong S. Endurance and metabolic responses to circuit training in female adolescents [thesis]. Melbourne: Victoria University, 1996
75. Cunningham LN. Physiologic characteristics and team performance of female high school runners. Ped Exerc Sci 1989; 1: 73-9
76. Blessing DL, Keith RE, Williford HN, et al. Blood lipid and physiological responses to endurance training in adolescents. Ped Exerc Sci 1995; 7 (2): 192-202
77. Docherty D, Wenger HA, Collis ML, et al. The effects of variable speed resistance training on strength development in prepubertal boys. J Hum Mov Stud 1987; 13: 377-82
78. Rimmer JH, Looney MA. Effects of an aerobic activity program on the cholesterol levels of adolescents. Res Q Exerc Sport 1997; 68 (1): 74-9
79. Eliakim A, Raisz LG, Brasel JA, et al. Evidence for increased bone formation following a brief endurance-type training intervention in adolescent males. J Bone Miner Res 1997; 12 (10): 1708-13
80. Ewart CK, Young DR, Hagberg JM. Effects of school-based aerobic exercise on blood pressure in adolescent girls at risk for hypertension. Am J Public Health 1998; 88 (6): 949-51
81. Andersen LB. Blood pressure, physical fitness and physical activity in 17-year-old Danish adolescents. J Intern Med 1994; 236 (3): 323-9
82. Baxter-Jones ADG, Helms PJ. Effects of training at a young age: a review of the training of young athletes study. Pediatr Exerc Sci 1996; 8 (4): 310-27
83. Theintz GE, Howald H, Weiss U, et al. Evidence for a reduction of growth potential in adolescent female gymnasts. J Pediatr 1993; 122: 306-13
84. Lindholm C, Hagenfeldt K, Ringertz B. Pubertal development in elite juvenile gymnasts: effects of physical training. Acta Obstet Gynecol Scand 1994; 73: 269-73
85. Pigeon P, Oliver I, Charlet JP, et al. Intensive dance practice: repercussions on growth and puberty. Am J Sports Med 1997; 25 (2): 243-7
86. Bass S, Bradney M, Pearce G, et al. Short stature and delayed puberty in gymnasts: influence of selection bias on leg length

and the duration of training on trunk length. J Pediatr 2000; 136 (2): 149-55
87. Malina R, Katzmarzyk PT, Bonci CM, et al. Family size and age at menarche in athletes. Med Sci Sports Exerc 1997; 29 (1): 99-106
88. Peltenburg A, Erich W, Zonderland M, et al. A retrospective growth study of female gymnasts and girl swimmers. Int J Sports Med 1984; 5: 262-7
89. Rowland TW. Intensive sports training in children: a search for guidelines. In: Chan KM, Micheli LJ, editors. Sports and children. Hong Kong: Williams & Wilkins, 1998: 162-77
90. Micheli LJ. Overuse injuries in the young athlete: stress fractures. In: Bar-Or O, editor. The child and adolescent athlete. Oxford: Blackwell Science, 1996: 189-201
91. Rowland TW. The physiological impact of intensive training on the prepubertal athlete. In: Cahill BR, Pearl AL, editors. Intensive participation in children's sport. Champaign (IL): Human Kinetics, 1993: 167-93
92. Malina RM. Physical growth and biological maturation of young athletes. Exerc Sports Sci Rev 1994; 22: 389-433
93. DiFiori JP. Overuse injuries in children and adolescents. Physician Sports Med 1999; 27 (1): 1-11
94. Kibler WB, Chandler TJ. Musculoskeletal adaptations and injuries associated with intense participation in youth sports. In: Cahill BR, Pearl AJ, editors. Intensive participation in children's sports. Champaign (IL): Human Kinetics, 1993: 203-16
95. Micheli LJ, Wood R. Back pain in young athletes: significant differences from adults in causes and patterns. Arch Ped Adol Med 1995; 149: 15-8
96. Bruckner P, Khan K. The younger athlete. In: Clinical sports medicine. Sydney: McGraw-Hill Book Company, 1993; 521-40
97. Collins HR, Evarts CM. Injuries to the adolescent athlete. Postgrad Med 1971; 49: 72-8
98. Caine D, Howe W, Ross W, et al. Does repetitive physical loading inhibit radial growth in female gymnasts? Clin J Sport Med 1997; 7 (4): 302-8
99. Segesser B, Morscher E, Goesele A. Lesions of the growth plate caused by sports stress. Orthopade 1995; 24 (5): 446-56
100. Current comment from the American College of Sports Medicine: the prevention of sport injuries of children and adolescents. Med Sci Sports Exerc 1993; 25 (8 Suppl.): 1-7

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