Review

Growth, pubertal development, skeletal maturation and bone mass acquisition in athletes

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ABSTRACT

The genetic potentials for growth can be fully expressed only under favourable environmental conditions. Excessive physical training may negatively affect growth, especially during puberty. Sports that require a strict control of energy input in the presence of a high energy output are of particular concern. In gymnastics, a different pattern in skeletal maturation was observed, leading to an attenuation of growth potential in Artistic Gymnasts (AG), more pronounced in males than in females, whereas in female Rhythmic Gymnasts (RG) the genetic predisposition to growth was preserved because of a late catch-up growth phenomenon. In all other sports not requiring strict dietary restrictions, no deterioration of growth has been documented. Intensive physical training and negative energy balance modify the hypothalamic pituitary set point at puberty, prolong the prepubertal stage and delay pubertal development and menarche in a variety of sports. In elite RG and AG the prepubertal stage is prolonged and pubertal development is entirely shifted to a later age, paralleling the bone age rather than the chronological age. Bone formation, and, consequently, BMD are enhanced by physical activity. In athletes, high-impact loading activities have been shown to improve BMD, while in sports requiring a lean somatotype, the delay in skeletal maturation and pubertal development, resulting from hypoenestrogenemia, predisposes athletes to osteopenia. In AG, an increase in bone density is observed using the bone age as denominator.

Key words: Athletes, Gymnasts, Growth, Pubertal development, Skeletal maturation, Bone acquisition.

INTRODUCTION

Growth and physical maturation are dynamic processes influenced by a variety of genetic and environ-

mental factors. Although traditionally the main focus of growth evaluation has been placed on the assessment of stature, changes in body composition, in body proportions, in skeletal maturation and in pubertal development also constitute essential components in the evaluation of the growth process. Therefore, growth and physical maturation should be viewed as a complex and dynamic process that includes a broad spectrum of cellular and somatic changes.

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Genetic predisposition to growth can be fully expressed only under favourable environmental conditions¹. Environmental factors can act independently or in combination to modify an individual's genetic potentials. Athletic training and performance when exerted during childhood and adolescence have a tremendous impact on growth and maturation.

There is strong evidence that moderate exercise is an important health habit. At the same time, there is a general concern regarding the influence of stress and intensive physical training on general health. Beginning at a young age, athletes performing at a high agonistic level are exposed to high levels of physical and psychological stress from the many hours of intense training and competitions. The detrimental effects of these factors on growth, skeletal maturation, and pubertal development have been documented in individuals involved in a variety of sports. Individual sports exert unique influences on biological maturation that are related to the heterogeneity of the sport-specific demands of training and the stage of growth and sexual maturation of the individual athlete. Therefore the whole picture is a complex one and should be approached with extreme caution.

SOMATIC GROWTH IN ATHLETES

The major determinant of linear growth is genetic predisposition. Both adult final height and the rate of growth are significantly influenced by genetic factors². Studies in twins revealed that the average difference in final height between monozygotic twins was less than 3 cm, compared to 12 cm for dizygotic twins³. Final height is best correlated with target height (mid-parental height), especially when parents are not of disparate heights⁴. Heredity and environment continuously interact throughout the entire period of growth. Children with similar genetic predisposition should reach comparable final height under optimal environmental conditions. By contrast, children with the same genetic background when exposed to entirely different environmental conditions, can reach a different adult height. Children of parents from underdeveloped areas, born and bred in industrialized countries, present higher adult height than their target height. In industrialized countries, an increase in height has been observed which has been attributed to the improvement of the socio-economic conditions¹.

Growth is a complex process and gene expression is maximally achieved only when favourable conditions operate throughout the entire period of growth. Among the environmental factors that can alter linear growth are physical exercise and stress. The effect of stress and intensive physical training on growth is related to the combined effects of intensity, frequency, and duration of prolonged exercise. Intensive athletic training of 18 hours per week is capable of attenuating growth⁵. Moderate physical activity has beneficial effects on growth, as it is associated with cardiovascular benefits and favourable changes in body composition. Extensive physical training, however, may negatively affect growth, especially during puberty⁶. The impact of intense physical training on growth depends on a variety of factors including the type of physical training, the age of training initiation, and the intensity of training. Each sport requires a specific type of exercising and is characterized by specific athletic requirements that favour a particular optimal somatotype.

The time of maximum training during the growth process is particularly important. For example, in female gymnasts, the maximum intensity of training coincides with the period of pubertal development while in males, the greatest physical exertion is required towards the end of puberty. The intensity of training, within the same sport, has substantially increased over the past few decades due to the demands for higher records. For example, it is known that gymnasts are trained much more intensely nowadays than previously, usually 26-28 hours per week compared to 15 h during the seventies and 20 h during the eighties.

Sports that require a strict control of energy input in combination with a high energy output are of particular concern. Therefore, it is not reasonable to generalize when seeking to identify the particular impact of each sport's activity on linear growth. The athletes that require intensive physical training during childhood and adolescence are mainly gymnasts (both Rhythmic and Artistic) and, to a lesser extent, wrestlers, rowers, track athletes, tennis players, and swimmers.

Rhythmic Gymnastics (RG) and Artistic Gymnastics (AG) are two distinct sports within the field of gymnastics. Their programme includes specific gymnastics and requires specialized and distinct skills. Each sport is characterized by specific athletic requirements

that favour a particular optimal somatotype. A short-limbed individual would have a greater mechanical advantage in artistic gymnastic performance, while a long-limbed individual could benefit from a similar advantage in rhythmic gymnastics. Indeed, performance scores in elite female artistic gymnasts are negatively correlated with the degree of fatness or endomorphy of the individual⁷. It is reasonable, therefore, for trainers (coaches) to select those individuals who best match the appropriate anthropometric criteria for each sport. The sport-specific selection criteria for artistic gymnastics suggests that a short stature with relatively short limbs, broad shoulders, and narrow hips is derived from genetic predisposition rather than a result of the specific sport activity⁸. Therefore, genetic predisposition should always be taken into account when studying the impact of gymnastics on growth.

AG ATHLETES

In AG, earlier anthropometric measurements and prospective growth predictions appeared within normal limits⁹⁻¹². In all these reports the adult height of AG remained proportional to the reported target height regardless of the method used to estimate predicted adult height. In another prospective study¹³, evidence for a reduction of growth potential and a decrease in mean height predictions over time was provided in a smaller group of AG. However, in another study by the same group¹⁴, it was reported that the predicted adult height was not reduced in AG, which demonstrates the inherent inaccuracy of height predictions. Although these data are highly informative, no definite conclusions should be drawn unless adult height has been attained.

In a large cross-sectional study, we have shown that female AG were shorter and lighter than average, with

mean height and weight SD scores below 0 (Table 1), in harmony with their respective target height SD score, which was also below 0¹⁵. However, the actually measured height was lower than the Target height (Table 1). Among factors that negatively affected height in AG were low weight, low body fat, and intensity of training.

Most previously reported data referred only to female AG. We, therefore, evaluated comparatively both male and female AG¹⁶. At the time of examination, both female and male AG were shorter than their age-related population mean and they both presented with a considerable delay in skeletal maturation. However, female AG showed a greater height deviation from their age-related population mean, with a greater delay in their skeletal maturation. Male AG, however, who presented with a height closer to their age-related population mean, had a genetic predisposition towards a much higher final height than female AG (Table 2). Therefore, the difference between target height and actual height SD score (D Target height – actual Height SD score) was greater in males than in females. It is thus reasonable to assume that, in male and female artistic gymnasts, the growth process in males might be more vulnerable to the detrimental effects of intensive physical training.

For both sexes, the measured current height was correlated to target height, a finding indicating that genetic predisposition to growth, although altered, was not disrupted. In a smaller subgroup of athletes, in whom final adult height was available for both sexes, final height fell short of genetic predisposition, providing additional evidence for growth deterioration in AG.

RG ATHLETES

In Rhythmic Gymnastics, female RG were taller

Table 1. Derived height and weight data (Mean $\pm$ SD) of examined Rhythmic and Artistic Gymnasts¹⁶.

Variable	Rhythmic		Artistic	
	Mean $\pm$ SD	n	Mean $\pm$ SD	n
Height SD score	+0.70 $\pm$ 0.8	129	-1.27 $\pm$ 1.1	142
Reported Target height (cm)	165.0 $\pm$ 4.7	121	161.1 $\pm$ 4.7	124
Target height SD score	+0.48 $\pm$ 0.8	121	-0.18 $\pm$ 0.8	125
Δ Height-Target height SD score	+0.10 $\pm$ 0.9	121	-0.89 $\pm$ 1.9	125
Weight SD score	-0.95 $\pm$ 0.6	129	-0.92 $\pm$ 0.7	142

Table 2. Collected somatometric data (Mean $\pm$ SD) of male and female Artistic Gymnasts¹⁶.

	Age (years)	Bone age (years)	Δ CA-BA	Height SDS	Weight SDS
Female	15.7 $\pm$ 2.0 n=169	13.4 $\pm$ 1.8 n=138	2.26 $\pm$ 2.2 n=138	-1.52 $\pm$ 1.1 n=168	-1.09 $\pm$ 0.7 n=168
Male	16.9 $\pm$ 2 n=93	16.2 $\pm$ 1.6 n=83	0.76 $\pm$ 1.2 n=83	-0.97 $\pm$ 1.0 n=93	-0.13 $\pm$ 0.7 n=93
p	p < 0.001 t = -5,03	p < 0.001 t = -11,99	p < 0.001 t = 5,78	p < 0.001 t = -3,53	p < 0.001 t = -11,06
	Target Height SDS	Predicted Height SDS	PH-TH SDS	BMI (kg/m ²)	Body fat (%)
Female	-0.18 $\pm$ 0.7 n=123	-0.70 $\pm$ 0.9 n=136	-0.6 $\pm$ 0.9 n=105	19.0 $\pm$ 1.7 n=168	19.5 $\pm$ 4.2 n=160
Male	+1.91 $\pm$ 0.7 n=83	-0.35 $\pm$ 0.8 n=82	-2.3 $\pm$ 0.8 n=77	21.5 $\pm$ 1.7 n=93	10.6 $\pm$ 5.0 n=91
p	p < 0.001 t = -18,9	p = 0.007 t = -2,7	p < 0.001 t = 13,03	p < 0.001 t = -11,5	p < 0.001 t = 14,95

CA; Chronological age, BA; Bone age, PH; Predicted height, TH; Target height.

and thinner than average for age (Table 1)^{17,18}, with height velocity SD score for each age group above 0 at all ages¹⁸. Interestingly, although growth in normal girls comes to an end by the year of 15, in RG growth continued up to the age of 18 (Figure 1). The RG presented a significant delay in skeletal maturation of 1.8 years, which was compensated towards the end of puberty. Their final adult height was identical to the estimated predicted height at first evaluation, and higher than the genetically determined target height, denoting that genetic potentials for final height was not only achieved but even exceeded. Moreover, target height was the only independent parameter which has been proved to positively influence height velocity, therefore genetic predisposition remained the main driving force for the observed efficient catch-up growth. Comparison of AG with RG revealed that their reported target height SD score was similar to their own measured heights (above 0 for the RG and below 0 for the AG), indicating once more the influence of genetic predisposition and preselection (Table 1). RG followed a growth pattern that was higher than their reported target height, while AG exhibited a lower growth pattern.

The delay in skeletal maturation both in AG and RG is probably multifactorial. Low serum concentrations of sex steroids due to a delay in pubertal development, lower GH secretion or a disturbance in insu-

Figure 1. Height velocity mean values per chronological age in Rhythmic Gymnasts. Lines represent the 3rd, 10th, 25th, 50th, 75th, 90th and 97th centiles of height velocity. The dark line represents the height velocity of our examined gymnasts. The dark gray area includes the velocity curves of all children who have their peak velocities up to two standard deviations of age before and after this average age. The arrows and diamonds mark the 3rd, 50th, and 97th centiles of peak velocity when the peak takes place at these early and late limits¹⁸.

lin-like growth factor homeostasis¹⁹ are capable of altering the hormonal control of growth. Intensive physical training, chronic psychological stress, and modifications in nutrition resulting in inadequate energy intake relative to energy output^{20,21} are well known factors. Nevertheless, overuse lesions of growth plates,

especially in the lower limbs, could add an additional end-organ effect²².

In conclusion, studies in gymnastics of the highest competitive level have revealed, a deteriorating of growth potential in AG that is more pronounced in males than in females, while in female RG the genetic predisposition to growth was promoted.

OTHER SPORTS

In all other sports, no deterioration of growth has been reported. In swimmers, probably due both to preselection bias by trainers and to high energy input, the measured height was well above the population mean²³⁻²⁵. In young distance runners, mean height for both males and females approximate the reference medians and estimated height velocities were, on average, similar to age and sex-specific population means²⁶. Girls training for approximately 12 hours per week in sports including rowing, track, and swimming for an average of 4 years during puberty presented no difference in height velocity from their population means, although a tendency towards a slightly later peak height velocity was noted²⁷. Female swimmers training for 8 hours/week presented at a follow-up of 2-3 years normal heights and normal height velocities compared to their population means, while AG training for 22 hours/week showed significantly lower growth velocities¹³. No difference in growth has been found between seasonal wrestlers and controls as all changes in dietary intake, body composition, and muscular strength were reversed during the post-seasonal period²⁸. Growth rate was assessed as normal in a large cohort of school wrestlers, although no information was provided as to whether or not a lower growth rate was observed during the sport season followed by a catch-up growth during the nontraining season²⁹. Anthropometric characteristics showed the male rowers to be similar in most respects to a student control sample³⁰, while data on a large sample of elite junior rowers showed a tendency towards a taller height, more pronounced among finalists, compared to non-finalists indicating the influence of preselection bias³¹.

In conclusion, intensive physical training did not negatively affect somatic growth in sports not requiring strict dietary restrictions which lead to energy imbalance. Caution should be exercised in the case of elite Artistic Gymnasts of both sexes engaged in highly

strenuous competitions.

PUBERTAL DEVELOPMENT IN ATHLETES

Growth specifically refers to increase in body size, whereas maturation refers to the progress towards the biologically mature state. Therefore, growth cannot be fully assessed without determining the timing and tempo of biological maturation. Puberty is a dynamic period of development with rapid changes in body size, shape, and composition. The onset of puberty corresponds to a specific biological age, as determined by skeletal maturation, namely, a bone age of 13 years for boys and 11 years for girls³². Intensive physical training has profound effects on skeletal maturation leading to a significant delay in bone age compared to chronological age. As in the general population, pubertal development in highly trained athletes seems to follow bone age rather than chronological age³³. However, genetic predisposition and interindividual variation should always be considered. Certain sports offer advantages to early maturers, and others, like gymnastics, favour the later developing individuals. Therefore, any assessment of sexual maturation must consider the biological indicators of bone age and peak height velocity.

Delay in pubertal development and sexual maturation has been documented in various types of athletes, mainly gymnasts, dancers, and long-distance runners³⁴. The observed delay is related to the type, the intensity, the frequency and the duration of exercise and is more pronounced in sports requiring strict dietary restrictions that lead to a deficient energy input in the face of higher energy output. For example, Gymnasts competing in the Olympic Games have been shown to have delayed menarche compared to high school, college, and club-level athletes⁷. Young women or adolescents engaged in sports requiring training less than 15 hours per week do not show menstrual disturbances or delay in sexual maturation³⁵.

In elite RG and AG the prepubertal stage was prolonged and pubertal development was shifted to a later age, maintaining a normal rate of progression (Table 3)¹⁵⁻¹⁸. As expected, the progression of puberty followed the bone age rather than the chronological age^{16,17}. It is noteworthy that for both RG and AG, pubertal progression, although delayed, was not prolonged. Normal girls require an average of 1.96 ± 0.93

years (mean $\pm$ SD) for their breast development to progress from Tanner stage II to Tanner stage IV³⁶. A similar period of time was observed for both RG and AG in our study. Thus, pubertal development was entirely shifted to a later age, maintaining a normal rate of progression.

The major factor responsible for the delay in the onset of breast and pubic hair development in both sports was low body weight. Low body weight reflects an energy deficit, evident in both sports as a consequence of intensive physical training (high energy output) on the one hand and low calorie diet (low energy input) on the other. Gymnasts indeed are subjected to a significant energy drain occurring early in preadolescence and are highly motivated to maintain low body weights due to their sports requirements for a thin somatotype.

In ballet dancers subject to high energy drain and low diet intake, a delayed thelarche and a normal pubarche was noted³⁷. This implies that independent central mechanisms are involved in triggering these two aspects of pubertal development. Indeed, breast development and subsequent menarche are related to estrogen levels, while pubarche is mainly related to adrenal androgen production³⁸. In conditions of energy deficit and consequent reduction in adipose tissue, estrogen production is decreased and breast development and menarche are delayed. It is the onset and the amount of energy deficit that dictates the degree of involvement of all aspects of pubertal development.

Table 3. Mean age $\pm$ SD of Rhythmic Gymnasts and Artistic Gymnasts at the various Tanner stages of breast and pubic hair development¹⁵.

Variable	Rhythmic Gymnasts		Artistic Gymnasts	
	Mean $\pm$ SD	n	Mean $\pm$ SD	n
Breast development				
II	15.5 $\pm$ 0.7	2	15.9 $\pm$ 0.9	13
III	16.6 $\pm$ 1.5	34	16.9 $\pm$ 1.3	33
IV	17.0 $\pm$ 1.3	48	17.5 $\pm$ 1.5	27
V	17.8 $\pm$ 1.3	37	18.1 $\pm$ 1.3	9
Pubic hair development				
II	15.0	1	15.8 $\pm$ 0.7	12
III	16.5 $\pm$ 1.2	22	16.8 $\pm$ 1.2	33
IV	17.4 $\pm$ 1.4	41	17.1 $\pm$ 1.5	32
V	17.3 $\pm$ 1.4	52	18.7 $\pm$ 0.5	7

Indeed, ballet dancers with a normal pubarche start their training at the age of 8-9 years of age with only 3.5-7.3 hours/week³⁷, while our examined RG and AG started their training at the age of 6.4-7.7 years of age with more than 30 hours of training per week.

Female athletes involved in a large variety of sports, including runners, swimmers, tennis players, ballet dancers, and gymnasts present a well documented delayed menarche³⁷⁻⁴¹. In RG, menarche was significantly delayed compared to their mothers' and non trained sisters', a finding militating against a genetic predisposition towards delayed menarche (Table 4)¹⁸.

It is well known that a minimum weight for height and a critical lean to fat mass ratio is required for menarche. According to the Frisch theory, the attainment of a critical percentage of body fat lowers the metabolic rate and induces a sensitization of the hypothalamus to gonadal steroids^{42,43}. Indeed, leptin and estrogen production by the adipose tissue play a crucial role in triggering menarche⁴⁴. These modulations reflect a natural adaptation of the body to high energy demands. In AG and RG, low body fat, low body weight (low energy input), and intensive physical training (high energy output) were the major factors influencing menarche. Low body weight, however, remained the most significant factor in delaying the onset of puberty. It is to be noted that in both RG and AG, older athletes without menarche presented lower height, weight, and BMI than their contemporaries with menarche.

In conclusion, in RG and AG intensive physical training and negative energy balance by modulating the hypothalamic pituitary set point at puberty prolong the prepubertal stage and delay pubertal development without affecting the duration of the pubertal process.

BONE ACQUISITION IN ATHLETES

Volumetric Bone Mineral Density (BMD) and skeletal size are similar in prepubertal girls and boys.

Table 4. Age at menarche (mean $\pm$ SD) in elite Rhythmic gymnasts and in their mothers' and sisters¹⁷.

	Gymnasts	Mothers	Sisters
Menarcheal age (years)	14.3 $\pm$ 1.5	13.7 $\pm$ 1.5	13.7 $\pm$ 1.5
p	-	0.008	0.05

Bone mass doubles between the onset of puberty and early adult life⁴⁵. During adolescence, bone mass increases according to chronological age and this increase becomes evident two years earlier in girls than in boys, as also occurs with pubertal onset¹³. Sex steroids are responsible for the maturation and increase in human skeleton and sexual dimorphism after the onset of puberty⁴⁶. Despite the fact that genetic factors considerably influence bone mass, other factors such as nutrition, exercise, various diseases and medicines, age of menarche and normal menstrual cycle affect the acquisition of bone mass⁴⁷.

Environmental factors such as physical activity promote the increase in bone mass during puberty to a greater degree than in adults^{48,49}. Frost has emphasized the role of biomechanical strain in determining the level of bone mass⁵⁰. The higher strain levels associated with growth or extreme physical activity will induce a type of modelling that increases bone mass by accretion on bone surfaces. There is general agreement that physical activity enhances mainly the bone formation and, consequently, increases BMD⁵¹. A key longitudinal study showed that up to 30% of peak bone mass is accrued in the three years surrounding puberty⁵². Subsequent research confirmed the importance of regular activity (and optimal nutrition) over these years, as well as into adolescence. Bailey et al. verified that recreationally active boys and girls achieved 9% and 17% greater total body bone mineral content respectively, as compared to inactive peers⁵³. Controlled exercise intervention trials provide the most stringent test of this relationship and generally confirm that, even in the prepubertal years, vigorous activity programs produce greater increments in bone mineral than those observed in sedentary children^{54,55}. It is noteworthy that those studies demonstrating the greatest impact of activity on bone mineral content or density in children or adolescents involve either a great diversity of recreational activity, high-impact activities (e.g. gymnastics, volleyball), or weight-bearing activities involving frequent, rapid changes of direction (e.g. tennis). Whether the advantage of an active lifestyle during childhood is maintained through the young adult years or affects later fracture risk is not known. The limited information available suggests that this early advantage of higher peak bone mass may be lost in old age^{56,57}. However, encouraging evidence does exist that a higher peak bone mass may be retained if physical activity is maintained, even at re-

duced levels⁵⁸.

Exercise and dietary calcium are vital for bone growth and maintenance of bone mass. Peak bone mass appears to be associated with calcium consumption during growth from early childhood to young adulthood⁵⁹. Although there exists some controversy about the relative importance of calcium intake versus weight-bearing exercise and hormonal factors in optimising bone health, there is no doubt that individuals who consume low levels of calcium-rich foods have on average lower bone mass (and greater risk of fracture) than age-matched individuals who consume adequate or high levels of calcium. These findings have been documented in adolescents and in young women⁶⁰.

In athletes, most reports on bone acquisition refer to a small number of adults, or to small/medium levels of exercise⁶¹⁻⁶³. There are few reports dealing with the effect of prolonged training periods on bone mineralization in children and adolescents. High-impact loading activities such as ice-skating, weight-lifting, soccer, swimming or ballet have shown an improving BMD in the athletes^{64,65}. Studies in prepubertal athletes found that 8 months of weight-bearing activity interventions resulted in greater increases (1.2-5.6%) in BMD^{66,67}. In contrast, in sports requiring a lean somatotype (therefore leading to a negative energy balance), the delay in skeletal maturation and pubertal development predispose athletes to osteopenia and osteoporosis, which along with eating disorders constitute the "female athlete triad". Unfortunately, the vast majority of clinical trials in athletes deal with menstrual disturbances in young adult females and few data are available for adolescents. Bone mass accretion was compromised in late-maturing adolescent girls⁶⁸, and low BMD has been reported in athletes with hypo-estrogenic amenorrhea⁶⁹. These athletes are in danger of failing to attain adequate peak bone mass ending up with significantly lower bone density than normal women.

Little is known about the effect of high level physical exercise on bone acquisition. Artistic gymnastics constitutes a type of exercise with intense mechanical load in the upper and lower limbs and in the trunk, known to exert a beneficial effect on bone density both in adolescent and adult athletes^{70,71}. Recently, a study was carried out on a group of artistic gymnasts of high competition level who began training at a young age and continued their intensive exercise throughout pu-

berty⁷². Bone age was delayed in both males and females, though more pronounced in the latter due to the energy deficit and to the continuous and severe somatic and psychological stress. The increase in bone density was observed clearly when pertaining to bone age, while this increase was not observed when related to the chronological age. This finding was more pronounced in females due to a greater delay in bone maturation (Figures 2 and 3). In females, bone acquisition was proportional to the development of puberty according to Tanner stages of breast development and there was a strong negative influence of early onset of training on bone acquisition. The latter indicates the vulnerability of bone metabolism before the onset of sex steroids production at puberty. In a recent review, it was suggested that exercise training-induced

increase in bone mineralization and strength is dependent on sexual maturity and hypothesized that factors which enhance bone formation such as estrogen, testosterone, GH and IGF-1 in premenarchal years improve the effect of exercise and mechanical loading on bone turnover and mineralization⁷³. However, the influence of the duration of exercise, on bone acquisition cannot be ruled out. The acquisition of bone mass for both sexes was related positively with height, BMI, lean body mass, body fat (only for females), and weight. The latter was the most powerful factor in males. In conclusion, bone acquisition in children and adolescents who are continuously and intensively trained from early ages, follows the normal pattern only when related to bone age and not to chronological age. Early onset of training, continuous and inten-

Figure 2. The bone density (mean+SD values) in high level adolescent female artistic gymnasts according to bone age $\bullet$ and chronological age $\diamond$ (n=120)⁷².

Figure 3. The bone density (mean+SD values) in high level adolescent male artistic gymnasts according to bone age and chronological age (n=68)⁷².

sive exercise, as well as duration of exercise attenuate bone acquisition, at least in adolescent female AG. Moreover, as judged by the range of measured bone density, which is clearly above the corresponding reported levels in matched normal sedentary populations, the excess mechanical load to which these athletes are exposed from a young age exerts its beneficial effect on bone acquisition, leading to a positive net-effect.

CONCLUSIONS

Genetic predisposition to growth can be fully expressed only under favourable environmental conditions. Moderate physical activity has beneficial effects on general health as it is associated with cardiovascular benefits and favourable changes in body composition. Conversely, extensive physical training may negatively affect growth, especially during puberty.

The effect of stress and intensive physical training on growth is related to the combined effects of exercise intensity, exercise frequency, and exercise duration.

Sports that require a strict control of energy input combined with a high energy output are of particular concern. In gymnastics of the highest competitive level, a delay in skeletal maturation was observed, leading to a deterioration of growth potential in AG, more pronounced for males than for females, while in female RG the genetic potentials for growth was finally achieved by compensation through the late catch-up growth phenomenon. In all other sports not requiring strict dietary restrictions, no deterioration of growth has been documented.

Intensive physical training and negative energy balance, by modulating the hypothalamic pituitary set point at the expected age of puberty, prolong the prepubertal stage and delay pubertal development in a variety of sports.

In elite RG and AG the prepubertal stage was prolonged and pubertal development was entirely shifted to a later age, harmonizing with the bone age rather than the chronological age and maintaining a normal rate of progression. Female athletes present a well documented delayed age of menarche, compared to their mothers' and non-trained sisters'.

Genetic factors, sex steroids, and nutrition mainly determine bone acquisition. Environmental factors

such as physical activity mainly enhance the bone formation and consequently BMD. In athletes, high-impact loading activities such as ice-skating, weight lifting, soccer, swimming, and ballet have shown an improving BMD. By contrast, in sports requiring a lean somatotype (therefore associated with a negative energy balance), the delay in skeletal maturation and pubertal development predispose hypo-estrogenic athletes to osteopenia and osteoporosis.

In AG, especially in females, an increase in bone density was clearly observed when related to the bone age, whereas this increase was not observed if related to chronological age. Early onset of training, continuous and intensive exercise, as well as prolonged duration of exercise attenuated the bone acquisition. Nevertheless, the excess mechanical load to which these athletes are exposed from a young age exerts its beneficial effect on bone acquisition, leading to a positive net-effect.

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