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Delayed but Normally Progressed Puberty Is More Pronounced in Artistic Compared with Rhythmic Elite Gymnasts Due to the Intensity of Training

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Context: Elite gymnasts are subjected to intense training, which may alter pubertal development.

Objective: The objective of the investigation was to study the impact of gymnastics on pubertal development in rhythmic (RGs) and artistic gymnasts (AGs).

Design: Evaluation of somatometric parameters, pubertal stage, and intensity of training in the competition field were studied.

Setting: The study was conducted at European and world championships of years 1997–2004.

Subjects: Subjects included 433 elite RGs and 427 AGs, aged 11–23 yr.

Intervention: There were no interventions.

Main Outcome Measures: Mean chronological and bone ages of each pubertal stage and their relation to the intensity of training were measured.

Results: AGs and RGs showed a delay in skeletal maturation (Δ age − bone age, 2.13 and 1.28, respectively; $P < 0.001$). AGs were subjected to higher levels of physical training. Thelarche occurred at 12.9 yr for RGs and 13.2 yr for AGs ($P = 0.003$) and pubarche at 12.5 and 12.9 yr, respectively ($P = 0.002$). Puberty was delayed but normally progressed. AGs entered each pubertal stage later than RGs. The delay was influenced by the amount of energy output. Menarcheal age was 14.6 yr for RGs and 14.9 yr for AGs. Menarche was influenced in AGs by bone age (b = 0.333; t = 2.521; $P = 0.020$), pubarche (b = 0.322; t = 2.401; $P = 0.026$), and body fat (b = −0.458; t = −3.412; $P = 0.003$) and in RGs by bone age (b = 0.378; t = 3.689; $P < 0.001$) and pubarche (b = 0.525; t = 6.017; $P < 0.001$).

Conclusion: In RGs and AGs, pubertal development was shifted to a later age, maintaining a normal rate of progression, which followed the bone age. AGs, who were exposed to a greater and more sustained energy output than RGs, presented a more pronounced delay in both skeletal maturation and pubertal development. **(*J Clin Endocrinol Metab* 90: 6022–6027, 2005)**

IT IS WELL known that the female reproductive system is vulnerable to different kind of stress. Delayed menarche, amenorrhea, or oligomenorrhea occur in 6–79% of female athletes (1). Elite rhythmic gymnasts (RGs) and artistic gymnasts (AGs) are subjected to high levels of physical and psychological stress due to intensive physical training and to psychological stress imposed by athletic 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 (2–6). Individual sports impart 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. Rhythmic and artistic gymnastics are two distinct sports which, although they share several similarities, also have striking differences that are related to unique requirements for these sports. Although both RGs and AGs present lower body weights than the population mean for their respective age groups, RGs are taller than AGs, with lower body fat (7). This is a reflection of not only specific sport requirements but also a self-selection bias by gymnasts and trainers who favor participation to each sport of the athlete with the appropriate somatotype. Their training program includes specific tasks and requires special and different skills such as bars, beam, and vault for AGs, whereas for RGs, the training program reflects that of dancers and includes balls, rope, ribbon, and clubs.

The effects of stress and intensive physical training on growth and pubertal development have been extensively studied in RGs (8, 9) and AGs (7), but still many questions remain to be answered. It is not known whether puberty is equally delayed in all sports and whether this delay is related to the intensity of training or the specific aims and needs of each particular sport or whether the duration of pubertal development is prolonged. Finally, the question whether the pubertal development follows a normal progression when expressed according to bone age remains unanswered.

The aim of the present study was to evaluate pubertal

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Abbreviations: AG, Artistic gymnast; BMI, body mass index; RG, rhythmic gymnast; SDS, SD score; TH, target height.

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development of RGs and AGs and determine whether the different type of gymnastics practiced by these athletes exerts an impact on pubertal development. This study is unique in character because all variables were measured on the field of competition and include high-level athletes exposed to different levels of energy deficit.

Subjects and Methods

All data for this study have been collected during the Rhythmic and Artistic Gymnastics World and European Championships, which took place in different places of the world from 1997 until 2004. The study was conducted under the authorization of the International Federation of Gymnastics and the European Union of Gymnastics. Informed consent was obtained in accordance to article 7 of the medical organization of the official International Federation of Gymnastics competitions, and all athletes participated voluntarily. The study was entirely cross-sectional and included 423 (49.8%) elite RGs and 427 (50.2%) elite AGs, aged 11–23 yr, from 32 different countries that represented all continents. Data concerning growth in RGs and AGs obtained from different population have been reported recently (7, 9).

The study protocol has been published elsewhere (8, 9). Briefly, it included noninvasive clinical and laboratory investigations and the completion of a questionnaire. The clinical evaluation included height and weight measurements and assessment of breast and pubic hair development according to Tanner's stages of pubertal development (10).

The laboratory investigation included determination of body composition by a portable apparatus (Futrex 5000; Futrex, Inc., Gaithersburg, MD), which estimates percent body fat and total body water based on infrared analysis (11), and has been validated to be equivalent to the standard methods of body composition assessment by skin fold measurements (12) and bioimpedance assessments (13).

Skeletal maturation was evaluated from an x-ray of the left hand and wrist under full-body protection against radioactivity. Bone age was determined according to Greulich-Pyle standards (14). Skeletal maturation was considered near total when bone age was greater than 16 yr of age.

All athletes completed a questionnaire that included questions on personal (onset of breast and pubic hair development, age of menarche, onset and intensity of training, number of competitions per year) and family data (maternal age of menarche, parental heights). Intensity of training was expressed as hours of training per week. Each national team has a specific timetable of training before each major competition and a usual one for training between competitions. It is to be noted that the athletes knew with high credibility parental heights and the onset of their menarche as well as the year of maternal and sister's menarche. The reported target height (TH) was estimated using the midparental height as an index of genetic predisposition to adult height. The equation used for reported TH was: TH = (father's height − 13 + mother's height)/2 (2).

Statistical analysis

Height and weight were expressed as the SD score (SDS) of the mean height and weight for age, according to Tanner's standards (10). The Pearson product moment correlation coefficient, with two-tailed test of significance, was used to assess all studied relationships. A multiple regression analysis (ANOVA) was used to ascertain the independent predictive value of each parameter proved to be significant according to Pearson correlation coefficient. The Student *t* test, with two-tailed test of statistical significance, was used to assess the power of all relationships within two groups. Correlations with a critical value of $P < 0.05$ were considered significant. All statistics were performed using SPSS for Windows (version 9.0.1; SPSS, Inc., Chicago, IL).

Results

Anthropometric characteristics

The mean values for collected and derived data are shown in Table 1. As we reported previously (6, 8), RGs were taller than average, with mean height SDS above the 50th percentile, whereas AGs were well below the 50th percentile. RGs presented an actual height SDS above their genetically predisposed target height SDS, whereas AGs presented a significant height deficit, as their actual height SDS was much shorter than their target height SDS (Δtarget height-height SDS: t = 14.579, $P < 0.001$).

TABLE 1. Demographic data of RGs and AGs

Variable	RGs (mean ± SD)	AGs (mean ± SD)
Age (yr)	15.9 ± 2.4	16.2 ± 2.4
P = NS	(n = 423)	(n = 429)
Bone age (yr)	13.5 ± 1.4	13.4 ± 1.2
P = NS	(n = 196)	(n = 139)
Δ Age − bone age	1.28 ± 1.35	2.13 ± 1.31
$P < 0.001$ (t = −5.689)	(n = 196)	(n = 139)
Height SDS	+0.44 ± 0.9	−1.37 ± 1.16
$P < 0.001$ (t = 24.33)	(n = 419)	(n = 429)
Target height (TH) SDS	0.39 ± 0.86	−0.12 ± 0.77
$P < 0.001$ (t = 8.106)	(n = 356)	(n = 321)
Δ Target height − height SDS	0.06 ± 0.97	−1.17 ± 1.21
$P < 0.001$ (t = 14.579)	(n = 353)	(n = 320)
Weight SDS	−0.87 ± 0.6	−1.02 ± 0.7
$P < 0.001$ (t = 3.439)	(n = 415)	(n = 427)
BMI (kg/m^2)	16.9 ± 1.8	19.2 ± 1.9
$P < 0.001$ (t = −17.43)	(n = 418)	(n = 428)
Body fat (%)	15.5 ± 4.6	21.8 ± 5.8
$P = 0.009$ (t = −17.44)	(n = 400)	(n = 388)
No. of competitions/yr	6.8 ± 3.03	7.7 ± 3.7
$P < 0.001$ (t = −3.758)	(n = 358)	(n = 367)
Training intensity (h/wk)	27.1 ± 10.4	29.7 ± 9.04
$P < 0.001$ (t = −3.669)	(n = 364)	(n = 367)
Onset of training (yr)	7.4 ± 2.3	6.5 ± 2.3
$P < 0.001$ (t = 5.488)	(n = 339)	(n = 365)
Menarche (yr)	14.6 ± 1.5	14.9 ± 1.4
P = NS	(n = 201)	(n = 184)
Onset of breast development (yr)	12.9 ± 1.6	13.2 ± 1.2
$P = 0.003$ (t = −2.937)	(n = 285)	(n = 297)
Onset of pubic hair development (yr), $P = 0.002$ (t = −3.103)	12.5 ± 1.4 (n = 299)	12.9 ± 1.2 (n = 314)

NS, Not significant.

In both groups, a significant delay in skeletal maturation was found, much more pronounced for AGs than for RGs (Δage − bone age 2.13 ± 1.31 and 1.28 ± 1.35 respectively, t = −5.689 $P < 0,01$).

It should be noted that AGs presented a significant higher level of physical training in all parameters tested (training expressed as hours per week, number of competitions per year, and onset of training) (Table 1).

Sexual maturation

The distribution of pubertal development according to chronological age is shown in Table 2 for both breast and pubic hair development (Tanner's stages I-V). Mean values are based on cross-sectional individual data obtained at the time of examination and represent the chronological age at a certain stage of pubertal development. The rate of progression of breast development according to Tanner stages was normal for chronological age as well as for bone age. No statistical significant difference was observed in breast Tanner stages according to bone age between RGs and AGs.

TABLE 2. Mean age of RGs and AGs in all Tanner stages of breast and pubic hair development

Tanner stages	RGs (mean ± SD)	AGs (mean ± SD)
Breast development		
I (P = 0.001, t = −4.348)	12.96 ± 1.3 (n = 27)	14.1 ± 1.0 (n = 50)
II (P = 0.001, t = −4.585)	13.6 ± 1.5 (n = 37)	14.8 ± 1.1 (n = 55)
III (P = NS)	14.97 ± 1.8 (n = 78)	15.4 ± 1.3 (n = 71)
IV (P = 0.001, t = −3.387)	16.1 ± 1.7 (n = 112)	16.9 ± 1.8 (n = 104)
V (P = 0.024, t = −2.279)	17.7 ± 2.2 (n = 132)	18.4 ± 2.5 (n = 61)
Pubic hair development		
I (P = 0.006, t = −2.896)	12.8 ± 0.9 (n = 18)	13.7 ± 0.9 (n = 19)
II (P = 0.001, t = −5.673)	13.1 ± 1.2 (n = 36)	4.7 ± 1.0 (n = 34)
III (P = NS)	15.0 ± 2.1 (n = 58)	15.6 ± 1.8 (n = 75)
IV (P = NS)	15.8 ± 2.1 (n = 118)	16.1 ± 1.9 (n = 113)
V (P = NS)	17.6 ± 1.9 (n = 159)	17.8 ± 2.7 (n = 114)

NS, Not significant.

Breast Tanner stage II occurs at a bone age of 12.0 ± 0.89 yr for RGs and 12.08 ± 1.0 yr for AGs, stage III occurs at a bone age of 12.97 ± 1.47 for RGs and 13.35 ± 1.3 for AGs, stage IV occurs a 14.01 ± 1.6 yr for RGs and 14.09 ± 1.66 for AGs and stage V a 15.3 ± 1.7 yr for RGs and 14.2 ± 1.6 yr for AGs. The reported age of breast and pubic hair development is presented in Table 1. The time of pubarche and thelarche in AGs was delayed, compared with RGs (t = −3.103, P = 0.002 and t = −2.937, P = 0.003, respectively). AGs entered each stage of pubertal level much later than RGs (Table 2).

The age of recalled menarche was 14.6 ± 1.5 yr for RGs and 14.9 ± 1.4 yr for AGs (ns). Concerning RGs over the age of 16.5 yr, 19 of 94 athletes (16.8%) had no menarche yet, whereas in AGs over the age of 16.5 yr, the percentage of athletes without menarche was 19.6% (28 of 115) (P = ns). Menarche was assigned by breast Tanner stage IV in the majority of athletes in both groups.

Maternal age of menarche in RGs was 13.9 yr and in AGs 14.5 yr (t = −3.669, P < 0.001), whereas menarche was significantly delayed in both AGs and RGs with respect to their mothers' menarche (P = 0.05 and P < 0.001, respectively).

Sister's age of menarche in RGs was 13.47 yr and in AGs 13.82 yr. Menarche was significantly delayed in RGs with respect to their untrained sister's (t = 5.004, P < 0.001) as well as in AGs (t = 4.814, P < 0.001).

Relationships

All correlations concerning the onset of breast development for both RGs and AGs are presented in Table 3, pubic hair development in Table 4, and menarche in Table 5.

A multiple regression analysis (ANOVA) was used to ascertain which of the above parameters had independent value in predicting the onset of breast and pubic hair development and the age of menarche for both RGs and AGs. Assuming that menarche and the onset of breast development are factors reciprocally influenced, the onset of breast development was not included in multiple regression analysis. It was found that in AGs (r^2 = 0.647), the main factors influencing menarche were the onset of pubic hair development (b = 0.322, t = 2.401, P < 0.026), bone age (b = 0.333, t = 2.521, P < 0.020), and body fat (b = −0.458, t = −3.412, P < 0.003), whereas in RGs (r^2 = 0.505), the main factors influencing menarche were the onset of pubic hair (b = 0.525, t = 6.017, P < 0.001) and bone age (b = 0.378, t = 3.689, P < 0.001).

In RGs and AGs more than 1 SD older than the mean age of menarche (over the age of 16.5 yr) the Student t test was applied to ascertain differences in their demographic data. Menstruating RGs had higher SDS height (0.87 ± 0.85 *vs.* 0.15 ± 0.77, t = 3.337, P < 0.001), SDS weight (−0.58 ± 0.51 *vs.* − 1.22 ± 0.49, t = 4.991, P < 0.001), body mass index (BMI) (18.3 ± 1.54 *vs.* 16.68 ± 1.26, t = 4.291, P < 0.001), and bone age (16.29 ± 0.77 *vs.* 14.18 ± 1.22, t = 5.639, P < 0.001) than those without menarche. The same differences were observed among AGs with and without menarche. Menstruating AGs had higher SDS height (−0.73 ± 0.99 *vs.* − 1.43 ± 0.71, t = 2.443, P = 0.016), SDS weight (−0.50 ± 0.58 *vs.* − 1.12 ± 0.6, t = 5.000, P < 0.001), BMI (20.38 ± 1.67 *vs.* 19.38 ± 1.67, t = 4.095, P < 0.001), and bone age (14.95 ± 1.65 *vs.* 13.37 ± 1.66, t = 3.202, P < 0.001) than those without menarche.

In RGs (r^2 = 0.691), the main factors influencing the onset of breast development were the SDS weight (b = −0.29, t = −4.41, P < 0.001), intensity of training (b = 0.12, t = 3.889, P = 0.0001), bone age (b = 0.29, t = 4.647, P = 0.001), and onset of pubic hair development (b = 0.645, t = 11.47, P = 0.001). In AGs (r^2 = 0.394), the main factors influencing the onset of breast development were the intensity of training

TABLE 3. Onset of breast development in RGs and AGs: correlation coefficients

Variable	RGs	AGs
Height SDS	n = 282, r = −0.02, P = 0.727	n = 248, r = −0.15, P = 0.015
Weight SDS	n = 278, r = −0.25, P < 0.001	n = 296, r = −0.11, P = 0.055
BMI (kg/m^2)	n = 268, r = −0.01, P = 0.928	n = 297, r = −0.01, P = 0.819
Body fat (%)	n = 268, r = −0.14, P = 0.022	n = 265, r = −0.09, P = 0.157
No. of competitions/yr	n = 265, r = 0.05, P = 0.379	n = 265, r = −0.00, P = 0.962
Training intensity (h/wk)	n = 268, r = 0.23, P < 0.001	n = 284, r = 0.23, P < 0.001
Onset of training (yr)	n = 259, r = 0.06, P = 0.284	n = 280, r = 0.08, P = 0.208
Menarche (yr)	n = 181, r = 0.62, P < 0.001	n = 156, r = 0.60, P < 0.001
Onset of pubic hair development (yr)	n = 277, r = 0.71, P < 0.001	n = 283, r = 0.64, P < 0.001
Bone age	n = 136, r = 0.24, P = 0.004	n = 113, r = 0.41, P = 0.409

TABLE 4. Onset of pubic hair development in RGs and AGs: correlation coefficients

Variable	RGs	AGs
Height SDS	n = 295, r = 0.04, $P = 0.453$	n = 264, r = −0.12, $P = 0.054$
Weight SDS	n = 291, r = −0.18, $P = 0.002$	n = 313, r = −0.09, $P = 0.087$
BMI (kg/m^2)	n = 294, r = 0.02, $P = 0.682$	n = 314, r = −0.00, $P = 0.875$
Body fat (%)	n = 281, r = −0.12, $P = 0.041$	n = 281, r = −0.00, $P = 0.955$
No. of competitions/yr	n = 277, r = 0.06, $P = 0.540$	n = 300, r = 0.10, $P = 0.084$
Training intensity (h/wk)	n = 280, r = 0.09, $P = 0.135$	n = 298, r = 0.13, $P = 0.024$
Onset of training (yr)	n = 272, r = 0.13, $P = 0.028$	n = 297, r = 0.12, $P = 0.041$
Menarche (yr)	n = 181, r = 0.49, $P < 0.001$	n = 155, r = 0.47, $P < 0.001$
Onset of breast development (yr)	n = 277, r = 0.70, $P < 0.001$	n = 283, r = 0.64, $P < 0.001$
Bone age	n = 142, r = 0.21, $P = 0.014$	n = 113, r = 0.09, $P = 0.366$

(b = 0.144, t = 2.561, $P = 0.01$) and onset of pubic hair development (b = 0.583, t = 10.81, $P = 0.001$).

Discussion

In previous reports from our laboratory, we clearly demonstrated that RGs and AGs exhibit a moderate but significant delay in skeletal maturation and pubertal development (7–9). Intense physical training, low body weight, and low body fat delay pubertal progression in a variety of sport activities including ballet dancers, swimmers, and runners (3–7, 15). The detrimental effects of these factors influence all aspects of puberty including the initiation of pubertal development and its progression from Tanner stage II to Tanner stages IV and V as well as menarche (3–7).

The observed delay is related to the type, intensity, frequency, and duration of exercise and is more pronounced in sports requiring strict dietary restrictions that lead to higher energy output in the face of a deficient energy input. Intensive athletic training of 18 h/wk is capable of attenuating growth (16). For example, AGs competing in the Olympic games have been reported to have delayed menarche, compared with club-level athletes (17). Adolescents engaged in sports requiring training less than 15 h/wk do not show menstrual disturbances or delay in sexual maturation (18). Nevertheless, the intensity of training, even within the same sport, has increased over the past few years due to the demands for higher records. It is known that gymnasts are trained much more intensely today than previously, usually 26–28 h/wk, compared with 15 h during the 1970s and 20 h during the 1980s.

Despite these general similarities, striking and important differences among various sport activities are observed. Individual sports have different impact on biological maturation due to their unique requirements in training, body composition, and onset of physical activity.

Rhythmic and artistic gymnastics are two distinct sports within the area of gymnastics. RGs are taller than AGs, with lower body fat and an older age of onset of training. Besides these differences, among the factors responsible for the delay in the onset of breast development in both sports were the low body weight and intensity of training. Low body weight reflects an energy deficit, evident in both sports as a consequence of intensive physical training (high energy output) on one hand and low caloric diet (low energy input) on the other. Gymnasts are exposed 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. SDS weight in RGs was indeed one of the most important factors influencing breast development, a finding not confirmed for AGs.

The onset of puberty corresponds to a mean specific biological age, as determined by skeletal maturation, occurring at a bone age of 11 yr for girls (19). We have previously shown that both RGs and AGs presented with a delay in skeletal maturation, which led to a prolonged prepubertal stage, whereas the progression of puberty in RGs followed the bone age rather than the chronological age (8, 9). In this study, the difference between chronological age and bone age was greater in AGs with respect to RGs. This difference could explain the delay of breast and pubic hair development in AGs with respect to RGs. Nevertheless, when estimated according to bone age, both RGs and AGs entered puberty at the same time.

Adrenarche, the production of adrenal androgens, generally occurs 1–2 yr before the other hormonal changes of puberty (1). In the present study, AGs followed this pattern, whereas in RGs, the time of thelarche and adrenarche nearly coincided. It is to be noted that for both RGs and AGs, pubertal progression, although delayed, was not prolonged. Normal girls require an average of 1.96 ± 0.93 yr (mean ±

TABLE 5. Time of menarche in RGs and AGs: correlation coefficients

Variable	RGs	AGs
Height SDS	n = 194, r = −0.09, $P = 0.20$	n = 145, r = 0.28, $P = 0.827$
Weight SDS	n = 197, r = −0.15, $P = 0.034$	n = 183, r = 0.04, $P = 0.566$
BMI (kg/m^2)	n = 185, r = −0.11, $P = 0.134$	n = 184, r = −0.05, $P = 0.470$
Body fat (%)	n = 187, r = 0.07, $P = 0.318$	n = 163, r = −0.25, $P = 0.001$
No. of competitions/yr	n = 187, r = 0.12, $P = 0.101$	n = 174, r = 0.16, $P = 0.039$
Training intensity (h/wk)	n = 190, r = 0.29, $P < 0.001$	n = 175, r = 0.09, $P = 0.192$
Onset of training (yr)	n = 187, r = −0.07, $P = 0.318$	n = 170, r = 0.02, $P = 0.804$
Onset of pubic hair development (yr)	n = 181, r = 0.49, $P < 0.001$	n = 155, r = 0.47, $P < 0.001$
Onset of breast development (yr)	n = 181, r = 0.62, $P < 0.001$	n = 156, r = 0.60, $P < 0.001$
Bone age	n = 85, r = 0.29, $P = 0.008$	n = 65, r = 0.31, $P = 0.013$

SD) to progress their breast development from Tanner stage II to Tanner stage IV (20), which is in accordance with the time period derived from cross-sectional data obtained in this study for RGs and AGs. Thus, pubertal development was entirely shifted to a later age, maintaining a normal rate of progression.

It is noteworthy that breast and pubic hair development followed the same pattern of progression, and the delayed onset was mainly influenced by low body weight. In ballet dancers under high-energy drain and low diet intake, a delayed thelarche and a normal pubarche was found (4). This implies that independent central mechanisms are involved in triggering these aspects of pubertal development. It is known that breast development and subsequently menarche are related to estrogen levels, whereas pubarche is mainly related to adrenal androgen production. It is well known that female adipose tissue is a significant extragonadal source of estrogens, converting androgens to estrogens (21). A decreased conversion of androgens to estrone because of decreased adipose tissue in athletes may also contribute to the delayed breast development (22). In AGs as well as RGs, pubarche is influenced by the onset of training. Indeed, ballet dancers with a normal pubarche start training at the age of 8–9 yr of age with only 3.5–7.3 h/wk (4), whereas our athletes started their training at the age of 6.5–7.4 yr with more than 30 h of training per week (23). In conditions of energy deficit combined with a 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. Indeed, AGs, which are exposed to a greater and more sustained energy output than RGs as concluded by the earlier onset of training, the greater intensity of training and the increased number of competitions per year presented a more pronounced delay in all aspects of pubertal development.

Female athletes involved in a large variety of sports including runners, swimmers, tennis players, ballet dancer, and gymnasts presented a well-documented delayed menarche (3–9). We previously reported that menarche is significantly delayed in RGs and AGs, compared with their mothers' and their untrained sisters', a finding against a genetic predisposition toward delayed menarche in gymnastics (8). The finding of this study that the mothers of AGs had significantly later menarche, compared with those of RGs, in conjunction with the observation that TH in AGs is significantly lower than in RGs strongly suggest that in AGs, besides the negative energy balance, genetic predisposition may have contributed to the observed differences. An alternative hypothesis would have been that the mothers of athletes were themselves former athletes and their delayed menarche was the result of their involvement to sports activities.

It is well known that a minimum weight for height and a critical lean to fat ratio are required for the induction of menarche. According to the theory of Frisch and colleagues (24, 25), the attainment of a critical percentage of body fat lowers the metabolic rate and induces a desensitization of the hypothalamus to gonadal steroids. Indeed, leptin and estrogen production by the adipose tissue plays a crucial role in triggering menarche, reflecting a natural adaptation of the body to high-energy demands (26). Recent experiments have shown that functional menstrual disorders are part of a catabolic response on the face of an energy deficit. The different stressors involved in any process of high-energy output on the face of inappropriate low-energy input exert their effect through the impact of their energy cost on energy availability (27). In this study, low body fat, low body weight (low energy input), and intensive physical training (high energy output) were among the factors influencing menarche. For AGs, low body fat (low energy input) and the number of competitions per year (high energy output) influenced menarche. Indeed, previous studies have attributed the intensive physical training as a causative factor for the menarcheal delay (5, 28). Low body weight and the intensity of training remained the most predictive factors in triggering the onset of puberty in RGs. It is to be noted that in both RGs and AGs, the older athletes without menarche were shorter and lighter and had lower BMI, compared with those with menarche. Therefore, these factors play an important role for the initiation and the completeness of puberty. Bone age significantly influenced the time of menarche for both RGs and AGs. Bone age also remained one of the most important factors influencing menarche in both RGs and AGs.

We must take notice of a limitation of this study. The limitation is that by necessity we have to use self-reported rather than objective data of recalled menarche, mother's menarche, and age of breast and pubic hair development as well as parental height.

In conclusion, in RGs and AGs intensive physical training and negative energy balance by modulating the hypothalamic pituitary set point at puberty prolonged the prepubertal stage and delayed pubertal development, which followed the retarded bone age rather than the chronological age. Menarche was influenced by the delay in skeletal maturation and somatometric (low body weight and body fat) and sport-related (intensive physical training) parameters. The earlier onset and intensity of training were important factors for the delay in pubertal initiation. The greater and more sustained energy output, as judged by the intensity of training, in AGs, compared with RGs, led to a more pronounced delay in both skeletal maturation and pubertal development. Different sports exhibit different demands and different energy intake and drain that could influence independently and in different ways the normal pubertal development.

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References

1. **Warren MP, Perlroth NE** 2001 The effects of intense exercise on the female reproductive system. J Endocrinol 170:3–11
2. **Bernink MJE, Erich WBM, Peltenburg AL, Zonderland ML, Huisveld IA** 1983 Height, body composition, biological maturation and training in relation

to socio-economic status in girl gymnasts, swimmers and controls. Growth 47:1–12
3. **Marcus R, Cann C, Madvij P, Minkoff J, Goddard M, Bayer M, Martin M, Gaudiani L, Haskell W, Genant H** 1985 Menstrual function and bone mass in elite women distance runners. Ann Intern Med 102:158–163
4. **Warren MP** 1980 The effects of exercise on pubertal progression and reproductive function in girls. J Clin Endocrinol Metab 51:1150–1157
5. **Baxter-Jones ADG, Helms P, Baines-Preece J, Preece M** 1994 Menarche in intensively trained gymnasts, swimmers and tennis players. Ann Hum Biol 21:407–415
6. **Peltenburg AL, Erich WBM, Bernink MJE, Zonderland ML, Huisveld IA** 1984 Biological maturation, body composition and growth of female gymnasts and control groups of schoolgirls and girls swimmers, aged 8 to 14 years: a cross-sectional survey of 1064 girls. Int J Sports Med 5:36–42
7. **Georgopoulos NA, Markou KB, Theodoropoulou A, Benardot D, Leglise M, Vagenakis AG** 2002 Growth retardation in artistic compared to rhythmic elite female gymnasts. J Clin Endocrinol Metab 87:3169–3173
8. **Georgopoulos N, Markou K, Theodoropoulou A, Paraskevopoulou P, Varaki L, Kazantzi Z, Leglise M, Vagenakis AG** 1999 Growth and pubertal development in elite female rhythmic gymnasts. J Clin Endocrinol Metab 84:4525–4530
9. **Georgopoulos NA, Markou K, Theodoropoulou A, Vagenakis GA, Benardot D, Leglise M, Vagenakis AG** 2001 Height velocity and skeletal maturation in elite female rhythmic gymnasts. J Clin Endocrinol Metab 86:5159–5164
10. **Tanner JM, Goldstein H, Whitehouse RH** 1970 Clinical longitudinal standards for height, weight, height velocity, and the stages of puberty. Arch Dis Child 51:170–179
11. **Lukaski HL** 1987 Methods for the assessment of human body composition: traditional and new. Am J Clin Nutr 46:537–556
12. **Hicks VL, Stolarczyk LM, Heyward VH, Baumgartner RN** 2000 Validation of near-infrared interactance and skinfold methods for estimating body composition of American Indian women. Med Sci Sports Exerc 32:531–539
13. **Fornetti WC, Pivarnik JM, Foley JM, Fiechtner JJ** 1999 Reliability and validity of body composition measures in female athletes. J Appl Physiol 87:1114–1122
14. **Greulich WW, Pyle JI** 1959 Radiographic atlas of skeletal development of hand and wrist. 2nd ed. Palo Alto, CA: Stanford University Press
15. **Theintz GE, Howald H, Allemann Y, Sizonenko PC** 1989 Growth and pubertal development of young female gymnasts and swimmers: a correlation with parental data. Int J Sports Med 10:87–91
16. **Tanner JM** 1986 Growth as a target-seeking function: catch-up and catch-down growth in man. In: Falkner F, Tanner JM, eds. Human growth. Vol 1. New York: Plenum; 167–179
17. **Claessens A, Lefevre J, Beunen G, Malina RM** 1999 The contribution of anthropometric characteristics to performance scores in elite female gymnasts. J Sports Med Phys Fitness 39:355–360
18. **Bonen A** 1992 Recreational exercise does not impair menstrual cycles: a prospective study. Int J Sports Med 13:10–120
19. **Tanner JM, Whitehouse RH, Marsall WA, Carter BS** 1975 Prediction of adult height, bone age, and occurrence of menarche, at age 4 to 16 with allowance for midparental height. Arch Dis Child 50:14–26
20. **Marshall WA, Tanner JM** 1969 Variations in pattern of pubertal changes in girls. Arch Dis Child 44:291–303
21. **Perel E, Killinger DW** 1979 The interconversion and aromatization of androgens by human adipose tissue. J Steroid Biochem 10:623–627
22. **Schindler AE, Ebert A, Friedrich E** 1972 Conversion of androstenedione to estrone by human fat tissue. J Clin Endocrinol Metab 35:627
23. **Zacharias L, Wurtman RJ, Shatzoff M** 1970 Sexual maturation in contemporary American girls. Am J Obstet Gynecol 108:833–846
24. **Frisch RE, Revelle R** 1971 Height and weight at menarche and a hypothesis of menarche. Arch Dis Child 46:695–701
25. **Frisch RE, McArthur JW** 1974 Menstrual cycles; fatness as a determinant of minimum weight for height necessary for their maintenance or onset. Science 185:949–951
26. **Moschos S, Chen JL, Mantzoros CS** 2002 Leptin and reproduction; a review. Fertil Steril 77:433–444
27. **Loucks AB, Redman LM** 2004 The effect of stress on menstrual function. Trends Endocrinol Metab 15:466–471
28. **Frisch RE, Gotz-Welbergen AV, McArthur JW, Albright T, Witschi J, Bullen B, Birnholz J, Reed RB, Hermann H** 1981 Delayed menarche and amenorrhea of college athletes in relation to age of onset of training. JAMA 246:1559–1563

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