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PARAMETERS OF BONE TISSUE METABOLISM AND HORMONAL CHARACTERISTICS IN ADOLESCENT ATHLETES WITH FUNCTIONAL HYPOGONADISM

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Introduction. The syndrome of relative energy deficiency (RED-S) in male athletes can lead to the development of functional hypogonadism (FH), characterized by testosterone deficiency and impairments in bone tissue remodeling processes.

Objective. Assessment of the hormonal profile and parameters of bone tissue metabolism in blood serum in adolescent athletes in the setting of FH presence/absence.

Materials and methods. A single-center, cross-sectional study involved 50 adolescent athletes aged 15–18 years (median age 16.5 [15.9; 16.9] years), all members of Russian national teams in three kinds of sport (Greco-Roman wrestling, judo, and football). The participants were divided into two groups based on FH presence (according to the total testosterone level ≤ 12 nmol/L). The FH group included 12 athletes; the comparison group without FH comprised 38 athletes. Serum levels of parathyroid hormone, 25-hydroxyvitamin D, and bone metabolism markers (osteocalcin, C-terminal telopeptide of type I collagen (β -CrossLaps), N-terminal propeptide of type I procollagen), as well as total alkaline phosphatase activity, were determined in all athletes. To assess the hormonal profile, serum levels of luteinizing hormone (LH), follicle-stimulating hormone (FSH), inhibin B, total testosterone, and leptin were investigated. Body composition assessment was performed using bioelectrical impedance analysis. Sexual development was assessed according to the Tanner rating. Statistical data processing was carried out using the Statistica v. 10.0 software package (StatSoft Inc.; USA).

Results. Athletes with FH exhibited a statistically significantly higher percentage of body fat compared to their peers with normal total testosterone levels (13.5 [8.1; 19.9]% and 10.1 [9.0; 12.2]%, respectively, $p = 0.034$). Conversely, the groups did not differ statistically significantly in terms of lean body mass (58.3 [46.5; 82.0] kg and 61.9 [55.4; 67.0] kg, respectively, $p = 0.742$). Athletes with androgen deficiency showed a lower level of LH compared to athletes without FH (1.6 [0.9; 2.9] IU/L and 2.7 [1.7; 3.4] IU/L, respectively, $p = 0.031$), while FSH and inhibin B levels were comparable (3.4 [2.2; 4.2] IU/L and 3.5 [2.1; 5.7] IU/L, respectively, $p = 0.547$; 176.5 [147.0; 248.6] pg/mL and 194.5 [166.0; 231.6] pg/mL, respectively, $p = 0.586$). The levels of bone metabolism markers in athletes with FH did not differ statistically significantly from those in the group of athletes with normal testosterone levels.

Conclusions. The FH development in adolescent male high-performance athletes can be accompanied by gonadostat dysfunction and preserved Sertoli cell function. Androgen deficiency in athletes is associated with an increase in body fat percentage, without changes in lean body mass. In young male athletes, FH does not have a negative impact on bone tissue remodeling processes, given the absence of statistically significant changes in bone metabolism parameters.

Keywords: adolescent athletes; sports medicine; functional hypogonadism; testosterone; osteocalcin; β -CrossLaps; vitamin D

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ПАРАМЕТРЫ МЕТАБОЛИЗМА КОСТНОЙ ТКАНИ И ГОРМОНАЛЬНЫЕ ХАРАКТЕРИСТИКИ НЕСОВЕРШЕННОЛЕТНИХ СПОРТСМЕНОВ С ФУНКЦИОНАЛЬНЫМ ГИПОГОНАДИЗМОМ

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Введение. Синдром относительного дефицита энергии у спортсменов мужского пола может приводить к развитию функционального гипогонадизма (ФГ), сопровождающегося дефицитом тестостерона и нарушениями процессов ремоделирования костной ткани.

Цель. Оценка гормонального профиля и параметров метаболизма костной ткани в сыворотке крови у несовершеннолетних спортсменов в зависимости от наличия ФГ.

Материалы и методы. Проведено одноцентровое одномоментное исследование, включившее 50 юных спортсменов 15–18 лет (средний возраст 16,5 [15,9; 16,9] года), входящих в состав сборных команд Российской Федерации по трем видам спорта (греко-римская борьба, дзюдо, футбол) и разделенных на две группы в зависимости от наличия ФГ (по уровню общего тестостерона ≤ 12 нмоль/л). В группу с ФГ включены 12 спортсменов; в группу сравнения без ФГ — 38 атлетов. У спортсменов в сыворотке крови определяли уровни паратиреоидного гормона, 25-гидроксивитамина D, маркеров метаболизма костной ткани (остеокальцин, С-концевой телопептид коллагена 1-го типа (β -CrossLaps), N-терминальный пропептид проколлагена 1-го типа), а также активность общей щелочной фосфатазы. Для оценки гормонального профиля исследовали уровни лютеинизирующего гормона (ЛГ), фолликулостимулирующего гормона (ФСГ), ингибина В, общего тестостерона и лептина в сыворотке крови. Оценка композиционного состава тела проведена методом биоимпедансного анализа. Половое развитие оценивали по классификации Tanner. Статистическую обработку данных проводили с использованием пакета прикладных программ Statistica v. 10.0 (StatSoftInc., США).

Результаты. У спортсменов с ФГ выявлено статистически значимо большее процентное содержание жировой ткани в организме по сравнению со сверстниками с нормальным уровнем общего тестостерона (13,5 [8,1; 19,9] и 10,1 [9,0; 12,2]% соответственно, $p = 0,034$, в то время как по количеству тощей массы тела исследуемые группы статистически значимо не различались (58,3 [46,5; 82,0] и 61,9 [55,4; 67,0] кг соответственно, $p = 0,742$). У спортсменов с андрогенным дефицитом выявлен более низкий уровень ЛГ по сравнению со спортсменами без ФГ (1,6 [0,9; 2,9] и 2,7 [1,7; 3,4] МЕ/л соответственно, $p = 0,031$) при сопоставимых значениях ФСГ и ингибина В (3,4 [2,2; 4,2] и 3,5 [2,1; 5,7] МЕ/л соответственно, $p = 0,547$; 176,5 [147,0; 248,6] и 194,5 [166,0; 231,6] пг/мл соответственно, $p = 0,586$). Уровни маркеров метаболизма костной ткани у спортсменов с ФГ статистически значимо не отличались от группы спортсменов с нормальным уровнем тестостерона.

Выводы. Развитие ФГ у несовершеннолетних высококвалифицированных спортсменов мужского пола сопровождается нарушением функции гонадостата и сохраненной функцией клеток Сертоли. Андрогенный дефицит у спортсменов ассоциирован с увеличением содержания жировой ткани в организме, но не сопровождается изменением количества тощей массы тела. У юных спортсменов мужского пола ФГ не оказывает негативного влияния на процессы ремоделирования костной ткани, учитывая отсутствие статистически значимых изменений параметров метаболизма костной ткани.

Ключевые слова: юные спортсмены; спортивная медицина; функциональный гипогонадизм; тестостерон; остеокальцин; β -CrossLaps; витамин D

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INTRODUCTION

High-quality and balanced nutrition in terms of quantity and composition is essential for full recovery after training, adaptation to intense physical loads, and prevention of sports injuries. Insufficient dietary intake of energy substrates required to maintain the body's optimal function during sports can lead to the development of the condition referred to as the Relative Energy Deficiency in Sport (RED-s) syndrome [1]. The risk of energy deficiency in male athletes is highest in endurance (marathon, cycling) and weight-category sports (judo, boxing, wrestling, and other combat sports) due to extremely high energy expenditure (endurance sports) and stringent body weight requirements (combat sports) in the aforementioned athletic specializations [2].

In adolescent males, RED-s can be accompanied by androgen deficiency as part of the development of functional hypogonadism (FH) [1, 3]. Furthermore, a decrease in testosterone levels in young male athletes can be observed in cases of overtraining syndrome [4]. It was previously shown that the development of functional hypothalamic amenorrhea in female athletes negatively affects bone structure and mineral density (BMD), being a leading risk factor in low-energy fractures [1, 5]. At the same time, the influence of FH on microarchitecture and BMD in male athletes is less pronounced [1, 3]. However, a number of studies demonstrate the negative impact of androgen deficiency in adult athletes on BMD, reduced performance, as well as increased fatigue and sexual dysfunction [6, 7].

In clinical practice, bone functional status is assessed using bone metabolism markers [8]. The high risk of low-energy fractures in athletes underscores the relevance of studying bone metabolism markers and their relationship with hormonal parameters in adolescent male athletes with FH, given the lack of data on this topic in the literature.

This study is aimed at assessing the hormonal profile and parameters of bone tissue metabolism in the blood serum of adolescent athletes depending on the presence or absence of FH.

MATERIALS AND METHODS

A single-center, cross-sectional study involved 50 male adolescent athletes of three sports specializations: Greco-Roman wrestling, *n* = 14; judo, *n* = 22; football, *n* = 14. The age of participants ranged 15–18 years (median age 16.5 [15.9; 16.9] years); all reached sexual maturity (Tanner IV–V). Due to the completed sexual maturity of the examined athletes, diagnostic criteria for hypogonadism in adult males were applied.

All athletes were divided into two groups based on the presence of functional hypogonadism (FH), diagnosed by a decrease in total testosterone level ≤ 12 nmol/L [9]. The group with FH included 12 athletes (median age 16.2 [15.9; 16.9] years); the comparison group with normal total testosterone levels comprised 38 athletes (median age 16.6 [15.9; 16.9] years).

Anthropometric measurements of the adolescent athletes included: height and body weight measurement, calculation of body mass index (BMI). BMI was assessed for the specific age and sex, presented as the number of standard deviations from the mean (SDS). Body composition assessment was performed using bioelectrical impedance analysis (InBody 570 analyzer,

South Korea). Assessment of the stage of sexual maturity of adolescent athletes was conducted according to the Tanner scale [10].

The inclusion criteria for participation in the study were male athletes from Russian national teams, aged 15–18 years, with achieved sexual maturity (Tanner IV–V). The inclusion criterion for the study group was the presence of FH (decreased total testosterone level ≤ 12 nmol/L). The inclusion criterion for the comparison group was the absence of FH (normal total testosterone level).

Blood was collected in the morning from a peripheral vein after overnight fasting. Serum levels of osteocalcin (Roche, Switzerland), C-terminal telopeptide of type I collagen (β-CrossLaps; Roche, Switzerland), 25-hydroxyvitamin D (25(OH)D; Roche, Switzerland), N-terminal propeptide of type I procollagen (P1NP; Roche, Switzerland), and parathyroid hormone (PTH; Roche, Switzerland) (ng/mL) were determined in all adolescent athletes using an electrochemiluminescence immunoassay. The osteocalcin/β-CrossLaps ratio was also calculated [11]. Leptin (ng/mL), luteinizing hormone (LH, IU/L), follicle-stimulating hormone (FSH, IU/L), and inhibin B (pg/mL) levels were measured in serum using an enzyme-linked immunosorbent assay (ELISA) (BenderMedSystems, Austria). The serum level of total testosterone (nmol/L) was determined by immunoassay on a Lazurit automated analyzer (USA). Total alkaline phosphatase (ALP) activity in serum (U/L) was measured using a kinetic colorimetric method.

Statistical data processing was performed using the Statistica v. 10.0 software package (StatSoft Inc.; USA). Since the studied quantitative parameters had a non-normal distribution (according to the Kolmogorov–Smirnov test), all data are presented as the median (*M*_e)

Table. Clinical characteristics of the studied athlete groups

Investigated parameter, units of measurement	FH Group (<i>n</i> = 12)	Group without FH (<i>n</i> = 38)	Statistical significance level, <i>p</i>
Age, years	16.2 [15.9;16.9]	16.6 [15.9;16.9]	0.682
Height, m	1.70 [1.63; 1.85]	1.73 [1.67; 1.80]	0.733
Height SDS	-0.41 [-1.11; 1.58]	0.32 [-0.85; 0.94]	0.785
Body weight, kg	74.6 [55.2; 105.1]	70.2 [62.3; 75.6]	0.716
BMI, kg/m ²	24.6 [20.1; 29.9]	23.1 [21.4; 24.1]	0.785
BMI SDS	1.04 [-1.65; 2.50]	0.91 [-0.53; 1.35]	0.665
Sport:			
judo	5 (46%)	17 (44%)	0.851
Greco-Roman wrestling	6 (50%)	8 (22%)	0.051
football	1 (4%)	13 (34%)	0.081

Table was compiled by the authors based on their own data

Note: *n* — number of athletes; FH — functional hypogonadism; SDS — standard deviation score; BMI — body mass index; BMI SDS — standard deviation score of body mass index.

with the upper and lower quartiles [Q_1 ; Q_3], corresponding to the 25th and 75th percentiles of the distribution. The Mann–Whitney U test was used to assess the statistical significance of differences in quantitative characteristics. Qualitative characteristics are presented as proportions (%) with absolute values indicated. To assess differences between qualitative characteristics, contingency tables were constructed followed by evaluation using Pearson's chi-square test (χ^2) with Yates' correction. Correlation analysis was conducted using Spearman's rank correlation coefficient. The statistical level of significance for differences was accepted at $p \leq 0.05$.

RESULTS

The studied groups did not differ statistically significantly and were comparable in age ($p = 0.682$) and basic anthropometric parameters (Table). The distribution by sport type in the groups with FH and without androgen deficiency is presented in the table.

An analysis of gonadotropin levels in adolescent male athletes did not reveal statistically significant differences between the studied groups in FSH levels (3.4 [2.2; 4.2] IU/L and 3.5 [2.1; 5.7] IU/L for the groups with FH and without androgen deficiency, respectively, $p = 0.547$). However, adolescents with androgen deficiency showed statistically significantly lower levels of LH and total testosterone compared to athletes without FH (1.6 [0.9; 2.9] IU/L and 2.7 [1.7; 3.4] IU/L, respectively, $p = 0.031$, and 9.3 [8.4; 11.0] nmol/L versus 21.6 [15.6; 25.9] nmol/L, respectively, $p < 0.001$).

When assessing the functional activity of Sertoli cells in the testicular tissue, no statistically significant differences were found between the studied groups of adolescent male athletes in inhibin B levels (176.5 [147.0; 248.6] pg/mL and 194.5 [166.0; 231.6] pg/mL, respectively).

Adolescent athletes with FH were characterized by statistically significantly higher leptin levels of 2.1 [0.8; 5.1] ng/mL compared to the group of athletes without testosterone deficiency — 0.8 [0.5; 1.5] ng/mL ($p = 0.017$).

Correlation analysis revealed a statistically significant inverse relationship between leptin levels and total testosterone ($r_s = -0.48$; $p < 0.05$), as well as between inhibin B and FSH ($r_s = -0.51$; $p < 0.001$).

Assessment of bone tissue metabolism parameters established that the presence of androgen deficiency did not lead to changes in the values of bone formation markers (P1NP, osteocalcin, ALP) or bone resorption markers (β -CrossLaps) compared to peers without FH.

Thus, in the group with FH, the osteocalcin level was 71.0 [38.1; 104.5] ng/mL, P1NP 245.1 [193.4; 516.9] ng/mL, total ALP 150.7 [108.9; 196.7] U/L, β -CrossLaps 1.51 [1.28; 2.3] ng/mL. In athletes

without androgen deficiency, the osteocalcin level was 68.0 [53.7; 88.0] ng/mL, P1NP 322.7 [260.7; 496.0] ng/mL, total ALP 147.2 [114.9; 181.5] U/L, β -CrossLaps 1.82 [1.41; 2.29] ng/mL.

The osteocalcin/ β -CrossLaps ratio in athletes with FH was 38.3 [35.8; 42.2] and did not differ statistically significantly from the values in athletes without androgen deficiency (42.3 [32.5; 49.4]; $p = 0.375$). The obtained values of bone metabolism parameters corresponded to reference intervals developed for athletes [11].

Assessment of vitamin D status in all athletes, regardless of FH presence, revealed low 25(OH)D levels, corresponding to a deficiency state in the body [12]. The studied groups did not differ statistically significantly in PTH levels (4.8 [3.3; 7.0] pmol/L and 4.3 [3.6; 5.4] pmol/L, respectively, $p = 0.699$).

Correlation analysis showed negative relationships between lean body mass in the athletes and levels of P1NP ($r_s = -0.56$; $p < 0.001$), osteocalcin ($r_s = -0.42$; $p = 0.002$), and β -CrossLaps ($r_s = -0.43$; $p = 0.002$).

Assessment of body composition in adolescent athletes with FH (Fig.) revealed a statistically significantly higher percentage (13.5 [8.1; 19.9]% vs 10.1 [9.0; 12.2]%, $p = 0.034$) and absolute (11.7 [5.9; 16.4] kg vs 6.6 [5.9; 8.4] kg, $p = 0.021$) content of body fat compared to the group without androgen deficiency. The groups did not differ statistically significantly in the amount of lean and skeletal muscle mass (Fig.).

Correlation analysis revealed a positive relationship between LH and leptin with the percentage of body fat in athletes ($r_s = 0.58$; $p < 0.001$ and $r_s = 0.38$; $p = 0.006$, respectively), and a negative relationship between the level of total testosterone and the absolute amount of body fat in young athletes ($r_s = -0.34$; $p < 0.05$).

DISCUSSION

The development of RED-s is associated with endocrine disorders and changes in bone metabolism, which increase the risk of fractures in both male and female athletes [11]. Currently, decreased levels of total or free testosterone are considered by the International Olympic Committee expert panel as a significant risk factor for the development of RED-s syndrome and impaired bone remodeling in adolescent male athletes [1].

Our study demonstrated that in adolescent male athletes with FH, the levels of key bone metabolism markers did not differ statistically significantly from peers without testosterone deficiency. This indirectly supports clinical data on the limited influence of androgen deficiency on the mechanisms of impaired bone remodeling in male athletes. The values of the studied bone metabolism markers in both investigated groups ranged within the reference intervals proposed for adolescent athletes with achieved or near-achieved sexual maturity [13]. The osteocalcin/ β -CrossLaps ratio may

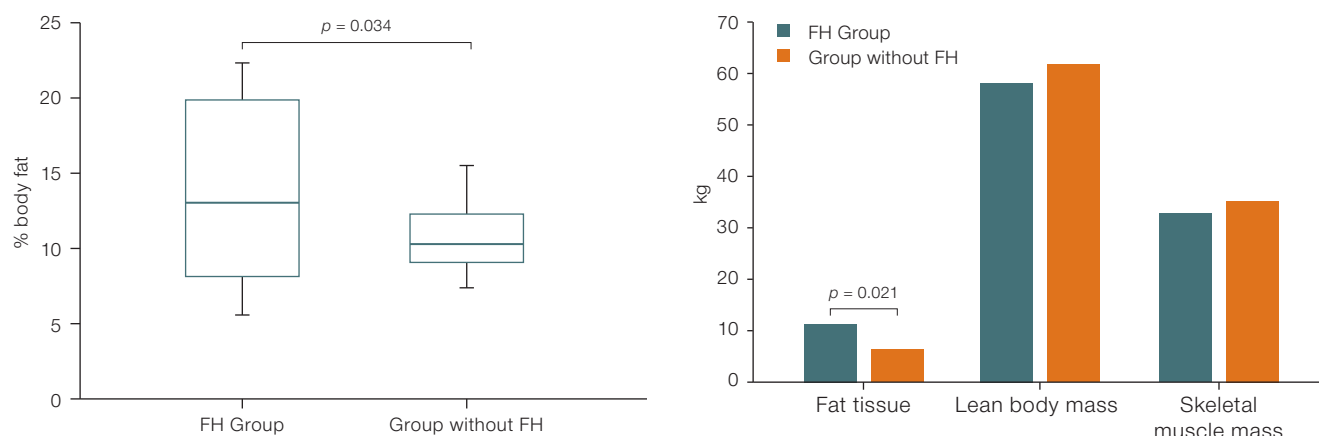


Figure prepared by the authors based on original data

Fig. Parameters of body composition in adolescent athletes depending on the presence of functional hypogonadism

reflect the balance between bone synthesis and re-sorption in athletes [11]. In our study, the ratio of bone marker indices did not differ statistically significantly between the groups with FH and without androgen deficiency and corresponded to the reference intervals proposed by Nikitina et al. [11]. The data obtained may indirectly indicate an absence of bone tissue remodeling disorders in adolescent athletes, regardless of the presence of hypogonadism syndrome.

According to Areta et al., the frequency of low-energy fractures in male athletes is three times lower compared to females [14]. Disruption of bone tissue remodeling processes in females is caused by hypoestrogenism in the setting of functional hypothalamic amenorrhea, whereas the development of FH in males leads to a slight decrease in circulating estrogen levels due to reduced aromatase activity [3]. Given the physiologically lower baseline level of estrogens in men compared to women, the development of pronounced disorders in bone tissue remodeling processes is not typical for the former group.

Assessment of gonadostat function in athletes with androgen deficiency revealed a statistically significantly lower level of LH compared to the group without FH. Reduced pulsatile secretion of LH by adenohypophyseal gonadotrophs is described as one of the key mechanisms in the development of functional hypogonadism within the chronic energy deficiency syndrome in athletes [1]. Normally, by interacting with Leydig cells, LH stimulates testosterone synthesis, and a decrease in LH level causes the androgen deficiency identified in the study group with FH. Prolonged and uncompensated hypogonadism syndrome in males may lead to a decrease in the number of Leydig cells, a reduction in LH receptors on their surface with decreased sensitivity, thus contributing to reduced fertility [15].

Inhibin B is secreted by Sertoli cells under the influence of FSH, acting as a regulator of its secretion via negative feedback. Thus, inhibin B can be used as a marker of spermatogenesis and Sertoli cell function; thus, its decrease is observed in male patients with various spermatogenesis disorders (oligospermia, azoospermia, etc.). Inhibin B levels reach peak values during puberty, around 12–17 years of age [16].

In our study, the levels of inhibin B and FSH in the investigated groups of young athletes did not differ statistically significantly, indicating no influence of androgen deficiency on Sertoli cell function in adolescent athletes. The study design did not allow us to assess the persistence and duration of the identified androgen deficiency in the athletes. Therefore, the absence of changes in Sertoli cell functional activity may be due to a short-term or transient decrease in testosterone levels.

According to Meyer et al., a low body fat percentage (<5%) in adolescent male athletes based on body composition analysis can be considered an indirect sign of insufficient energy intake [17]. None of the athletes we examined had a low body fat percentage. In young athletes with androgen deficiency, a statistically significantly greater amount of body fat was found compared to their peers without FH. The obtained results are consistent with data from other researchers, demonstrating a reduction in body fat in males with normal or elevated testosterone levels [18].

The amount of body fat in adolescent male athletes positively correlated with the level of circulating leptin. This adipokine (adipose tissue hormone) is currently considered an endogenous regulator and modulator of reproductive system functions; however, in male athletes, leptin apparently reflects the dynamics of body fat content to a greater extent and does not have a significant influence on gonadostat function [19]. In our study, the

median leptin level in adolescent male athletes without androgen deficiency was lower compared to the general population norm, which is associated with a lower body fat content in athletes.

At the same time, the studied groups did not differ significantly in the amount of skeletal muscle mass, which is not consistent with the data of Aksenova et al., demonstrating a direct relationship between testosterone levels and an increase in skeletal muscle mass in adolescent athletes [20].

Our study has one important limitation, since the vast majority of athletes in both study groups had vitamin D deficiency or insufficiency, which could have influenced the levels of the analyzed bone metabolism markers. It is known that prolonged, uncompensated vitamin D deficiency leads to increased PTH secretion to maintain calcium homeostasis, which occurs through an intensification of resorptive processes in bone tissue and is accompanied by an increase in bone resorption markers in serum [21]. Assessing bone metabolism markers against a background of adequate vitamin D sufficiency in male athletes with FH would allow for a more precise determination of the influence of androgen deficiency on bone remodeling processes.

Within the scope of this work, we did not investigate the influence of sports type, level of athletic skill, or stage of the training cycle on the studied bone metabolism markers due to the restricted sample size of athletes with FH. Furthermore, the serum level of total testosterone was determined only once as part of the comprehensive medical examination, which does not allow for conclusions regarding the persistence and duration of the identified androgen deficiency.

It can be hypothesized that the decrease in total testosterone levels in adolescent athletes is an adaptive mechanism to high physical loads or short-term energy deficiency (during rapid weight loss in combat sports athletes), which explains the absence of associations between androgen deficiency and body composition parameters or bone metabolism markers.

Given that a decrease in testosterone levels can serve as a marker of overtraining syndrome in athletes [4], special attention should be paid to young athletes with FH to clarify their tolerance to training loads in this group.

Further investigation of the characteristics of the hormonal profile and bone remodeling markers and their influence on BMD may have important practical significance for developing an individualized approach to diagnosing bone remodeling disorders and stratifying the risks of low-energy fractures in adolescent male athletes with functional hypogonadism.

CONCLUSION

Functional hypogonadism in adolescent male athletes does not lead to changes in bone tissue metabolism parameters and does not exert a negative influence on bone remodeling processes. The development of androgen deficiency in young athletes is associated with impaired gonadostat function, rather than with changes in the functional activity of Sertoli cells. The androgen deficiency identified in male athletes with FH is associated with an increase in body fat content, although not being accompanied by a reduction in lean and skeletal muscle mass. Considering the functional nature of androgen deficiency and its lack of impact on bone remodeling processes and fertility prognosis, in our opinion, there is no need to impose restrictions on sports participation for young athletes with FH. However, to ensure the continuation of productive training, special attention must be paid to preventing the development of overtraining syndrome in these young athletes. Furthermore, in this athlete group, it is advisable to continue monitoring hormonal indicators over time, assessing additional markers of overtraining.

Clarifying the persistence of the identified gonadostat dysfunction and androgen deficiency in male athletes with FH requires further research to determine the strategy for their medical and biological support.

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