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Article *in* The Journal of sports medicine and physical fitness · November 2015

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THE JOURNAL OF SPORTS MEDICINE AND PHYSICAL FITNESS
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J Sports Med Phys Fitness 2015 Nov 26 [Epub ahead of print]

THE JOURNAL OF SPORTS MEDICINE AND PHYSICAL FITNESS

Rivista di Medicina, Traumatologia e Psicologia dello Sport
pISSN 0022-4707 - eISSN 1827-1928
Article type: Original Article

The online version of this article is located at http://www.minervamedica.it

Androgen receptor CAG and GGN repeat polymorphisms influence performance in boys and girls.

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Running title: "Androgen receptor polymorphisms and performance in children"

Keywords: Muscle, strength, androgen receptor polymorphisms, fitness, pre and pubertal growth.

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Abstract

Shorter CAG and GGN androgen receptor (AR) repeat polymorphisms are associated with stronger androgen signaling, and therefore, could influence lean mass and exercise performance during growth. Physical fitness and body composition were measured by standardized procedures and the length of CAG and GGN repeats was determined by PCR and fragment analysis in 152 boys (11.5 ± 2.6 years; Tanner ≤ 5) and 116 girls (10.1± 3.2 years; Tanner ≤ 5). Individuals were grouped as CAG short (CAG_S) if harboring repeat lengths of ≤21 and CAG long (CAG_L) if CAG >21. Moreover, subjects were grouped as GGN short (GGN_S) if harboring repeat lengths of ≤23 and GGN long (GGN_L) if GGN >23. No significant differences in anthropometrics and body composition were observed between either CAG_S and CAG_L groups and GGN_S and GGN_L groups. Boys harboring CAG_S completed the 300m test faster than their CAG_L counterparts. Moreover, girls from the GGN_L group showed a significant higher VO_2max than those in the GGN_S group. In summary, carrying a short allele of the androgen receptor CAG repeat polymorphism is associated to higher anaerobic performance in boys, whereas long alleles of androgen receptor GGN polymorphisms are associated to higher aerobic capacity in girls.

Introduction

The androgen receptor (AR), also known as NRC4 (nuclear receptor subfamily 3, group C, member 4), is a type of nuclear receptor activated by transmembrane binding to androgenic hormones such as testosterone or dihydrotestosterone[1]. The AR gene is located on the long arm of chromosome X (Xq 11-12) [2]. This gene consists of 8 exons and encodes a protein having 919 amino acid residues. The exon 1 of this gene has two polymorphic repeats motives (CAG and GGN), which encode polyglutamine and polyglycine respectively, of variable lengths at the N-terminal (transactivation domain) of AR protein [3]. Several studies have shown high expression of ARs in skeletal muscle tissue [4, 5] this being even higher in response to muscle overload [6].

The CAG repeat varies in length or number of repetitions between 8 and 35, with an average of 22. The GGN, which is a complex repeat, is represented as $(GGT_3GGG_1GGT_2GGC)n$ and ranges from 10 to 30 repetitions [3]. The length of CAG repeat polymorphism and AR transactivation potential are inversely correlated [7, 8]. Although GGN polymorphism has been less studied than CAG polymorphism, a short GGN is associated with more androgenic activity in cell cultures [9].

Consequently, a greater androgenic effect is expected for AR with lower CAG and GGN repeat number. In agreement, lower number of CAG repeat polymorphisms has been linked to greater fat-free mass in healthy elders [10]. However, in 282 physically active young men no relationship between physical fitness, muscle mass, levels of free testosterone, osteocalcin and the length of the CAG repeat number has been reported [11, 12].

In contrast to adults, little is known about the potential effect of AR CAG and GGN repeat polymorphisms in the development of physical fitness during growth.

Several traits influencing fitness in children may be modulated by androgens, and therefore, by an enhanced androgen transductional activity facilitated by shorter CAG or GGN AR polymorphisms. For example, free androgen index levels in prepubertal and early pubertal boys engaged in systematic resistance training are shown to be higher than their aerobic training and sedentary counterparts, evidencing the significant effect of a specific training stimulus in this hormone-sensitive phase of maturation [13]. Such associations have not been observed in girls, mainly due to the fact that testosterone is thought not to be the main determinant of muscle growth in girls, even at the pubertal stage [14]. However, the potential benefit of an enhanced androgen signaling in girls during the prepubertal stage is still largely unknown. Some authors suggest that there is a progressive increase in the exposure of peripheral tissues to testosterone, given by the raise in the systemic testosterone levels from 4 to 7 years old [15]. This increment might play a role in the muscle mass development of girls before the onset of puberty, and might be enhanced by shorter CAG AR polymorphisms.

Although less studied, endurance capacity may be also enhanced by the action of androgens. Human lungs express AR [16] and studies performed in mammal lung showed that androgen treatment increases expression of genes involved in oxygen transport, such as those encoding hemoglobin, oxygen binding, heme binding and iron ion binding, therefore suggesting a potential role of enhanced AR signaling and aerobic fitness [17]. Although no differences have been observed in respect to aerobic fitness among prepubertal boys and girls [18], genetic determinants that could influence the oxygen transport system via an enhanced androgenic environment remain unexplored.

Therefore our hypothesis is that short CAG or GGN repeat polymorphisms of the gene encoding the androgen receptor are associated with greater muscle mass, lower fat mass and enhanced fitness in children.

The aim of this study was to determine whether the CAG and GGN androgen receptor repeat polymorphisms are associated with body composition and the physical fitness in children.

Methods

Pre and peripubertal children (152 boys and 116 girls, all Tanner stage <5) agreed to participate in this study. Subjects were recruited from sports clubs in Gran Canaria (Spain) aged between 7 and 13 years. Children with chronic illness of taking medications were excluded. The study was performed in accordance with the Helsinki Declaration of 1975 as regards the conduct of clinical research, being approved by the Ethical Committee of the University of Las Palmas de Gran Canaria. Volunteers and parents were informed about the study risk and benefits and provided written consent before the start of the studies. Pubertal Tanner status was self-assessed [19], using a valid [20] and reproducible (r = 0.97) [21] procedure.

Body composition

Whole body composition was assessed with dual-energy X-ray absorptiometry (DXA; QDR-1500, Hologic Corp., Software version 7.10, Waltham, Massachusetts, USA). Upper and lower limbs lean mass were calculated using the regional analysis feature of the software, as previously described [22, 23].

Vertical jump performance and running sprint tests

The forces generated during vertical jumps were measured with a force platform (Kistler Quattro Jump, Winterthur, Switzerland). During this test, two differing types of

jumps were executed. The first was a squat jump (SJ), performed from a static position with the knees bent at 90º. The second type of jump was a countermovement jump (CMJ) starting from the upright position. The CMJ was executed with a fast downward movement down to a 90º knee flexion, followed by a rapid vertical jump. Jumping height (JH) and the maximal power (Wmax), generated were determined in the best of three trials.

Thirty meters running speed was measured indoor with photocells (General ASDE, Valencia, Spain). Subjects performed three maximal 30m sprints with 5 minute rest periods in between.

Anaerobic Capacity

The anaerobic capacity was estimated using a 300 m running test. This distance was chosen because it has been shown that for all test lasting between 30 and 60 seconds more than 50% of the overall energy yield is supplied by anaerobic energy sources [24]. The test was performed in the 400m track of the University and the time needed to complete the distance was determined using an electronic stopwatch.

Maximal aerobic power

The maximal oxygen uptake (VO_2max) was estimated using the 20m shuttle-running test, as previously reported [25].

CAG and GGN repeat polymorphisms

DNA was extracted from saliva samples (200 µl) using High Pure PCR Template Preparation Kits (Roche Applied Science). To determine the length of the CAG and GGN repeats the corresponding regions located on the exon 1 of the AR gene (Genbank

accession no. M27423) were amplified using two pairs of primers whose sequences have been previously reported [26]. One primer from each pair was marked with fluorescent dye (FAM or VIC). Amplification was performed in a 25 µl reaction volume, containing 50 ng of genomic DNA, 200 µM of each deoxynucleotide triphosphate, 1x Fast Start Taq DNA polymerase Buffer (Roche Applied Science, Mannheim, Germany), 1x GC-rich solution buffer (Roche Applied Science) and 1U of Fast Start Taq DNA polymerase (Roche Applied Science). The concentration of each pair of primers was 1.2 and 1.5 µM for the amplification of the CAG and GGN repeats, respectively. PCR conditions were: 30 cycles of 95ºC for 45 sec, 56ºC for 30 sec and 72ºC for 30 sec for CAG amplification; 30 cycles of 95ºC for 1 min, 55ºC for 2 min and 72ºC for 2 min for GGN amplification. Each PCR was initiated with a denaturation step at 95ºC for 5 min and terminated with an extension step at 72ºC for 5 min. The PCR product was diluted 1:100 in distilled water and 1 µl of the dilution was mixed with 10µl of formamide and 0.3 µl of GeneScan 500 LIZ Size Standard (Applied Biosystems, Warrington, UK), denatured at 98ºC for 5 min and cooled on ice. Fragment separation was performed by automated capillary electrophoresis, using an ABI Prism 3100 Genetic Analyzer (Applied Biosystems) and the length was determined with Gene Scan Analysis Software (version 3.7) (Applied Biosystems). Internal standards supplied by the manufacturer were used for quality control. We blindly repeated the genotype analysis in 54 of the samples, and the results were completely coincident. The fragments size was confirmed by sequencing 48 DNA samples harboring different size alleles for both repeats by using the Big Dye Terminator Sequencing Kit (Applied Biosystem) at University of Las Palmas Sequencing Facility with excellent agreement between both procedures. Genotyping was performed specifically for research purposes based on the hypothesis that the aforementioned polymorphisms may influence VO_2 max, lean mass

and muscle strength. The genotype data of the subjects were not previously analyzed for other non-research purposes and as such were not presented a posteriori for the present paper. The researchers in charge of genotyping were totally blinded to the subjects' identities, that is, blood samples were tracked solely with code numbers, and personal identities were only made available to the main study researcher who was not involved in actual genotyping.

Statistical analysis

Descriptive statistics are presented as mean values with standard deviations (SD). The homogeneity of variances was verified using the Levene test. Data normal distribution was checked by the Shapiro-Wilks test. When necessary, the statistical analysis was performed with logarithmically transformed data. Subjects were classified as short CAG (CAG_S), long CAG (CAG_L), short GGN (GGN_S) or long GGN (GGN_L) using the median value that resulted in the most balanced grouping as a cutoff threshold[27, 28]. Therefore, subjects with a CAG $\leq$ 21 were classified as short CAG (CAG_S) and those with a CAG > 21 were classified as long CAG (CAG_L). In the case of GGN, subjects carrying a number of repetitions $\leq$ 23 were classified as short GGN (GGN_S), and those showing a number of repetitions > 23 were considered as long GGN (GGN_L). These median CAG and GGN values are identical to those reported for the population of Gran Canaria[28] and similar to those observed in other Caucasian cohorts[29-31]. Between-group comparisons were performed with a one-way ANOVA with age, weight, height and sexual maturation as covariates. Statistical analysis was performed with SPSS (SPSS Inc., Chicago, IL, USA, v 15.0). Significant differences were assumed at $P < 0.05$.

Results

Information about boys' and girls' body composition, anthropometrics, growth status and fitness is shown in Table 1. In boys, there was a normal distribution of 16 different CAG alleles, ranging from 13 to 33 repeats, and 12 GGN alleles ranging from 14 to 30 repeats. In girls, 11 different CAG alleles were found, ranging from 14 to 25 repeats, whereas the number of GGN alleles was 9, ranging from 13 to 24 repeats.

CAG repeat polymorphism

Boys' and girls' body composition, anthropometrics, growth status and fitness in the CAG_S and CAG_L groups are reported in Table 2. No significant differences in age, height, body mass, segmental lean mass and fat mass were observed between CAG_S and CAG_L groups. The aerobic capacity (VO_2max), mean speed in the 30 m running test, as well as SJ and CMJ jumping height were similar in both groups. However, in boys, the CAG_S group completed the 300m running test 5.5% faster than the CAG_L group ($P<0.05$, Table 2). This difference remained significant even after accounting for age, height, body mass and sexual maturation.

GGN repeat polymorphism

Boys' and girls' body composition, anthropometrics, growth status and fitness in the GGN_S and GGN_L groups are reported in Table 3. No significant differences in age, height, body mass, segmental lean mass and fat mass were observed between GGN_S and GGN_L groups. Mean speed in the 30 m and 300m running test, as well as SJ and CMJ jumping height were similar in both groups and sexes. However, in girls, the GGN_L group showed higher VO_2max values compared to the GGN_S group (48.6 ± 6.0 vs 45.4

± 4.4 respectively $P<0.05$, table 3). This difference remained significant even after accounting for age, height, body mass and sexual maturation.

Discussion

This study shows that pre pubertal boys carrying a CAG_s allele have a 5.5% better performance in the 300m running test, indicating that s CAG_s genotype may be associated with increased anaerobic capacity. In addition, we have also shown that a long GGN genotype is associated with enhanced VO_2max in girls. This is in contrast with the observation of no association between CAG and GGN repeat lengths and either body composition or performance in 282 young men [32], and muscle mass in 677 men [33].

It has been shown that a shorter repetition number of CAG and GGN AR polymorphisms is associated with a higher androgen activity[7, 9]. This could facilitate greater muscular mass acquisition during growth in humans. During growth, the hyphotalamic-pituitary-gonadal axis is relatively silent and the gonads only produce minor amount of androgens. In this environment of low androgen bioavailability, those individuals carrying either CAG or GGN short repetition polymorphisms could benefit from a more efficient androgenic signaling, stimulating greater muscle mass acquisition compared to their longer allele carrying counterparts. In agreement, the present investigation shows that boys carrying a CAG≤21 showed a 5.5% higher velocity in the running speed test (300m) than those carrying a CAG>21. Performance in the 300 m test depends on the muscle mass, VO_2max and on the anaerobic capacity [22]. Since we did not find differences in either muscle mass or VO_2max between the short and long CAG groups, the most likely explanation for the differences in performance is a greater anaerobic capacity associated with the CAG_S phenotype. This agrees well with the greater anaerobic capacity of men compared to women, in part due to their higher muscular glycolytic capacity [34], and increased expression of type II muscle fibers with

increased androgen sensitivity. In fact, some studies suggest that men possess greater proportion of type II fibers in the musculus vastus lateralis than women [35, 36].

Voorhoeve et al. investigated the relationship between the AR CAG repeat polymorphism and longitudinal growth from pre puberty until young adult age [37]. In this study, it was found that height-standard deviation scores were inversely associated with AR CAG repeat length in boys at young, pre pubertal and early pubertal age. This association diminishes in the following years and completely disappears after the age of 16 years. In agreement, we did not see any differences in the length of our CAG_S and CAG_L in our older children. Moreover, no associations were found by Voorhove et al. [37] between CAG length and body composition variables. However, fitness was not assessed (or reported) in this investigation.

The influence of AR GGN repeat polymorphisms on body composition or performance has not been studied in children and data on adults is scarce [12, 32, 38]. So far, studies have reported lack of association between GGN repeat length with both performance [12, 32, 38] and body composition [12, 32, 33, 38]. In girls, we have observed a greater aerobic power (VO_2max) in the GGN_L group. Since this difference was present after accounting for differences in body composition, it seems reasonable to assume that the GGN repeat polymorphism may influence the oxygen transport system and/or utilization system. During whole body exercise VO_2max depends mostly on O_2 delivery and to lower extend on O_2 extraction by the muscles [39]. The association between the GGN repeat length and VO_2max could be due to greater oxygen transport capacity in the GGN_L group, which could explained by enhanced pumping capacity of the heart or increased arterial O_2 content by either increased hemoglobin concentration or more efficient pulmonary gas exchange. In that sense, testosterone undecanoate therapy has been shown to increase more markedly erythropoiesis in hypogonadal men carrying

shorter AR CAG repeats [40]. However, other authors have not observed any association between hematological parameters and AR polymorphisms in children [41]. The heart expresses both androgen and estrogen receptors and is functionally responsive to circulating sex steroids [42]. Although testosterone may facilitate cardiac hypertrophy,[43] no association has been reported so far between the pumping capacity of the heart and androgens. Moreover, the length of the GGN repeat polymorphism is linearly and inversely associated with AR protein content in cell cultures [9]. Due to the sex steroids interaction in the heart a longer GGN_L may translate into a more efficient calcium handling, which may favor a greater pumping capacity of the heart [42]. However, the latter would require specific testing, which is not possible in children, due to the necessity of using catheter to obtain accurate peak cardiac output values during exercise. Finally, another mechanism by which a longer GGN polymorphism could influence VO_2max is by favoring a greater expression of type I fibers. A greater androgenic action is associated with greater expression of type IIa fibers, as shown by the higher cross sectional area proportion of type IIa fibers in men than women [44]. Predominantly type I fiber muscles have higher oxidative capacity compared to type IIa and IIx predominant muscles [45] and such a phenotype would favor a greater O_2 extraction and, hence, VO_2max [46]. In agreement with this explanation, necropsy-based studies have shown in boys a progressive increase in the proportion of type II fibers with maturation [47], i.e., with the increase of testosterone levels.

Conclusions

Our results indicate for the first time that androgen CAG and GGN repeat polymorphisms are associated with exercise capacity in growing children in a sex specific way. In boys, a shorter CAG genotype is associated with increased performance in prolonged sprint, likely mediated by an enhanced anaerobic capacity. In girls, a

longer GGN repeat polymorphism is associated with increased VO_2max, after accounting for body size. Further studies with a mechanistic approach are required to determine why a longer GGN repeat polymorphism may favor a greater VO_2max in girls. This study contributes to the current knowledge about the influence of genetic variability on exercise capacity, highlighting the role of the androgen receptor gene to be taken into consideration as a target gene related to physical performance during growth.

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Table legends

Table 1. Body composition, anthropometrics, physical activity and fitness in boys and girls (mean ± standard deviation). Ht: height; SJJH: jumping height in squat jumps; SJWmax: maximal power in squat jumps; CMJJH: jumping height in countermovement jumps; CMJWmax: maximal power in countermovement jump; T30m and T300m running time in the 30 and 300 m running sprint, respectively.

Table 2. Body composition, anthropometrics, physical activity and fitness in boys and girls with CAGs and CAG_L androgen receptor polymorphisms (mean ± standard deviation). Ht: height; SJJH: jumping height in squat jumps; SJWmax: maximal power in squat jumps; CMJJH: jumping height in countermovement jumps; CMJWmax: maximal power in countermovement jump; T30m and T300m running time in the 30 and 300 m running sprint, respectively. Subjects were grouped as CAG short (CAG_s) if harboring repeat lengths of ≤21 and CAG long (CAG_L) if harboring repeat lengths of >21. *P<0.05 vs CAG_L.

Table 3. Body composition, anthropometrics, physical activity and fitness in boys and girls with GGN_s and GGN_L androgen receptor polymorphisms (mean ± standard deviation). Ht: height; SJJH: jumping height in squat jumps; SJWmax: maximal power in squat jumps; CMJJH: jumping height in countermovement jumps; CMJWmax: maximal power in countermovement jump; T30m and T300m running time in the 30 and 300 m running sprint, respectively. Subjects were grouped as GGN short (GGN_s) if harboring repeat lengths of ≤23 and CAG long (GGN_L) if harboring repeat lengths of >23. *P<0.05 vs GGN_L.

Table 1. Body composition, anthropometrics, physical activity and fitness in boys and girls (mean ± standard deviation).

	boys			n	girls			n
Age	11.5	±	2.6	152	10.1	±	3.2	116
Height (cm)	147.9	±	14.8	152	138.4	±	15.9	116
Body mass (kg)	41.7	±	13.1	152	36.4	±	12.6	116
Percentage of body fat (%)	21.1	±	8.7	152	27.2	±	8.5	116
Lean body mass (kg)	30.6	±	9.9	152	24.4	±	7.8	116
Lean mass arms (kg)	2.9	±	1.1	152	2.1	±	0.7	116
Lean mass legs (kg)	10.1	±	3.8	152	7.5	±	2.8	116
Lean mass extremities (kg)	5.7	±	1.0	152	4.8	±	0.8	116
Lean mass arms/Ht^2 ($kg.m^{-2}$)	1.3	±	0.2	152	1.1	±	0.2	116
Lean mass legs/Ht^2 ($kg.m^{-2}$)	4.5	±	0.8	152	3.8	±	0.6	116
Lean mass extremities/Ht^2 ($kg.m^{-2}$)	5.7	±	1.0	152	4.8	±	0.8	116
Tanner G	2.5	±	1.2	152	2.0	±	1.2	116
Tanner H	2.6	±	1.2	152	2.2	±	1.4	116
Jumping tests								
SJJH (m)	0.2	±	0.1	134	0.2	±	0.1	79
CMJJH (m)	0.2	±	0.1	133	0.2	±	0.1	77
SJWmax(W)	1429.7	±	657.9	133	1308.6	±	559.4	44
CMJWmax(W)	1539.8	±	690.5	132	1473.1	±	586.2	43
Running tests								
T30m (s)	5.5	±	0.5	133	6.4	±	1.1	103
T300m (s)	72.0	±	12.6	114	85.2	±	17.8	99
Maximal aerobic power								
VO_2max ($ml.kg^{-1}.min^{-1}$)	47.7	±	4.7	130	47.2	±	5.6	102

Ht: height; SJJH: jumping height in squat jumps; SJWmax: maximal power in squat jumps; CMJJH: jumping height in countermovement jumps; CMJWmax: maximal power in countermovement jump; T_{30m} and T300m running time in the 30 and 300 m running sprint, respectively.

ble 2. Body composition, anthropometrics, physical activity and fitness in boys and girls with CAGs and CAG_L androgen receptor polymorphisms (mean ± standard deviatio

	CAG_S boys			n	CAG_L boys			n	CAG_S girls			n	CAG_L girls			
e	11.3	±	2.4	90	10.1	±	3.4	51	11.7	±	2.8	62	10.2	±	3.0	
ight (cm)	146.1	±	14.0	90	138.2	±	15.8	51	150.5	±	15.6	62	138.6	±	16.0	
dy mass (kg)	39.9	±	12.0	90	44.2	±	14.4	51	36.3	±	13.3	62	36.5	±	12.2	
rcentage of body fat (%)	20.9	±	8.1	90	27.0	±	8.7	51	21.3	±	9.5	62	27.3	±	8.4	
an body mass (kg)	29.5	±	9.0	90	24.3	±	7.6	51	32.3	±	10.9	62	24.4	±	8.0	
an mass arms (kg)	2.8	±	1.0	90	2.1	±	0.7	51	3.1	±	1.2	62	2.1	±	0.7	
an mass legs (kg)	9.6	±	3.4	90	7.5	±	2.7	51	10.8	±	4.3	62	7.5	±	2.9	
an mass extremities (kg)	5.6	±	0.9	90	4.9	±	0.8	51	5.9	±	1.1	62	4.8	±	0.8	
an mass arms/Ht^2 ($kg.m^{-2}$)	1.3	±	0.2	90	1.1	±	0.2	51	1.3	±	0.2	62	1.1	±	0.2	
an mass legs/Ht^2 ($kg.m^{-2}$)	4.4	±	0.7	90	3.8	±	0.6	51	4.6	±	0.9	62	3.8	±	0.7	
an mass extremties/Ht^2 ($kg.m^{-2}$)	5.6	±	0.9	90	4.9	±	0.8	51	5.9	±	1.1	62	4.8	±	0.8	
nner G	2.5	±	1.1	90	2.0	±	1.2	51	2.5	±	1.2	62	2.0	±	1.3	
nner H	2.6	±	1.2	90	2.2	±	1.3	51	2.6	±	1.2	62	2.2	±	1.4	
mping tests																
SJJH (m)	0.2	±	0.1	77	0.2	±	0.1	28	0.2	±	0.1	57	0.2	±	0.1	
SJWmax (w)	1376.0	±	608.5	77	1326.2	±	542.1	19	1503.5	±	719.4	56	1295.2	±	583.0	
CMJJH (m)	0.2	±	0.1	76	0.2	±	0.1	28	0.2	±	0.1	57	0.2	±	0.1	
CMJWmax (w)	1473.4	±	645.9	76	1515.3	±	615.2	19	1629.8	±	743.3	56	1439.8	±	573.4	
nning tests																
30m (s)	5.6	±	0.5	82	6.4	±	1.1	44	5.5	±	0.5	51	6.4	±	1.2	
300m (s)	70.3*	±	12.3	68	84.6	±	14.1	41	74.4	±	12.8	46	85.7	±	20.1	
ximal aerobic power																
VO_2max ($ml.kg^{-1}.min^{-1}$)	47.6	±	4.4	78	47.2	±	6.2	43	47.8	±	5.2	52	47.1	±	5.2	

Table 3. Body composition, anthropometrics, physical activity and fitness in boys and girls with GGNs and GGN_L androgen receptor polymorphisms (mean ± standard deviation).

	GGN_S boys			n	GGN_L boys			n	GGN_S girls			n	GGN_L girls			n
Age	11.3	±	2.4	93	10.7	±	3.3	50	11.8	±	2.8	59	9.7	±	3.0	66
Height (cm)	147.6	±	14.9	93	142.1	±	16.6	50	148.4	±	14.7	59	135.7	±	14.9	66
Body mass (kg)	41.1	±	12.5	93	39.5	±	13.7	50	42.6	±	14.2	59	34.0	±	11.3	66
Percentage of body fat (%)	21.0	±	8.6	93	28.1	±	8.8	50	21.1	±	8.9	59	26.5	±	8.4	66
Lean body mass (kg)	30.2	±	9.3	93	26.2	±	8.4	50	31.3	±	10.7	59	23.0	±	7.0	66
Lean mass arms (kg)	2.9	±	1.0	93	2.2	±	0.8	50	3.0	±	1.2	59	2.0	±	0.6	66
Lean mass legs (kg)	9.9	±	3.6	93	8.2	±	3.0	50	10.4	±	4.1	59	7.0	±	2.5	66
Lean mass extremities (kg)	5.7	±	0.9	93	5.0	±	8.6	50	5.9	±	1.1	59	4.7	±	0.7	66
Lean mass arms/Ht^2 ($kg.m^{-2}$)	1.3	±	0.2	93	1.1	±	0.2	50	1.3	±	0.3	59	1.1	±	0.2	66
Lean mass legs/Ht^2 ($kg.m^{-2}$)	4.4	±	0.7	93	3.9	±	0.7	50	4.5	±	0.9	59	3.7	±	0.6	66
Lean mass extremties/Ht^2 ($kg.m^{-2}$)	5.7	±	0.9	93	5.0	±	0.8	50	5.9	±	1.1	59	4.7	±	0.7	66
Tanner G	2.5	±	1.2	93	2.3	±	1.3	50	2.6	±	1.2	59	1.8	±	1.2	66
Tanner H	2.5	±	1.2	93	2.5	±	1.4	50	2.7	±	1.2	59	1.9	±	1.3	66
Jumping tests																
SJJH (m)	0.2	±	0.1	83	0.2	±	0.1	44	0.2	±	0.1	51	0.2	±	0.1	35
SJWmax (w)	1380.4	±	602.8	83	1361.0	±	599.7	26	1511.5	±	739.6	50	1232.8	±	502.5	18
CMJJH (m)	0.2	±	0.1	82	0.2	±	0.1	42	0.2	±	0.1	51	0.2	±	0.1	35
CMJWmax (w)	1488.3	±	628.8	82	1468.8	±	591.2	25	1624.2	±	780.6	50	1479.2	±	596.4	18
Running tests																
T30m (s)	5.6	±	0.5	81	6.3	±	1.1	47	5.5	±	0.6	52	6.4	±	1.2	56
T300m (s)	72.1	±	12.8	74	84.9	±	18.3	45	71.8	±	12.4	40	85.6	±	17.5	54
Maximal aerobic power																
VO_2max ($ml.kg^{-1}.min^{-1}$)	47.6	±	4.5	77	45.4	±	4.4	46	45.4*	±	4.4	53	48.6	±	6.0	56

Ht: height; SJJH: jumping height in squat jumps; SJWmax: maximal power in squat jumps; CMJJH: jumping height in countermovement jumps; CMJWmax: maximal power in countermovement jumps; T_{30m} and T300m running time in the 30 and 300 m running sprint, respectively.*P<0.05 vs GGN_L