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The relationship between *ACTN3 R577X* gene polymorphism and physical performance in amateur soccer players and sedentary individuals

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ABSTRACT: The aim of this study was to determine the distribution of *ACTN3 R577X* gene polymorphism in soccer players and sedentary individuals, and to investigate the relationship of this distribution with performance tests. A total of 100 soccer players and 101 sedentary individuals were enrolled in the study. Standing long jump and countermovement jump (with arm swing, without arm swing and repeated) scores were recorded, using a jump meter. Maximum VO_2 levels were measured using a treadmill-connected cardiopulmonary exercise device, Masterscreen CPX. *ACTN3 R577X* polymorphism was evaluated by real-time PCR. *ACTN3 R577X* genotype distribution was found to be similar in soccer players and controls ($p>0.05$). The only statistically significant finding was a shorter countermovement jump with arm swing scores in the RR-genotyped soccer players, compared with their RX genotyped counterparts ($p<0.05$). In the soccer player group, RX-genotyped subjects were observed to have lower respiratory threshold values compared with RR-genotyped subjects ($p<0.05$). No significant correlation was detected between this distribution and performance test results. *ACTN3 R577X* genotype distribution was found to have no effect on sprint and endurance characteristics in amateur soccer players. The *ACTN3 R577X* polymorphism may not be a specific enough genetic marker to determine athletic performance in soccer.

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INTRODUCTION

One of the genes associated with sports performance is the *ACTN3* gene encoding α-actinin-3. α-Actinins are members of the actin binding protein family and the basic structural component of the Z-line in skeletal muscle. The structure with several functional roles provides a platform for the protein-protein interactions of signal and metabolic proteins [1]. There are four types of α-actinin isoforms in mammals, and they assume similar functions in different cells. *ACTN3* expression is mostly limited to type 2 (fast glycolytic) muscle fibres; however, it is also expressed in the brain in small amounts [2]. *ACTN3 R577X* polymorphism is the transformation of the codon comprising the arginine (R) amino acid at position 577 into a stop codon (X) as a result of the C→T transversion occurring at position 1747 in exon 16. If the X allele is homozygous (XX genotype), α-actinin-3 is never synthesized [3].

Approximately one billion people are estimated to have α-actinin-3 deficiency worldwide. Despite this common deficiency, no restriction is observed in the expression of fast glycolytic fibre, because this deficiency is compensated by α-actinin-2 [4]. *ACTN3* genotype may affect the performance of skeletal muscle. Studies conducted in knockout mice by Mac Arthur *et al.* revealed that decreased α-actinin-3 expression activated aerobic pathways in muscle and increased endurance, but reduced muscle strength (grip strength) [5, 6].

Many studies have demonstrated that XX genotype frequency is significantly lower than the control group for sprint/power athletes [7-11]. No XX genotype was encountered in elite female sprint athletes by Yang, in black power athletes by Roth, and in elite sprinters by Niemi [8, 10, 11]. In endurance athletes, genotype distribution was found to be similar to the control group in many studies [8, 9, 12-15]. However, while Yang found that XX genotype frequency was higher in female endurance athletes than the control group, Ahmetov found that XX genotype frequency was higher than the control group for both female and male endurance athletes [11, 16].

The results of these two studies contradict each other. When all of these studies are assessed, one can surmise that XX genotype frequency is low in elite sprint/power athletes; however, the results related to the effect of the *ACTN3* gene on endurance performance are contradictory.

When studies conducted with soccer players are examined, detected *ACTN3* polymorphism distributions are quite different from each other. A clear relationship could not be revealed between the *ACTN3 R577X* genotype and sprint or endurance performance in soccer [17-22].

The aim of this study is to determine *ACTN3 R577X* genotype distribution in soccer players and sedentary individuals, and to investigate the relationship of this distribution with both sprint and endurance performance, which are important parameters in the game of soccer. The study hypothesizes that RR-genotyped soccer players will achieve scores higher than XX-genotyped athletes in countermovement jump and long jump tests, and that XX-genotyped soccer players will achieve higher scores than RR-genotyped soccer players in the VO_2max measurement. The matching control group was chosen to determine whether *ACTN3* genotype distribution is different in the population, independent of physical capacity and training effect.

MATERIALS AND METHODS

Research type and selection of subjects

This is a cross-sectional case-control study. The tests and analyses of all cases were performed in a sports medicine clinic. *ACTN3 R577X* gene polymorphism was evaluated by a private laboratory.

All subjects were selected among Caucasians for the homogeneity of the genetic effect. Explanatory letters were sent to soccer league clubs in the Ege region, and players matching the criteria were included in the study. All players were amateur athletes. The soccer player group consisted of 100 healthy male soccer players aged 18-30 years, involved in mid season soccer training or matches for at least 75 min (mean 103 min), for four or more times per week (mean 5.5 times) within the last three months (mean 5.1 months). Sedentary individuals were selected upon responses to widespread notices in the university campus. The sedentary control group of 101 healthy volunteers had age and morphologic characteristics similar to the soccer player group, and were involved in exercises of less than 60 min, at most once a week within the last three months.

Measurements

Subcutaneous adipose tissue thicknesses of the subjects were measured using a skinfold caliper (Holtain, United Kingdom) applying 10 g of pressure to 1 mm^2 in each expansion with ± 2 mm error. Measurements were made at the right triceps, subscapular, abdominal, suprailiac, femoral and gastrocnemius areas of the subjects. Measurements were repeated three times for each area, and the three measurements were averaged.

Measurement of body weight and body fat ratio by bioelectrical impedance

Measurements were performed with the Tanita, TBF-310-A Body Composition Analyzer & Scale (Tanita Corp, Tokyo, Japan) device.

Standing countermovement jump and standing long jump measurements

Before measurements, all subjects warmed up by pedalling at 50 W in the first 5 min, and at 100 W in the following 5 min, on a cycle ergometer. Then, they were subjected to short-term dynamic and static lower extremity stretching exercises.

Standing countermovement jump

A Newtest Powertimer (Newtest Oy, Finland) was used for the standing countermovement jump. Countermovement jump measurements were performed with an arm swing, without an arm swing and repeated.

Countermovement jump with arm swing: Subjects start standing upright with their feet shoulder-width apart on the test mat, then they jump as high as possible with the knees and hips flexed. The value measured when landing on the mat appears in cm on the device monitor. Measurements are repeated three times, and the best value is recorded.

Countermovement jump without arm swing: Subjects start standing stable upright with hands on the waist and with their feet shoulder-width apart on the test mat, then they jump as high as possible with the knees and hips flexed. This measurement is also repeated three times and the best value is recorded.

Repeated countermovement jump: Subjects jump 20 times without any interruption with a free jump technique. The best and worst five measurements are excluded, and the remaining 10 measurements are averaged.

Standing long jump

The participant slightly squats down with the feet shoulder-width apart and jumps as far forward as possible with the leg push-off supported by the backward and forward movement of the arms. Following the fall, the distance between the heel closest to the starting point and the starting point is measured in cm. Measurements are repeated at least three times and the best value is recorded.

Maximal oxygen consumption (VO_2max)

VO_2max levels were measured through a Masterscreen CPX (CareFusion, California) connected to the treadmill. Before each measurement, the device was calibrated with a 16% oxygen and 5% carbon dioxide gas mixture. Tests were performed at an ambient temperature of 22-24°C and a humidity level of 50-60%. Tests were not performed when an individual had an infectious condition or a body temperature >37°C. Protocol stages were as follows: In sedentary individuals, the Bruce protocol application starts at a speed of 2.7 km/h and a slope of 0%, the slope increases to 10% in 3 min, but the speed

remains stable, and the speed and slope increase gradually every 3 min, after 6 min. In soccer players, the test starts with a slope of 2% and a speed of 8.0 km/h; the slope remains stable during the test, but the speed increases gradually by 1.0 km/h every min.

Genetic analysis

Two tubes of venous blood were obtained for the study. The samples taken were stored at -80°C in EDTA-containing tubes.

DNA isolation

It was performed using the High Pure PCR Template Preparation Kit (Roche, Basel, Switzerland) in blood or serum samples. Following isolation, DNA concentrations were measured by spectrophotometry. It is considered that 30-100 ng of DNA is sufficient for MSI detection per reaction.

Studying ACTN3 R577X gene polymorphism by real-time PCR

Firstly, primers were added with the required amount of water and diluted to 100 μM (i.e. 100 pmol/μl). *ACTN3* was studied by creating interim stocks. The amount of water required for probes was added and diluted to 20 μM (i.e. 20 pmol/μl). For probes, interim stocks of 4 μM were created by taking 4 μl from the main stock of 20 μM, and adding 16 μl of water to it in the same manner for all parameters. The procedure was carried out in the same manner for all parameters. Primer main stock preparation: forward (F) and reverse (R) primers were used. An interim stock of 4 μM was created from primer F (by taking 4 μl from the main stock of 100 μM, and adding 96 μl of water to it) and that of 10 μM was created from primer R (by taking 10 μl from the main stock of 100 μM, and adding 90 μl of water to it). Interim stocks of 4 μM were prepared for the probes as specified above, and all reactions were studied as 15 μl of mix + 5 μl of DNA. The classic titration formula of M1*V1=M2*V2 was used when preparing the interim stocks.

Data analysis

LightCycler 480 software analyzes data by the "tm calling" mode, and whether each case has a tm difference is examined. Samples with a tm difference were determined to be *ACTN3* C-C, T-T and C-T alleles.

Statistical analysis

IBM SPSS software (v20.0) was used in statistical assessment. Descriptive statistics, mean and standard deviations (SD) were calculated for numerical data and frequency tables were created. The t-test was used to compare means of two independent groups, and means of more than two groups were compared using one-way analysis of variance (ANOVA). The level of statistical significance was defined as $p<0.05$.

Ethics

The experiments reported in the manuscript were performed in accordance with the ethical standards of the Helsinki Declaration and the participants signed an informed consent form. The study was approved by the Research Ethics Committee of Ege University.

RESULTS

The study sample consisted of a total of 201 individuals including 101 sedentary males and 100 male soccer players. Physical characteristics of the study and control groups are specified in Table 1. Genotype frequencies were found to comply with the Hardy-Weinberg equilibrium in the total group ($p=0.27$), in addition to the control ($p=0.90$) and soccer player groups ($p=0.15$). No significant difference was detected between the groups in terms of *ACTN3* gene polymorphism (Table 2). When height, BW (body weight), BMI (body mass index), BFR (body fat ratio) and subcutaneous fat thickness measurements of the control and soccer player groups are compared according to *ACTN3 R577X* gene polymorphism distribution, no statistically significant differences were observed in either group ($p>0.05$) (Table 3).

TABLE 1. Age, height and body weight (BW) parameters of soccer player and control groups (Mean ± SD).

Parameter	Soccer Players (n=100)	Controls (n=101)
Age (years)	22.3 ± 3.2	22.0 ± 2.4
Height (cm)	178.0 ± 6.7*	176.0 ± 6.3*
Body weight (kg)	73.1 ± 7.3	74.0 ± 11.0

*p< 0.05.

TABLE 2. *ACTN3 R577X* gene polymorphism distributions of soccer player and control groups.

Parameter	ACTN3 RR		ACTN3 RX		ACTN3 XX		R Allele		X Allele	
Group	N	%	N	%	N	%	N	%	N	%
Control	33	32.7	50	49.5	18	17.8	116	57.4	86	42.6
Soccer Players	29	29.0	56	56.0	15	15.0	114	57.0	86	43.0
Total	62	30.8	106	52.7	33	16.4	230	57.2	172	42.8

TABLE 3. Body composition measurements of soccer players and control groups according to *ACTN3* gene polymorphism distribution (Mean ± SD).

PolyM	Parameter	Soccer Players (n=29)	Controls (n =33)	Total (n=62)
RR	**Height** (cm)	176.6 ± 6.2	175.3 ± 6.0	175.9 ± 6.1
	BW (kg)	72.6 ± 7.9	72.8 ± 8.8	72.7 ± 8.3
	BMI (kg/m^2)	23.2 ± 2.2	23.6 ± 2.9	23.4 ± 2.6
	BFP (%)	13.0 ± 3.7	14.6 ± 4.4	13.8 ± 4.1
		Soccer Players (n=56)	**Controls (n =50)**	**Total (n=106)**
RX	**Height** (cm)	178.4 ± 7.2	176.6 ± 6.3	177.6 ± 7.0
	BW (kg)	72.5 ± 7.4	74.9 ± 12.4	73.6 ± 10.1
	BMI (kg/m^2)	22.7 ± 1.7	24.0 ± 3.6	23.3 ± 2.8
	BFP (%)	12.7 ± 2.9	14.8 ± 5.2	13.7 ± 4.2
		Soccer Players (n=15)	**Controls (n =18)**	**Total (n=33)**
XX	**Height** (cm)	179.0 ± 5.5	175.5 ± 5.6	177.1 ± 5.8
	BW (kg)	76.2 ± 5.5	73.8 ± 13.8	74.9 ± 10.8
	BMI (kg/m^2)	23.7 ± 1.2	23.9 ± 4.4	23.8 ± 3.3
	BFP (%)	13.8 ± 1.8	14.2 ± 5.9	14.0 ± 4.5

PolyM: Polymorphism, BW: Body Weight, BMI: Body Mass Index, BFP: Body Fat Percentage.

Table 4. Cardiorespiratory test results of soccer player and control groups according to *ACTN3* gene polymorphism distribution (Mean ± SD).

PolyM	Parameter	Soccer Players (n=29)	Controls (n=33)	Total (n=62)
RR	**VO_{2max}** (ml/min/kg)	60.5 ± 6.4	42.1 ± 6.7	50.7 ± 11.3
	VT (ml/min/kg)	40.9 ± 10.3*	27.1 ± 5.6	33.5 ± 10.6
	RER	1.02 ± 0.05	1.10 ± 0.10	1.10 ± 0.10
	MHR (/min)	193.9 ± 7.4	199.6 ± 7.2	196.7 ± 7.7
		Soccer Players (n=56)	**Controls (n=50)**	**Total (n=106)**
RX	**VO_{2max}** (ml/min/kg)	57.0 ± 7.9	43.6 ± 6.6	50.7 ± 9.9
	VT (ml/min/kg)	35.8 ± 7.2*	29.3 ± 6.4	32.7 ± 7.5
	RER	1.05 ± 0.05	1.10 ± 0.08	1.10 ± 0.09
	MHR (/min)	190.0 ± 7.9	198.7 ± 6.7	194.1 ± 8.5
		Soccer Players (n=15)	**Controls (n=18)**	**Total (n=33)**
XX	**VO_{2max}** (ml/min/kg)	58.6 ± 3.8	45.7 ± 7.1	51.6 ± 8.7
	VT (ml/min/kg)	39.8 ± 8.0	28.7 ± 7.9	33.8 ± 9.6
	RER	1.04 ± 0.06	1.10 ± 0.10	1.10 ± 0.10
	MHR (/min)	191.9 ± 8.0	196.6 ± 8.4	194.4 ± 8.4

PolyM: Polymorphism; VT: Ventilatory Threshold; MHR: Maximal Heart Rate; RER: Respiratory Exchange Ratio; *: $p<0.05$.

Comparison of cardiorespiratory tests of soccer player and control groups

At the end of the cardiorespiratory tests, statistically significant differences were detected in favour of soccer players ($p<0.01$). When the results of soccer player and control groups were assessed according to *ACTN3 R577X* gene polymorphism distribution, it was detected that in the soccer player group, RX-genotyped athletes have respiratory threshold scores lower than the RR group and the difference was significant ($p<0.05$). No effect of *ACTN3 R577X* gene polymorphism was observed on other cardiorespiratory test results (Table 4).

TABLE 5. Jump test results of soccer player and control groups according to *ACTN3* gene polymorphism distribution (Mean ± SD).

PolyM	Test	Soccer Players (n=29)	Controls (n=33)	Total (n=62)
RR	**CJ, with arm swing** (cm)	44.6 ± 3.7*	41.8 ± 6.0	43.1 ± 5.2
	CJ, without arm swing (cm)	37.5 ± 3.3	35.0 ± 4.6	36.2 ± 4.2
	CJ, repeated (cm)	37.3 ± 3.5	31.0 ± 4.5	33.9 ± 5.1
	SLJ (cm)	227.6 ± 15.4	200.8 ± 18.4	213.3 ± 21.7
		Soccer Players (n=56)	**Controls (n=50)**	**Total (n=106)**
RX	**CJ, with arm swing** (cm)	47.0 ± 4.5*	41.4 ± 6.9	44.4 ± 6.4
	CJ, without arm swing (cm)	39.5 ± 4.2	35.6 ± 5.7	37.7 ± 5.3
	CJ, repeated (cm)	38.0 ± 3.8	30.6 ± 4.9	34.5 ± 5.7
	SLJ (cm)	231.9 ± 15.8	204.0 ± 25.1	218.8 ± 24.9
		Soccer Players (n=15)	**Controls (n=18)**	**Total (n=33)**
XX	**CJ, with arm swing** (cm)	45.0 ± 4.8	43.2 ± 6.6	44.0 ± 5.8
	CJ, without arm swing (cm)	38.0 ± 4.4	36.0 ± 5.6	36.9 ± 5.1
	CJ, repeated (cm)	37.9 ± 5.1	31.5 ± 3.5	34.4 ± 5.3
	SLJ (cm)	229.2 ± 15.8	209.0 ± 30.9	218.1 ± 26.8

PolyM: Polymorphism; CJ: Countermovement Jump, SLJ: Standing Long Jump; *p<0.05.

Comparison of jump tests of soccer player and control groups

In the soccer player group, it was determined that the RR-genotyped athletes had jump with arm swing measurements lower than RX-genotyped athletes, and this difference was statistically significant (p<0.05). No relationship of *ACTN3 R577X* polymorphism was detected with other jump measurements (Table 5).

DISCUSSION

ACTN3 R577X polymorphism distribution varies in different populations. In the studies performed with Caucasians, *ACTN3* genotype distributions were observed to be 29-36% RR, 45-54% RX, and 16-20% XX. The genotype distributions of athlete and control groups in this study were similar to the previous studies [3, 4, 7, 15, 23-25]. In this study consisting of athletes selected among Caucasians, no difference was detected in the soccer player and control groups in terms of *ACTN3* genotype distribution (p>0.05). The reason for this may be the difference in anthropometric and biomechanical factors, level of training as well as the presence of many other genes and environmental factors determining physical performance.

Soccer is a sport requiring both endurance and explosive power such as long runs, jumps, sudden diversions, sprints, etc. In the studies performed with elite soccer players relating to the subject, *ACTN3* polymorphism reflected lower [19] or higher [18, 21, 22] RR rates. RR frequency in soccer players was significantly higher than that of the control (29%) and endurance athlete (26.5%) groups [22]. According to this distribution, the researchers concluded that elite soccer players were prone to sprint in terms of genotype.

The soccer players' genotype distribution in this study included 29% RR genotype, 56% RX genotype and 15% XX genotype. This distribution was different from the soccer player genotype distribution in the four studies mentioned; particularly the RR polymorphism was more rare [18, 19, 21, 22]. The genotype distributions of the control and athlete groups were similar. A reason for this may be the inability to select soccer players from upper leagues.

Jumping is known to play a key role for superior performance in soccer, and it was shown that there was a high correlation between jump height and sprint performance [26]. Pimenta *et al.* found that among 200 elite Brazilian soccer players, RR- and RX-genotyped soccer players had scores higher than XX-genotyped soccer players in squat jump (SJ) and countermovement jump without arm swing tests (RR 38 cm, RX 37 cm, XX 35 cm) (p<0.05) [21]. In our soccer group countermovement jump without arm swing results were 37.5 cm in RR, 39.5 cm in RX and 38.0 cm in XX. However, the same researchers found no relationship between jump and 10-20-30 m sprint tests and genotype distribution in athletes playing in professional, U20 and U17 premier leagues [17]. Similar to the latter study, no correlation was found between jump tests and the ACTN3 genotype. Physiological, anthropometric, training and biomechanical factors might have contributed to this issue, along with the amateur level of soccer players in the study. In young male soccer players from professional minor leagues, RR/RX players have better jump and sprint test results [27]. A relationship between countermovement jump without arm swing test and *ACTN3* genotype was examined in rugby athletes, and no difference was found according to genotype distribution (p=0.80). Contrary to the expectations, mean jump scores of XX-genotyped athletes were higher than the mean of RR and RX genotypes despite there being no statistically significant difference [28].

When different fields are in question, no relationship was detected between *ACTN3* genotype distribution and jump tests in elite male volleyball players and elite male and female basketball players [24, 29]. In Polish athletes from different disciplines (volleyball, swimming, canoe, ice hockey), RR-genotyped athletes jumped significantly higher than XX- genotyped athletes [30]. Only two of these six studies examining the relationship between *ACTN3 R577X* polymorphism and jump tests found a relationship with genotype. One of the hypotheses of the study was that RR-genotyped soccer players would achieve scores higher than XX-genotyped athletes in countermovement jump and long jump tests. However, when the jump tests of soccer player and control groups were assessed according to *ACTN3 R577X* gene polymorphism distribution, it was found that RR-genotyped athletes displayed jumps with arm swing measurement lower than RX-genotyped athletes only in the soccer player group, and this difference was significant ($p<0.05$) (Table 5). Hands-free jumping might be more related to genetic variability, as it requires less technical aptitude. In this respect, explosive power and muscle strength would be expected to be higher in the RR genotype, which was not the case in the hands-free jumping scores. It is not possible to explain sports performance with only a single genetic polymorphism. The reason why RR-genotyped athletes had countermovement jump with arm swing scores lower than those in RX- genotyped athletes in the study may result from different genes associated with performance or other factors.

In a study performed with a knockout mouse (KO) model, Mac Arthur *et al.* found that *ACTN3*-/- mice had increased oxidative enzyme activities compared with *ACTN3*+/+ mice, and the reason for this increase was not the loss of fast fibres, but the changed metabolism of these fibres [6]. Mice were subjected to an exercise capacity test to reveal the effect of this metabolism change in muscle on the endurance performance; the exercise capacities of *ACTN3*-/-mice were found to be 33% higher. Extensor digitorum longus (fast contracting) muscles of knockout mice and natural type mice were isolated, and it was found that KO mice had a longer semi-relaxation time and better fatigue recovery compared with the natural type. These findings demonstrated that fast contracting muscles gained the character of slow contracting muscle fibres in KO mice, and accordingly they ran distances longer than natural type mice did.

One of the hypotheses of the study was that XX-genotyped soccer players would achieve higher VO_2max measurements than RR-genotyped soccer players. Regarding VO_2max scores, no significant differences were observed according to ACTN3 genotype. In the soccer player group, RX-genotyped athletes were determined to have lower respiratory thresholds than the RR group, and this difference was statistically significant ($p< 0.05$). No effect of *ACTN3 R577X* gene polymorphism was observed on other cardiorespiratory test results (Table 4). VO_2max was determined to be 43.5 ± 6.8 ml/min/kg in the control group and 58.2 ± 7.1 ml/min/kg in the soccer player group. In the literature, VO_2max levels were reported to be 50-75 ml/min/kg in adult male soccer players and 50-55 ml/min/kg in goalkeepers [31].

Pimenta *et al.* measured VO_2max values in 200 elite Brazilian soccer players with the Yo-Yo test, and found that XX-genotyped soccer players had a mean VO_2max higher than RR-genotyped soccer players ($p<0.05$) [21]. However, no difference was detected according to *ACTN3* genotype in VO_2max measurements determined by the same researchers with the Yo-Yo test in 138 soccer players playing in professional, U20 and U17 premier leagues in Brazil [17]. When Lucia *et al.* compared oxygen consumption capacities according to *ACTN3 R577X* polymorphism distribution in 50 elite long distance cyclists (VO_2max: 71-75 ml/min/kg) and 52 long distance runners (VO_2max: 70-75 ml/min/kg), no significant difference was detected in either group ($p>0.05$) [12]. Papadimitriou *et al.* found no association between *ACTN3 R577X* polymorphism and endurance running times in Caucasian athletes [32]. While it was shown that muscle metabolism shifted to the oxidative aspect in the XX genotype in the studies with mice, a relationship between increased aerobic capacity and genotype was not revealed in athletes.

Studies assessing the relationship of genotype distribution with performance tests in sedentary individuals are rare. In a study with controls of similar age distribution, VO_2max measured by a method similar to ours yielded similar results, with no relationship with genotype distribution [33].VO_2max levels measured through shuttle run tests in sedentary adolescents were lower than our subjects' levels, again with no relationship with genotype distribution [23]. Countermovement jump scores obtained in three different studies evaluating jumping tests and genotype distribution in sedentary individuals were similar to those obtained in our study, again without any relationship with genotype distribution [24, 28, 29].

CONCLUSIONS

This is the first study in Turkey to examine the relationship between sprint and endurance performance tests, and *ACTN3 R577X* gene polymorphism. No significant difference was detected regarding *ACTN3 R577X* genotype distribution between amateur soccer players and sedentary individuals. *ACTN3 R577X* genotype distribution was found to have no effect on sprint and endurance characteristics in the two groups.

Conflict of interest declaration

We have no conflict of interest.

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