Original Article

Sox15 T/G may be a candidate gene polymorphism for athlete performance

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Abstract

Muscle formation and regeneration are critical factors for athlete endurance performance, and the SOX15 gene is one of the important genes for muscle formation and regeneration. Although there are many studies on the relationship between the SOX15 gene and muscle tissue, there are few studies on the relationship between the SOX15 gene and athlete performance relation are very limited number. This study aimed to analyse the SOX15 T/G (rs4227) polymorphisms of soccer referees, who performed 3000 m and above in the Cooper test, and of the control group. For this purpose, 30 male soccer referees, who performed 3000 m and above in the Cooper test, were selected. For the control group, 30 male participants without a sportsman license were selected. In addition, in this study, ACE I/D (rs4646994) and HGF C/A (rs5745697) polymorphisms were analysed. The results showed a statistically significant difference in SOX15 T/G ($\chi^2 = 10.24$, $p = 0.006$) between the referees and the control group. The SOX15 T allele showed a significantly higher frequency in soccer referees than in the control group. However, our data did not show statistically significant differences in ACE I/D and HGF C/A. Our results suggested that SOX15 T/G polymorphism could be related to the endurance performance of athletes.

Keywords: Athletes, Endurance, Polymorphism, rs4227, Sports Genomics, SOX15

Introduction

Muscles are vital body components for athletes. Therefore, factors that have a role in muscle formation are also important. The SOX15 (Sry-box 15) gene is one of these factors.

SOX15 is a transcription factor and it is known that involved in embryonic growth (Rui, Xu, Jiang, Guo, & Jiang, 2017). SOX15 gene, located in 17p13, is a high mobility group and includes 20 different protein codes. SOX15 activates and represses transcription on cell growth and differentiation (Bernard & Harley, 2010). It is known that the SOX15 gene has been expressed in fetal tissues and mature tissues (Meyer, Wirth, Held, Schempp, & Scherer, 1996; Vujić, Rajić, Goodfellow, & Stevanović, 1998).

It has been shown the high expression of SOX15 in myoblast cells (Béranger, Méjean, Moniot, Berta, & Vandromme, 2000). Similarly, in a detailed analysis, Lee et al. have shown that SOX15 has a critical role in muscle differentiation (Lee et al., 2004). The same group showed that muscle regeneration was lowered after muscle injury in SOX15 silenced animals. Another study showed that skeletal muscle regeneration disrupted SOX15 silenced animals (Meeson et al., 2007).

Savage et al. showed that the amount of Pax3 and Meox1 proteins, transcription factors involved in muscle formation, was decreased in SOX15 silenced P19 cells (Savage, Conley, Blais, & Skerjanc, 2009). Moreover, the same study showed Msx1 and Id1, suppressors on myogenic growth, levels were increased in SOX15 silenced cells. Parallely, Meeson et al. showed that SOX15 silenced C1C12 myoblast cells stopped over in the G0/G1 stage of the cell cycle (Meeson et al., 2007). In a similar study, Yan et al. showed that at least six genes, which related to the cell cycle, were overexpressed with SOX15 overexpression (Yan et al., 2007). Another study has suggested that SOX15 affects MyoD expression and in this way, it might have a relation skeletal muscle regeneration (Ito, 2010).

Although there are a great number of studies about SOX15 gene - muscle relation, the number of studies about the relation of the SOX15 gene and athlete performance are limited. Pruna et al. showed SOX15 T/G polymorphism (rs4227) has a relation with non-contact muscle injuries of footballers. The group has shown that the footballers who have SOX15 T allele have a fewer frequency of non-contact injuries. In the same study, the footballers who have SOX15 G/G genotype have had abnormal skeletal muscle formation and have increased frequency of non-contact injuries (Pruna, Artells, Lundblad, & Maffulli, 2017). However, there isn't any study in the literature about the relation of SOX15 T/G polymorphism with athlete endurance performance.

This study analyzed the SOX15 T/G (rs4227) polymorphisms of soccer referees who could run 3000 m and above in the Cooper test and compared with a control group who without a sportsman license. And this research also analyzed the ACE I/D (rs4646994) and HGF C/A (rs5745697) polymorphisms. The aim of this study is to analyze whether the relation of SOX15 T/G polymorphism with athlete endurance performances.

Material & methods

Subjects

30 male soccer referees (Age: 23.33 ± 2.52 years; height: 178.19 ± 5.23 ; weight: 72.64 ± 7.16) from Ankara participated in this research. For the selection of referees who would participate, the Cooper test had applied to the referees. Referees who performed 3000 m and above were selected to participate in this study. For this study, permission was obtained from the provincial referee board of Ankara. For the control group, 30 male sedentary (Age: 22.47 ± 1.92 years; height: 176.71 ± 7.02 ; weight: 76.81 ± 9.62) participants without a sportsman license were selected from Ankara University Science Faculty with permission of faculty deanery. In this research, all experiments were performed in accordance with the ethical standards of the Helsinki Declaration and that the participants signed an informed consent form. Our study was approved by the Ankara University School of Medicine (Decision No: 05-409-19).

Cooper Test

The participants run as long-distance as they can in 12 minutes. The running distances of participants have recorded after 12 minutes and approximate maxVO_2 value has calculated through Bandyopadhyay's formula (Bandyopadhyay, 2015):

$$\text{maxVO}_2 (\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}) = (22.351 \times \text{distance value in "km"}) - 11.288$$

Three tests had applied to referees in September 2019, January 2020, and March 2020, and referees who could run 3000 m distance and above in all the three tests selected for genotyping. For 3000 m, maxVO_2 value calculated roughly $55.77 \text{ ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$.

Collecting Cells and DNA Isolation

The cells from participants were isolated by using buccal swabs. For collecting buccal cells, the buccal swabs were brushed inside both cheeks of participants one minute along. After brushing, the cells on buccal swabs were collected inside an Eppendorf which contains NaCl (0,9 %). After collecting, cells were taken to the laboratory for analysis.

All DNA analysis, including DNA isolation, of this experiment was done in Intergen Genetics and Rare Diseases Diagnosis Research & Application Center, Ankara, Turkey. For DNA isolation, an automatic system was used that uses a magnetic beads technique. For isolation, Zinexts – Magpurix Kit (ZP02001) was used.

SNPs and Primer Design

In this research, SOX15 T/G, HGF C/A, and ACE I/D SNPs were analyzed. For directly sequencing SOX15 T/G and HGF C/A amplicons after PCR, proper sequences added 5' ends of primers. ACE I/D polymorphisms genotyped directly by agarose-gel electrophoresis. All primers and amplicon lengths have shown in Table 1 (Table 1).

Table 1. Forward (F) and reverse (R) primers for SOX15, HGF, and ACE genotyping.

Gene	Sequence	Length
SOX15_F	ATTGAGTGCTTTCGTAAGCCCTGTAC	173 bp
SOX15_R	TGCCGTCCAGACTCAGTCTTCCAG	
HGF_F	AAGAATGAAAGTCAAGATTGCCAG	210 bp
HGF_R	AACATGAAATATTACTTGCCCACTTGC	
ACE_F	GGACTCTGTAAGCCACTGCTGGA	I: 530 bp
ACE_R	TCCCATGCCATAACAGGTCTTC	

Polymerase Chain Reaction

For amplification of SNP regions with PCR, proper primers designed for next-generation sequencing were used. For every reaction; 15 μl dH₂O, 5 μl 5x buffer solution (Thermo Inc.), 0,5 μl dNTP mix, 1 μl (5 μM) forward primer, 1 μl (5 μM) reverse primer, 0,5 μl PhireII HS DNA Polymerase (Thermo Inc.), 2 μl template DNA (20-50 ng/ μl) were used.

For each sample, samples were indexed by using different primer sets and made suitable for next-generation sequencing. In this stage, the mixed products of the first PCR were used as a template. For next-generation sequencing; 17 μl dH₂O, 5 μl 5x buffer solution (Bioline Inc.), 1 μl (5 μM) forward primer, 1 μl (5 μM) reverse primer, 0,5 μl MyTaq DNA Polymerase (Bioline Inc.), 0,5 μl template DNA was used for each reaction.

Purification and Measurement

The PCR products were purified by using the MN – NucleoFast 96 Kit. The purified samples were measured by Nanodrop – ND1000 spectrophotometer.

Next-Generation Sequencing

Miseq device (Illumina) was used for next-generation sequencing. Because of the indexing PCR performed, the sample preparation kit wasn't used. The purified and measured samples were diluted properly and loaded directly. For sequencing, a v2 300cy kit was used. For analyzing samples, Miseq Reporter Software (Illumina Inc.) and IGV 2.3 (Broad Institute) were used.

Statistical Analysis

For statistical analysis of this study, the IBM SPSS 24.0 was used. For SOX15 T/G, ACE I/D, and HGF C/A SNPs, Hardy-Weinberg equilibrium of distribution alleles between the referees and control group were tested. For statistical analysis of SNP differences between groups, the Chi-Square/Fisher's Exact Test was used. $p < 0.05$ was considered statistically significant.

Results

All numbers for genotypes of SOX15 T/G, ACE I/D, and HGF C/A in referees and control groups were showed in Table 2. A sample gel-electrophoresis view of ACE I/D polymorphism was showed in Figure 1 (Figure 1).

Figure 1. An example image of agarose gel electrophoresis for ACE I/D polymorphism. A. Ladder: 100 bp. 8th well: Positive control sample. 1, and 3. I/I genotypes. 2, and 5. D/D genotypes. 4,6, and 7 I/D genotypes.

There was a statistically significant difference between referees and control groups on SOX15 T/G (Referees: TT: 2; TG: 11; GG: 17, Control Group: TT: 1; TG: 5; GG: 24) polymorphism ($\chi^2 = 10.24$, $p = 0.006$). SOX15 T allele showed significantly higher frequency in soccer referees compared with the control group. However, our data did not show statistically significant differences in ACE I/D and HGF C/A (Table 2).

Table 2. Genotype and allele frequencies of SOX15 T/G, ACE I/D, and HGF C/A polymorphisms.

Gene	Polymorphism	Referees		Control	
SOX15*	T/T	2	(7%)	1	(3%)
	T/G	11	(37%)	5	(17%)
	G/G	17	(56%)	24	(80%)
	T	15	(15%)	7	(12%)
	G	45	(75%)	53	(88%)
ACE	I/I	8	(27%)	6	(20%)
	I/D	9	(30%)	14	(47%)
	D/D	13	(43%)	10	(33%)
	I	25	(42%)	26	(43%)
	D	35	(58%)	34	(57%)
HGF	C/C	1	(3%)	0	(0%)
	C/A	6	(20%)	6	(20%)
	A/A	23	(77%)	24	(80%)
	C	8	(13%)	6	(10%)
	A	52	(87%)	54	(90%)

*($p < 0,05$)

Discussion

In this study, we analyzed SOX15 T/G, HGF C/A, and ACE I/D polymorphisms of referees, who performed 3000 m and above distances in the Cooper endurance test, and the control group. Our results showed that the frequency of the SOX15 T allele was higher in referees than the control group significantly ($p < 0,05$). Although many studies showed the SOX15 gene and muscle regeneration relationship, the researches about SOX15 and athlete performance are very limited. Pruna et al. showed a relation with non-contact muscle injuries and SOX15 T allele in football players. According to the research, football players who have SOX15 T allele had lower injury frequencies, and players who have SOX15 G allele had abnormal muscle formations (Pruna et al., 2017). In parallel, our results showed that SOX15 T allele frequency is higher in referees who performed 3000 m and above than the control group. These results suggested that SOX15 T/G polymorphism could be related to the athlete endurance performance.

Researches showed that the SOX15 gene has a powerful relation with muscle tissues as the higher expression in myoblast cells (Béranger et al., 2000) and has a critical role in muscle tissue differentiation (Lee et al., 2004). Likewise, muscle regeneration was slowed down in SOX15 silenced animals (Meeson et al., 2007), and the levels of transcription factors related to muscle differentiation were decreased in SOX15 silenced cell lines (Savage et al., 2009). According to these results, the SOX15 gene might be a very important factor for muscle tissue generation. The difference of SOX15 T/G polymorphisms between the referees and the control group might be for these reasons. SOX15 T allele might be effective in the athlete endurance performance.

Athletes with the SOX15 T allele may have an acute effect of faster muscle regeneration. It is known those eccentric muscle contractions while endurance performance induces muscle damages (Clarkson & Hubal, 2002). It has shown that elongated or high efforted endurance exercises lead to disturbances in cellular components of muscles (Mancora & Bosio, 2007; Mastaloudis, Traber, Carstensen, & Widrick, 2006; Santos, Bassit, Caperuto, & Costa Rosa, 2004; Saunders, Kane, & Todd, 2004). The acute exercise-induced muscle damages restrict the endurance of the athletes (Nosaka, Chapman, Newton, & Sacco, 2006). It might be considered that the athletes who have the SOX15 T allele might have injury-resistant muscles. The significant difference in this study may be due to injury-resistant muscles of soccer referees. Our results did not show difference in ACE I/D polymorphism between the referees and the control group. Although ACE I allele was shown as an endurance marker in the first periods of sports genomics researches (Alvarez et al., 2000; Montgomery et al., 1998; Nazarov et al., 2001), most recent studies have shown that the ACE I/D polymorphism has no significant relation with endurance performance of athletes (Ahmetov et al., 2009; Ash et al., 2011; Tobina et al., 2010). Furthermore, the most recent review showed that the number of negative results about ACE I/D polymorphism and endurance performance relation was above the positive results (Szalata, Słomski, Balkó, & Balkó, 2019). Exemplary, researchers showed Ethiopian (Ash et al., 2011), Japanese (Tobina et al., 2010), Greek (Papadimitriou, Papadopoulos, Kouvatsi, & Triantaphyllidis, 2009), and Kenyan (Scott et al., 2005) endurance athletes no had a significant difference in ACE I/D polymorphism. Similarly, our results did not show significant difference in ACE I/D polymorphism between the referees and the control group.

HGF C/A polymorphism has also analyzed in this research. Our results did not show significant difference in HGF C/A polymorphism between the referees and the control group. Researches showed that the HGF expression increased after muscle injuries (Tatsumi, Anderson, Nevoret, Halevy, & Allen, 1998) and overexpression of the HGF gene could increase muscle regeneration (Gal-Levi, Leshem, Aoki, Nakamura, & Halevy, 1998; O'Reilly, McKay, Phillips, Tarnopolsky, & Parise, 2008; Tatsumi et al., 1998). Similarly, that was shown the high expression of HGF related to higher handgrip power (Shimizu et al., 2018). Based on these studies, the HGF C/A polymorphism could have been effective on endurance performances of referees who performed 3000 m and above in the Cooper test but the difference with the control group was meaningless.

Conclusions

This is the first study that directly analysis relation SOX15 T/G and HGF C/A polymorphisms with athlete endurance performance. Sports genomics researches generally focused on the difference between elite athletes and sedentary control groups. Differently, our study was based on the Cooper test that an indirect maxVO₂ measurement test. Our results showed that the SOX15 T allele frequency was significantly higher in referees who performed 3000 m and above in the Cooper test than the control group. The physiological effects of SOX15 T/G polymorphism are still unclear. The criticality of the SOX15 gene for muscle tissue formation increases the importance of this gene for athletes. Coaches/practitioners might design different training programs for athletes based on SOX15 T/G polymorphism. On the other hand, our data did not show significant difference in HGF C/A and ACE I/D polymorphisms between the referees and the control group. Considering the increase in negative results, the decision of whether the ACE I allele is an endurance marker needs more researches. Repeating this research with a larger number of participants is necessary to detail and clarify the results.

Conflicts of interest

We know of no conflicts of interest associated with this publication, and there has been no significant financial support for this work that could have influenced its outcome. As Corresponding Author, I confirm that the manuscript has been read and approved for submission by all named authors.

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