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Section II – Exercise Physiology and Sports Medicine

Genotypic and Allelic Frequencies of the ACE I/D Polymorphism in Elite Moroccan Athletes and Controls: A Pilot Study

by

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This study sought to investigate the genotypic and allelic frequencies of ACE I/D polymorphisms in elite Moroccan road cyclists and field hockey players. Forty-three Moroccan elite male athletes (19 cyclists; 24 field hockey players) and 28 healthy non-athletes were recruited for the study. All participants underwent ACE I/D polymorphism genotyping by the polymerase chain reaction (PCR) using genomic DNA from blood samples. The genotypic distribution of the ACE I/D polymorphism was similar in elite athletes (DD: 46.50, ID: 44.20, II: 9.30%) and controls (DD: 42.90, ID: 46.40, II: 10.70%; $X^2 = 0.103$, $p = 0.949$). The allelic distribution was also similar in elite athletes (D allele; 68.60, I allele: 31.40%) and controls (D allele; 66.07, I allele: 33.93%; $X^2 = 0.099$, $p = 0.752$) as well as cyclists (D allele; 63.16, I allele: 36.84%) and field hockey players (D allele; 72.92, I allele: 27.08%; $X^2 = 0.937$, $p = 0.332$). These novel data indicate no significant differences in the genotypic and allelic frequencies of the ACE I/D polymorphism between athletes and controls or between elite road cyclists and field hockey players of Moroccan origin.

Keywords: *genetics; athletic performance; angiotensin-converting enzyme; cyclists; field hockey players*

Introduction

Several factors are known to influence athletic performance including anthropometry, physical, mental and physiological capacities, response to exercise training, nutrition and ergogenic aids, capacity to recover, injury predisposition, and other environmental factors (El Ouali et al., 2023; Nédélec et al., 2015). Genetic and epigenetic factors can also influence athletic performance, as the likelihood of becoming an elite athlete is influenced by several innate factors (El Ouali, et al., 2024; Pickering et al., 2019). Some physical traits and abilities are strongly influenced by genetic factors, while modulation of gene expression by epigenetic factors can improve or reduce an athlete's performance potential (Ehlert et al., 2013; Humińska-Lisowska et al., 2023; Ipekoglu et al., 2023). Several genes and polymorphisms (of > 250 genes) can potentially influence some aspects of athletic performance (Bray et al., 2009), as they may be involved in the innate physiological capacity of athletes (El Ouali, et al., 2024), their response to exercise training (El Ouali, et al., 2023) or their predisposition to injury (Zouhal et al., 2021). Genetic factors have a significant impact on athletic performance, contributing to substantial variations in cardiorespiratory capacity (~40–60%),

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anaerobic performance (~50–90%) and muscular strength (~30–70%) (Bouchard and Hoffman, 2011; Reichert et al., 2025). Among the various genes linked to athletic performance, the gene encoding the angiotensin-converting enzyme (*ACE*) has received particular attention (Ma et al., 2013; Zilberman-Schapira et al., 2012) as the protein that codifies this gene is linked with several processes of blood pressure regulation and skeletal muscle function (Jones et al., 2002).

The ACE converts angiotensin I (Ang I) into angiotensin II (Ang II), thereby increasing vasoconstriction (Chen et al., 2018; Simonyte et al., 2017). Variations in the concentration of the ACE may be linked with muscle growth as Ang II plays a key role in facilitating the hypertrophic reaction of overloaded cardiac muscle (Gordon et al., 2001). The *ACE* gene is situated on the long arm (q) of chromosome 17 (17q23.3) and is composed of 25 introns and 26 exons (Sayed-Tabatabaei et al., 2006). Variations in the *ACE* gene can affect serum concentrations of the ACE (Danser et al., 1995; Rigat et al., 1990) and alter blood pressure regulation and skeletal muscle function both at rest and when exercising. The I/D polymorphism of the genetic variations of *ACE* is well characterized in case-controlled studies (Semenova et al., 2023). The *ACE* I/D polymorphism results in the insertion (I allele) or deletion (D allele) of a 287 base pair segment in intron 16 of the gene (Rigat et al., 1990). This genetic variation results in three different genotypes: II (homozygous for the insertion allele), ID (heterozygous, containing both alleles) or DD (homozygous for the deletion allele). Individuals who have two copies of the I allele have lower levels of the ACE compared with the other genotypes (Rigat et al., 1990), leading to decreased conversion of Ang I to Ang II, and potentially reduced vasoconstriction, resulting in an increased oxygenated blood flow during muscular effort (Jones and Woods, 2003; Sayed-Tabatabaei et al., 2006). On the other hand, people with two copies of the D allele have higher levels of the ACE (Alvarez et al., 2000; Rigat et al., 1990), resulting in pronounced vasoconstriction (Jones and Woods, 2003; Sayed-Tabatabaei et al., 2006) and a potentially higher capacity for stronger muscle contractions. Variations in the *ACE* gene and its phenotypical consequences in serum ACE concentration can be beneficial for athletic performance, as the I allele could increase the oxygenated blood flow during aerobic exercise, while the D allele could favor performance of strength exercise activities. The *ACE* D allele is generally overrepresented in elite athletes of strength sports, while the I allele is overrepresented in high-level endurance athletes (Ahmetov et al., 2016; Silva et al., 2022; Woods, 2009). This distinction is attributed to the potential effect of the *ACE* I/D polymorphism on muscle fiber composition, with higher concentrations of the enzyme being correlated with fast-twitch muscle fibers.

Most investigations on the link between the *ACE* gene and athletic performance have been conducted on Caucasian and Asian athletes. In the Caucasian population, the prevalence of the three genetic variants (II, DD, ID) is around 25%, 25%, and 50%, respectively (Jones et al., 2002). Remarkably, these proportions are similar to those found in the Asian population, particularly in Korea, where the distribution is 23%, 66% and 11% for genotypes II, ID and DD, respectively (Oh, 2007). This suggests a possible similarity in the genetic composition linked to these variants between the two populations. Additionally, in both East Asian and Caucasian athletes, a correlation between elite swimmer status (short, middle and long distance) and the *ACE* I/D polymorphism was found (Wang et al., 2013). However, in African athletes, the expression of the *ACE* I/D polymorphism is unrelated to athletic performance of Ethiopian elite endurance athletes (Ash et al., 2011) and Kenyan endurance athletes (Scott et al., 2005). Furthermore, the *ACE* gene was not an important factor in performance of elite African-American and Jamaican sprinters (Scott et al., 2010), nor in 100-m sprinters of the African origin in a multicohort study (Papadimitriou et al., 2016). Consequently, the conflicting results indicate that the impact of the *ACE* gene on athletic performance may be dependent on the ethnic origin of athletes.

Studies investigating the relationship between the allelic and genotypic distribution of the *ACE* gene and the status of high-level athletes among the Arab population are limited. Therefore, our main objective was to investigate the genotypic and allelic frequencies of the *ACE* gene in elite Moroccan cyclists and field hockey players.

Methods

Ethics Statement

The Research Ethical Committee of the Ibn Tofail University Doctoral Center approved the current study which adhered to established guidelines for biomedical research involving human subjects (approval number: 23/2020, date of approval: 24 September 2020). All participants received a full explanation of the procedures involved, as well as the potential risks and benefits associated with the study. The volunteers were informed of the potential benefits of the study, and they voluntarily signed a written informed consent form to signify their agreement to participate in the experiment. The procedures were in accordance with the consensus statement of the International Federation of Sports Medicine for genetic information (Tanisawa et al., 2020) and all procedures were carried out in accordance with relevant guidelines and regulations of the Declaration of Helsinki.

Study Design and Participants

Forty-three (n = 43) Moroccan elite male athletes participated voluntarily in this study. From this sample, 19 were road cyclists from the Moroccan national cycling team, of which two had qualified for the Olympic Games in Paris 2024, six had qualified for the 2023 World Championships, and two had already won the Tours of Africa. The remaining 24 athletes were players from the Moroccan national field hockey team, who was ranked sixth in Africa and forty-ninth in the world according to the latest continental and international rankings. Additionally, we randomly selected 28 healthy male students to form a control group from a pool of students (n = 69) of the Ibn Tofail University (Kenitra, Morocco). The selection was performed by the assignation of one number to each potential participant and then by selecting 28 random numbers using Excel software. All participants (n = 71) underwent anthropometric measurements under the same conditions and blood samples (4 ml) were taken by nurses working under standard security conditions. Blood tubes were assigned an alphanumeric code to be confidentially stored at −80° C for later use.

Genotyping

The extraction of genomic deoxyribonucleic acid (DNA) was done from leukocyte samples using a kit (MagPurix Blood DNA Extraction Kit) in accordance with the manufacturer's instructions. To determine DNA concentration, a nanodrop assay was carried out using a microspectrophotometer. Genotyping was carried out by the polymerase chain reaction (PCR) using a thermal cycler (Applied Biosystems VERITYTM). The protocol and primers have already been described (Chiu et al., 2019): forward primer: 5′-CTGGAGACCACTCCCATCCTTTCT-3′ and reverse primer: 5′-GATGTGG CCATCACATTCGTCAGAT-3′. The PCR procedure included an initial denaturation step at 98°C for 120 s. This was followed by 30 amplification cycles, each consisting of denaturation at 98°C for 20 s, annealing at 58°C for 60 s and extension at 72°C for 30 s. Finally, the extension step was performed at 72°C for 180 s. The PCR products obtained were electrophoresed on a 1% agarose gel with a 1 kb molecular weight marker for visualization. The PCR method described by Rigat et al. (1992) was used to determine the genotyping *ACE* I/D: polymorphism (I) was characterized by the presence of a fragment of 490 base pairs when amplified using the primers mentioned above. The (D) polymorphism was characterized by the presence of a 190 base pair fragment. The I/I genotype of the *ACE* gene was identified by a single band measuring 490 base pairs, indicating a homozygous insertion polymorphism. Conversely, the I/D genotype was distinguished by the presence of two bands: one at 490 base pairs and another at 190 base pairs. This motif signifies a heterozygous state, with both insertion and deletion polymorphisms. Finally, the D/D genotype was characterized by a single band of 190 base pairs, indicating a homozygous deletion polymorphism (Figure 1). The laboratory provided an identification of the genotype for each alphanumeric code/sample and the researchers were then able to link the information from the laboratory with athletes and controls to avoid the identification of athletes during the genotyping process.

Statistical Analysis

To assess if the samples used in this study met the Hardy-Weinberg Equilibrium (HWE), the

chi-square (X^2) test was used. This assessment involved a comparison between the observed genotype frequencies within each group and the expected genotype frequencies derived from the principles of the HWE (Chen, 2010). The X^2 test was also used to compare allelic and genotypic frequencies between cyclists, field hockey players and the control group. For continuous variables and to assess the normality of the anthropometric data, we used the visual method (QQ-Plot) and a statistical test (D'agostino-Pearson). We used a one-way analysis of variance (ANOVA) for normally distributed data and a Kruskal-Wallis test for non-normally distributed data to compare anthropometric data among groups. If a significant difference was observed among groups, post hoc multiple comparisons were carried out using the Tukey's test for parametric data or the Dunn's test for non-parametric data. These tests were applied to identify statistically significant differences in pairwise comparisons. Consequently, these data were presented in a form of medians and as interquartile ranges (IQR, Q1–Q3) for data that were not normally distributed and means and standard deviations (SD) for normally distributed data. For all analyses the level of significance was set at $p < 0.050$. All analyses were conducted using GraphPad Prism 9.2.0 statistical software (GraphPad Software Inc, San Diego, USA).

Results

The anthropometric characteristics of all athletes and controls are summarized in Table 1. Significant differences in anthropometric data were observed between groups related to age [controls vs. all athletes ($p = 0.005$), controls vs. cyclists ($p < 0.0001$), cyclists vs. field hockey players ($p < 0.001$)], the body mass index (BMI) [controls vs. all athletes ($p < 0.001$), controls vs. cyclists ($p = 0.003$), controls vs. field hockey players ($p = 0.004$)] and body weight [controls vs. all athletes ($p = 0.04$)].

Genotype success was 100% and the distribution of *ACE* gene genotypes showed no deviation from the HWE in any of the groups. The genotypic and allelic frequencies of the *ACE* gene in all participants are presented in Table 2. There were no differences in *ACE* I/D genotypes when comparing cyclists, field hockey players and the control group as shown in Table 3, where there was a similar distribution in the overall sample ($X^2 = 1.792$, $p = 0.938$), controls vs. all athletes ($X^2 = 0.103$, $p = 0.949$), controls vs. cyclists ($X^2 = 0.277$, $p = 0.871$), controls vs. field hockey players ($X^2 = 0.864$, $p = 0.649$) and cyclists vs. field hockey players ($X^2 = 1.715$, $p = 0.424$). Furthermore, no significant difference was found between the D and I when comparing cyclists, field hockey players and the control group, as summarized in Table 4, with similarities in the overall ($X^2 = 1.053$, $p = 0.789$), controls vs. all athletes ($X^2 = 0.099$, $p = 0.752$), controls vs. cyclists ($X^2 = 0.084$, $p = 0.771$), controls vs. field hockey players ($X^2 = 0.568$, $p = 0.450$) and cyclists vs. field hockey players ($X^2 = 0.937$, $p = 0.332$) in I and D alleles distribution.

Table 1. Anthropometric data of elite Moroccan cyclists, field hockey players and control groups.

	Cyclists	Field hockey players	All athletes	Controls	*p* value	Controls vs. All athletes	Controls vs. Cyclists	Controls vs. Field hockey players	Cyclists vs. Field hockey players
Age (yr)	23.58 ± 3.76	18.5 (18–20.75)	20 (18–23)	18.71 ± 0.76	<0.0001	0.005	<0.0001	>0.999	<0.001
Weight (kg)	64.37 ± 4.90	64.25 ± 7.22	64.30 ± 6.23	72 (62–80.50)	0.03	0.04	0.13	0.16	>0.999
Height (m)	1.77 ± 0.05	1.76 ± 0.05	1.77 ± 0.05	1.76 ± 0.08	0.78	0.96	0.80	>0.999	0.81
BMI (kg/m²)	20.50 ± 1.90	20.75 ± 2.00	20.64 ± 1.93	23.04 ± 3.43	<0.001	<0.001	0.003	0.004	0.98

Values are presented as means and SD (normally distributed) or medians; IQR (not normally distributed).
BMI: body mass index

Table 2. The distribution of the *ACE* I/D polymorphism in elite Moroccan cyclists, field hockey players and the control group.

Groups		Cyclists	Field hockey players	All athletes	Controls
Size		19	24	43	28
Genotype distribution n (%)	DD	8 (42.10%)	12 (50.00%)	20 (46.50%)	12 (42.90%)
	ID	8 (42.10%)	11 (45.80%)	19 (44.20%)	13 (46.40%)
	II	3 (15.80%)	1 (4.20%)	4 (9.30%)	3 (10.70%)
Allele distribution n (%)	D	24 (63.16%)	35 (72.92%)	59 (68.60%)	37 (66.07%)
	I	14 (36.84%)	13 (27.08%)	27 (31.40%)	19 (33.93%)
Dominant	p (D)	0.632	0.729	0.686	0.661
Recessive	q (I)	0.368	0.271	0.314	0.339
HWE-*p* value		0.917	0.734	0.986	0.982

HWE: Hardy Weinberg equilibrium

Table 3. Comparison of the *ACE* I/D polymorphism genotypes (DD, ID and II) among elite Moroccan cyclists, field hockey players and the control group.

Groups	**X^2**	**df**	***p* value**
Overall	1.792	6	0.938
Controls vs. All athletes	0.103	2	0.949
Controls vs. Cyclists	0.277	2	0.871
Controls vs. Field hockey players	0.864	2	0.649
Cyclists vs. Field hockey players	1.715	2	0.424

df: degrees of freedom

Table 4. Comparison of the *ACE* I/D polymorphism alleles (I and D) among elite Moroccan cyclists, field hockey players and the control group.

Groups	X^2	df	*p* value
Overall	1.053	3	0.789
Controls vs. All athletes	0.099	1	0.752
Controls vs. Cyclists	0.084	1	0.771
Controls vs. Field hockey players	0.568	1	0.450
Individual vs. Field hockey players	0.937	1	0.332

df: degrees of freedom

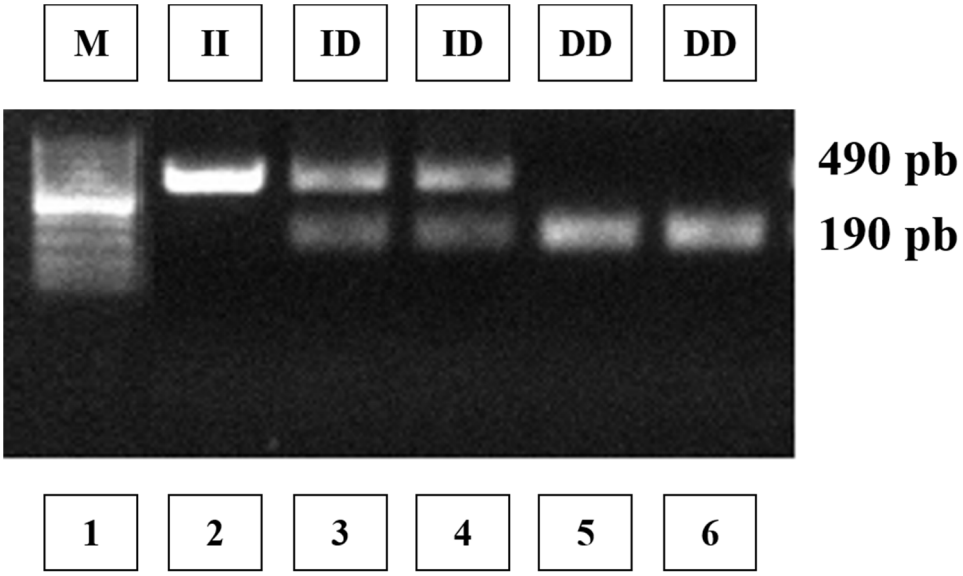

Figure 1. *ACE* I/D polymorphism electrophoresis (agarose gel images). Lane 1: Molecular weight marker with 100 base pair fragments (M). Lane 2: homozygous insertion (II, 490pb). Lane 3: heterozygous insertion/ deletion (ID, 190, 490 pb). Lane 4: heterozygous insertion/ deletion (ID, 190, 490 pb). Lane 5: homozygous deletion (DD, 190pb). Lane 6: homozygous deletion (DD, 190pb).

Discussion

The results of our study show no significant difference for the genotypic and allelic distribution of the *ACE* I/D polymorphism among road cyclists, field hockey players and the control group. However, the genotypic and allelic frequencies of the *ACE* I/D polymorphism showed a high percentage of DD and ID genotypes and dominance of the D allele in cyclists (DD: 42.10, ID: 42.10 and D allele: 63.16), field hockey players (DD: 50.00, ID: 45.80 and D allele: 72.92) and the control group (DD: 42.90, ID: 46.40 and D: 66.07). In addition, we also showed remarkably low frequencies of the II genotype and the I allele in both athletes and controls. Collectively, our findings indicate that the frequency of *ACE* I/D genotypes in Moroccan elite athletes followed a DD > ID > II hierarchy irrespective of the sports discipline. Consequently, the lack of significance in the genotype distribution of the *ACE* I/D polymorphism in Moroccan athletes and non-athletic subjects, suggests that *ACE* genotypes had a non-significant effect on the likelihood of becoming a high-level athlete in these sports.

To the best of the authors knowledge, the current study is the first to examine the distribution of the *ACE* I/D polymorphism in elite Moroccan road cyclists and field hockey players. In particular, studies examining the *ACE* I/D polymorphism, specifically in cyclists and field hockey players, are limited, reducing the comparability of results. Consequently, our discussion focused on research on individual and team sport disciplines, which share similar metabolic demands characterized by aerobic dominance, as is the case in cycling and field hockey. However, the energy demands of cyclists are mainly covered by the aerobic metabolism, taking into account the intensity of the effort and the distance traveled (Abbiss et al., 2013; Van Erp and Sanders, 2021). Additionally, road cycling is also characterized by the repetition of short

periods of high intensity (overtaking and finishing) which require the activation of anaerobic metabolism (Abbiss et al., 2013). Nevertheless, high power demand (>500 W) was observed during sprints in the last 90 s of the race in international level cyclists (van Erp et al., 2021). On the other hand, in field hockey, players primarily use their aerobic energy system to cope with the extended duration and long distances covered during the game (Kusnanik et al., 2017). Still, the dynamic nature of the sport introduces sprints and bursts of action, resulting in the activation of anaerobic metabolism to meet the rapid and intense energy demands associated with these moments of the match (Boyle et al., 1994; Kusnanik et al., 2017). Interestingly, field hockey players perform anaerobic-based (sprints, change of directions and accelerations/decelerations) actions when close to the hockey ball, what makes this type of metabolism basic for success.

Consistently with our results, previous studies have shown no significant differences in the distribution of the *ACE* gene between elite Kenyan endurance athletes and controls (Scott et al., 2005) and in Ethiopian populations (Ash et al., 2011). Interestingly, the frequency of the II genotype in studies of African athletes was low (i.e., < 20%), and could explain the limited variations of the *ACE* I/D genotypes in athletes of African ancestry. Collectively, these results suggest that the *ACE* gene is a poor determinant of becoming an elite athlete (endurance and power sports) in athletes of African ancestry. On the other hand, in the group of Korean athletes from different sport disciplines (judo, soccer, baseball, volleyball, gymnastics, ice hockey, basketball, and long-distance running), there were no significant differences between control groups and athletes (Oh, 2007). Additionally, in Brazilian soccer players, a similar distribution of the *ACE* I/D polymorphism was demonstrated in athletes and controls (Coelho et al., 2016). The same results were found among soccer players of Australian origin (Jacob et al., 2022). Furthermore, no correlation was found between the I/D genotype and soccer Iranian elite status and better aerobic performance (Falahati and Arazi, 2019).

Conversely, it has been shown that the *ACE* II genotype is more frequent in cyclists than in controls (Konopka et al., 2022). Moreover, an association between the D allele of the *ACE* gene and endurance fitness has been shown in elite Japanese runners (Tobina et al., 2010), elite Japanese wrestlers (Kikuchi et al., 2012) and in elite East Asian swimmers (Wang et al., 2013). A high frequency of the I allele was observed in Indian Army triathletes in comparison with a control group (Shenoy et al., 2010). These results strongly suggest an association between the *ACE* I/D polymorphism in Asian elite athletes and endurance athletic performance. Furthermore, a correlation between the I allele of the *ACE* gene and endurance fitness was observed in rowers from Poland (Cieszczyk et al., 2009). It was reported that VO_2 peak was higher in ice hockey players with the genotype II than with the genotype DD (Doğan et al., 2022). Moreover, a correlation between II genotypes and improved performance in anaerobic and aerobic tests in soccer players was recently reported (Coelho et al., 2022).

Generally, the association between the *ACE* DD genotype and success in strength and speed disciplines, such as short-distance swimming, the high jump, track sprints and the long jump, has been highlighted in several case-controlled studies (Cieszczyk et al., 2010; Grenda et al., 2014; Papadimitriou et al., 2016). Additionally, athletes with the *ACE* DD polymorphism often perform better during exercises engaging greater muscle mass (Charbonneau et al., 2008), have greater strength (Williams et al., 2005), and a larger proportion of fast-twitch muscle fibers (Zhang et al., 2003). Collectively, these attributes can improve athletic performance in activities recurring strength and power, supporting the overrepresentation of DD athletes in strength/power sports (Semenova et al., 2023). On the other hand, individuals with the *ACE* II genotype tend to perform better in sports associated with aerobic energy provision, such as middle- and long-distance running, race walking, hockey and rowing (Cieszczyk et al., 2009; Gronek et al., 2013; Oh, 2007). However, some investigations indicate that the *ACE* genotype may not be a defining genetic variation in all sports disciplines (Orysiak et al., 2013). For example, the genotype distribution and allele frequency of the *ACE* gene was unrelated to performance in elite rugby players (Pasqualetti et al., 2022), with no differences found in the distribution of the *ACE* I/D polymorphism between team rugby players and

non-athletic controls (Heffernan et al., 2016).

In our investigation, despite a high prevalence of ID and DD genotypes in elite road cyclists and field hockey players compared to controls, this frequency was not significantly different in the two groups. In contrast to our results, professional football players were found to have a higher prevalence of the ID and DD genotypes of the ACE gene (Gineviciene et al., 2014). Similarly, American football players also showed increased frequencies of these genotypes (Santoro et al., 2019). Likewise, other studies also demonstrate a correlation between the *ACE* DD genotype and power and strength in American football (Liu et al., 1998; Woods et al., 2001). These findings suggest a possible association between the ID and DD genotypes as well as the D allele of the *ACE* I/D polymorphism and team sports athletes.

Finally, previous research suggests a link between the *ACE* gene and the status of elite athletes in different sport disciplines, mostly in samples of elite Caucasian and Asian athletes (Wang et al., 2013). Our results indicate no correlation between the *ACE* I/D polymorphism and the elite performance level of Moroccan cyclists and field hockey players. These contradictory results could be attributed to geographical variations likely to influence the genetic distribution among athletes and the potential contribution of this polymorphism to performance.

Limitations of the Study

The results of our pilot study on the distribution of genotypes and alleles of the *ACE* gene in road cyclists and field hockey players in African and Arab populations are based on a relatively small sample of Moroccan elite athletes. For this reason, the generalizability of these results to athletes of other African and Arab ethnicities may be uncertain. Additionally, although the specific requirements of road cycling and field hockey include clear differences in exercise intensity, duration, and technical demands, both sports are characterized by high demands of the aerobic system (Abbiss et al., 2013; Kusnanik et al., 2017). Therefore, the common need of high aerobic fitness to reach excellence in road cycling and field hockey may have contributed to the lack of differences in the distribution of *ACE* genotypes between these sports. For this reason, further investigations studying the influence of the *ACE* genotype depending on the sports discipline may consider the use of sports with more definite differences in terms of energy demands, such as the comparison of endurance athletes (e.g., marathoners) with sprinters (e.g., specialists in 100 m). Last, we measured only anthropometric characteristics and no other athletic performance variables were obtained in this study. Future studies should also consider other phenotypic characteristics such as VO_{2max}, sprint performance or muscle mass so as to better associate *ACE* genotypes with sport-specific phenotypes with either aerobic or strength/power status.

Conclusions

Our study showed no significant differences in the genotypic and allelic frequencies of the *ACE* I/D polymorphism among elite Moroccan road cyclists, field hockey players and control non-athletic individuals. These results indicate that the *ACE* gene is not associated with the probability of achieving elite status in these disciplines, at least in a sample of Moroccan athletes.

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