Sports Science

Can a genetic profile be related to performance in young talent track and field athletes? A pilot study

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Abstract - Aim: This study analyzed the influences of ACE and ACTN3 gene variants in sprinters, jumpers, and endurance young athletes of track and field. **Methods:** 36 school-level competitors of both sex (15 girls and 21 boys; aged 16.4 ± 1.2 years; training experience 4 ± 1.2 years) practitioners of different sport disciplines (i.e., sprint, jump, and endurance athletes) participated in the study. The deoxyribonucleic acid (DNA) was extracted from peripheral blood using a standard protocol. Anthropometric measurements, 30 m sprint, squat jump (SJ), and maximal oxygen uptake (VO_{2max}) tests were measured. **Results:** Genotype distribution of the ACE and ACTN3 genes did not differ between groups. In ACE DD and ACTN3 RX genotypes, the SJ test was bigger in sprinters and jumpers than in the endurance runners. In contrast, when analyzing the ACE ID genotype, sprinters had higher SJ than endurance athletes. Moreover, in the ACE DD genotype, the sprinters and jumpers' athletes had lower time in 30 m tests compared to endurance runners. However, the ACE ID and ACTN3 RX genotypes was greater aerobic fitness in endurance runners than in jumpers' athletes. **Conclusion:** Although the genetic profile is not a unique factor for determining athletic performance, the ACE DD and ACTN3 RX genotypes seem to favor athletic performance in power and sprint versus endurance sports. Thus, this study evidenced that assessing genetic variants could be used as an auxiliary way to predict a favorable profile for the identification of young talents of track and field.

Keywords: ACE, ACTN3, sport, talent.

Introduction

Genetic variants have an important influence over determinants of athletic performance, such as endurance, strength, power, neuromuscular coordination, size, and composition of muscle fiber[1]. Furthermore, recent research has shown considerable evidence of a significant association between genetic and optimal environmental conditions, it seems to exert an important role in certain types of sport phenotypes[2]. The complex interactions concerning athletic potential are related to the synergy of several physiological and psychological factors, combined with external factors including nutritional status, altitude, training, and socioeconomic factors[3].

Different genetic markers have been explored to identify important polymorphisms and genetic variants with the potential to predict successful elite sport[4,5]. The most promising gene in that regard is ACTN3, which has commonly been associated with strength, speed, power, and endurance performance[6,7]. Studies reported that ACTN3 is a gene that encodes for α-actinin-3, present only in type-II muscle fibers. A common genetic variation in the ACTN3 gene results in a cytosine-to-thymine replacement transforms the arginine base (R) to a premature stop codon (X) at amino-acid 577, whose carrier of the RR homozygotes and the R allele may be related to muscle size and strength, faster race times and a higher proportion of type II muscle fibers[8]. Another important gene for athletic performance is the ACE, it regulates blood pressure, and therefore has an essential role in cardio-respiratory efficiency. The ACE can also influence skeletal muscle function and growth factors involved in overload-induced muscle hypertrophy[9-13]. The most studied polymorphism of this gene is an insertion-deletion polymorphism (I/D) that has been associated with

endurance sports performance (allele I) and strength/power (allele D)[14].

Nevertheless, little is known about talent identification and genotype-phenotype relationships to predict future sporting success based on young athletes. Current findings contradict existing potential relationships between the prevalence of ACE DD homozygotes, and consequently a better performance of young soccer players in sprint test[15]. Whereas studies suggest there is no association between the ACE and ACTN3 genes, individually or in combination, for muscle strength/power in Polish-trained athletes of different disciplines. This finding suggests that both epigenetic and environmental factors act as a key regulator of athletic performance[16]. On the other hand, it is also important to emphasize that RR, RX, and DD genotypes benefit athletes in strength and speed tasks, while ACE II homozygotes provide bigger fitness in endurance athletes, although there are environmental interactions that may influence sport performance[17].

A recent review showed that both ACE and ACTN3 genes can predict elite athletic capability to take into account multifactorial factors, as athletic training[18]. Therefore, if there are possibilities to trace a favorable genetic profile combined with an ideal training environment, it can be used to predict future athletic potential. Moreover, talent detection programs using genetic have been an integral part of the process of building elite athletes. However, the set of variables that can predict a sporting talent and its subsequent success, is still unclear for many sports organizations[19].

In this way, we hypothesized that there may be specific genetic variants that could be associated with better results for sprinters, jumpers, and endurance athletes. Thus, the purpose of this pilot study was based on the need to assess the feasibility of the ACE and ACTN3 gene variants on muscle power, speed, and endurance tasks; particularly in young talent track and field athletes.

Methods

Participants and experimental design

The sample consisted of 36 track and field athletes of both sexes (15 girls, 21 boys; aged 16.4 ± 1.2 years; training experience 4 ± 1.2 years) engagement in the Olympic talent program. All participants were chosen as the best school-level athletes in their sports discipline by state sports federations. All athletes won their sport discipline medals in the 2016 school championships. Written consent was obtained from each subject and his/her responsible. The study was complied with the Declaration of Helsinki and was approved by the national ethics committee (CONEP 5231).

Different sport disciplines enabled us to divide the participants into three athletic groups (sprint, jump, and endurance runners). The sprint group comprised 11 athletes (4 girls, 7 boys; aged 16.1 ± 1.4 years; height 171 ± 7.8 cm; weight 59.9 ± 9.24 kg; fat mass 8.2 ± 3.94%; training experience 3.9 ± 1.28 years) from the following sport disciplines: sprints 100-400 m (n = 6) and 100-400 m hurdles (n = 5). The jumping group comprised 14 athletes (8 girls, 6 boys; aged 16.7 ± 1.4 years; height 171 ± 7.3 cm; weight 59.63 ± 6.6 kg; fat mass 11.16 ± 4.56%; training experience 4.5 ± 1.11 years) from the following sport disciplines: pole vault (n = 3), long jump (n = 5), high jump (n = 3), and triple jump (n = 3). The endurance group comprised 11 athletes (3 girls, 8 boys; aged 16.1 ± 0.87 years; height 169 ± 8.9 cm; weight 58.16 ± 6.15 kg; fat mass 8.48 ± 6.99%; experience 3.66 ± 1.41 years) from the following sport disciplines: middle-distance 800-1500 m (n = 3), long-distance 3000-5000 m (n = 5) and 2000 m steeplechase (n = 3). The experimental design is shown in Figure 1.

Blood collection and genotype assessment

Blood samples were collected from the antecubital vein into 4 mL tubes with ethylenediamine tetraacetic acid (EDTA). Peripheral blood mononuclear cells (PBMCs) were separated from whole blood by density gradient centrifugation with a Histopaque®-1077 solution (Sigma-Aldrich, St. Louis, USA), as previously described[20]. Then, the DNA was isolated from PBMCs pellets according to the phenol-chloroform method.

The I/D polymorphism of ACE was determined as previously described[21]. The D and I alleles were identified using polymerase chain reaction (PCR) amplification of the respective fragments from intron 16 of the ACE gene and size fractionation and visualization by electrophoresis. The primers for intron 16 were as follows: hace 3s 5' TGGGACCACAGCGCCCGCCACTAC 3' e hace 3 as 5'

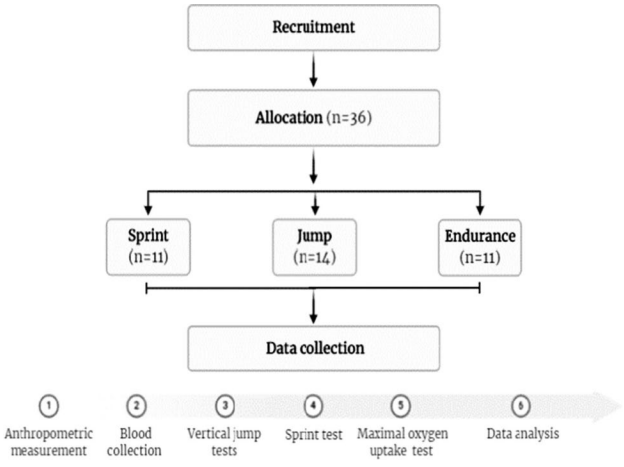

Figure 1 - Illustration of the experimental design.

TCGCCAGCCCTCCCATGCCCATAA 3'. The PCR was performed in a T100™ Thermal Cycler (BioRad) consisting of initial denaturation at 94 °C for 5 min; 35 cycles of 94 °C for 30 s, 56 °C for 45 s, 72 °C for 2 min and final extension at 72 °C for 7 min. The PCR product was analyzed by electrophoresis 1% using Quantum ST4 (Biosystems). This method yields 318 and 597 bp fragments for D and I alleles, respectively.

Because the D allele in heterozygous samples is preferentially amplified[22], PCR amplifications were also performed with an I-specific primer pair (5′ TGGGACCACAGCGCCCGCCACTAC 3' and 5' TCGCCAGCCCTCCCATGCCCATAA 3') as previously described[21], with identical PCR conditions except for an annealing temperature of 67ºC. The reaction yields a 335 bp amplicon only in the presence of an I allele, and no product in samples homozygous for DD.

The R577X polymorphism of ACTN3 was typed using a TaqMan SNP Genotyping Assay (Assay ID: rs1815739; Applied Biosystems, Foster City, CA, USA), and the reaction was performed in a CFX96 Touch™ Real-Time PCR Detection System (BioRad). The genotyping mixture (total of 10 µL) contained 10 µl of TaqMan Universal Master Mix, 1 µl of assay mix, genomic DNA (100 ng/µL), and 7 µL of ultra-pure water for each reaction. The PCR was performed by initial denaturation at 95 °C for 5 min; 40 cycles at 94 °C for 10 s, 60 °C for 15 s; and a final extension at 72 °C for 10 s. The results were analyzed using CFX Manager software (BioRad).

Phenotype assessment

Anthropometric measurement

Height (cm) was evaluated by a standardized protocol. Weight (kg), fat mass (%), and fat-free mass (%) were assessed by an air displacement plethysmograph (BOD POD Gold Standard Body Composition Tracking System).

Vertical jump test

SJ tests were performed using a platform Jumptest® model (Hidrofit Ltda., Brazil) as reported previously[23]. At the starting position, subjects kept both hands on the hips and reached 90° of knee flexion angle, and then, the subjects performed a vertical jump. The test was performed three times (each separated by a 1 min rest period) and the best score was retained.

Sprint test

Subjects performed the 30 m sprint test using a photoelectric cell (Multi Sprint™, Hidrofit®) as previously described[24]. The sprint test involves running a maximum sprint over 30 m, with the time recorded. The 30 m tests were performed two times (each separated by a 5 min rest period) and the best time was registered.

VO_{2max} test

The maximal incremental test was assessed on the treadmill (Inbramed 10.500 ATL, Porto Alegre, Brazil) using a Cosmed K4b^2 portable gas analysis system. Participants started exercising at a treadmill speed of 7 km.h^{-1} and an incline of 1% gradient for 3 min. After, the workload was subsequently increased by 1 km.h^{-1} each minute until exhaustion as previously described[25].

Statistical analysis

The Shapiro-Wilk test was used to confirm the normality of quantitative variables. With normal data distribution, a one-way analysis of variance (ANOVA), followed by Tukey's Test for post-hoc comparison. A χ^2 test was used to compare genotype frequencies between athletes from different disciplines. Significance was set at $p < 0.05$, and continuous data were expressed as the mean and standard deviation of the mean (SD), while categorical data were expressed as a percentage. The Eta-Squared (η^2) was estimated to reveal the effect sizes for a between-groups ANOVA and classified as small ($\eta^2 = 0.01$), medium ($\eta^2 = 0.059$), and large ($\eta^2 = 0.138$) effects. Statistical analysis was performed using GraphPad (version 8.0 for Macintosh OSX, GraphPad Software, San Diego, CA).

Results

Our data showed only three subjects with XX polymorphism, however, but did not find with II polymorphism. Moreover, the sample size presented within groups with XX polymorphism was small; n = 1 in each group (i.e., sprint, jump, and endurance runners). For these reasons, we have decided that it is not possible to report.

There was no difference in anthropometric measurement and training experience among the athletes of sprint, jump, and endurance. The results of the genotypes distribution did not differ between groups for ACTN3 ($\chi^2_{(4)} = 4.489$; $p = 0.334$) or ACE ($\chi^2_{(4)} = 0.257$; $p = 0.880$) (see Table 1).

Figure 2 illustrates the effects of ACE and ACTN3 gene variants on SJ test. In DD genotype, analysis showed

Table 1 - Distribution of ACTN3 and ACE genes in each group.

		Group			Overall
		Sprint	Jump	Endurance	
ACE	DD	4 (11.1%)	4 (11.1%)	3 (8.3%)	11 (30.6%)
	DI	7 (19.4%)	10 (27.8%)	8 (22.2%)	25 (69.4%)
	Overall	11 (30.6%)	14 (38.9%)	11 (30.6%)	36 (100.0%)
ACTN3	RR	7 (19.4%)	4 (11.1%)	3 (8.3%)	14 (38.9%)
	RX	3 (8.3%)	9 (25.0%)	7 (19.4%)	19 (52.8%)
	Overall	10 (30.6%)	13 (38.9%)	10 (30.6%)	33 (100.0%)

Different letters indicate significant differences between groups (one-way test (ANOVA) followed by Tukey's test, $p < 0.05$).

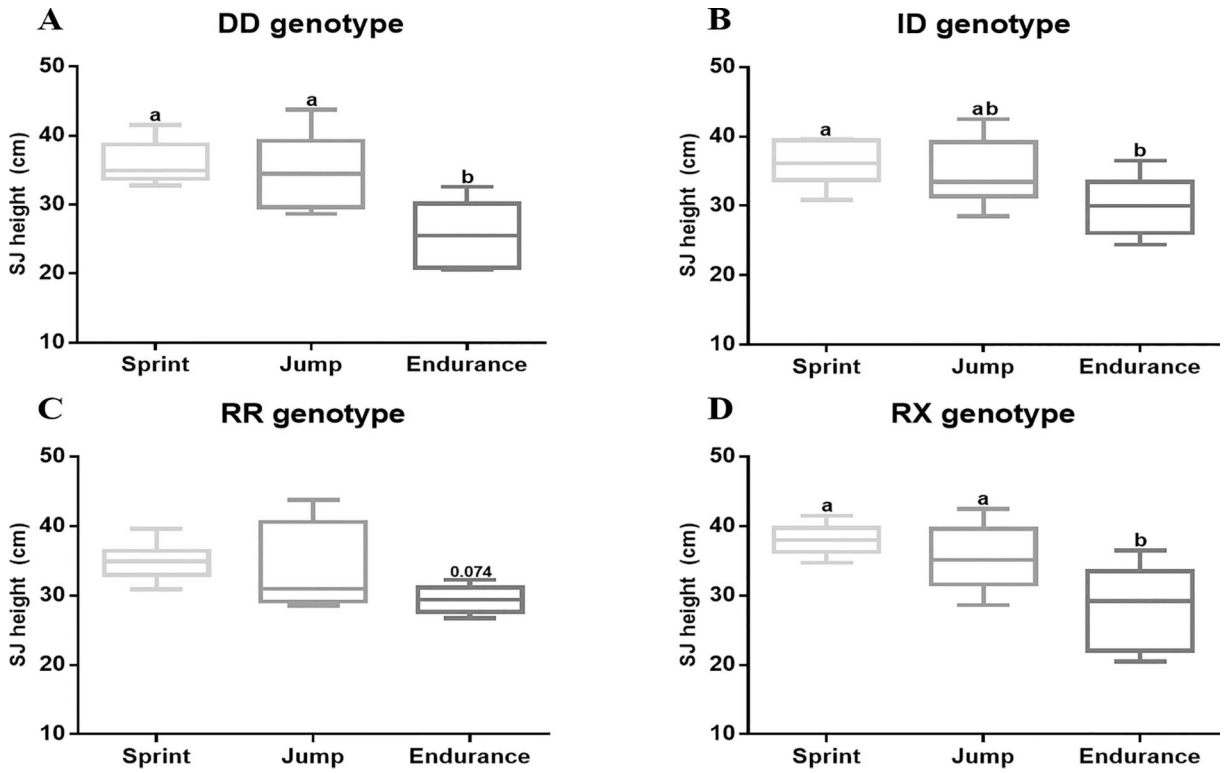

Figure 2 - Effect of ACE and ACTN3 gene variants on SJ in young talents. The DD genotype (a), ID genotype (b), RR genotype (c) and RX genotype (d). Different letters indicate significant differences between groups (one-way test (ANOVA) followed by Tukey's test, $p < 0.05$).

a higher SJ performance in the sprinters and jumpers when compared to endurance athletes ($F_{(2,12)} = 6.928$; $p = 0.01$; $\eta^2 = .53$) (Figure 2a), while in ID genotype was showed increase SJ in sprinters when compared to endurance ($F_{(2,21)} = 4.913$; $p = 0.017$; $\eta^2 = .31$) (Figure 2b). Moreover, in ACTN3 RX genotype, lower performance of the SJ was observed in the endurance when compared to the other groups ($F_{(2,20)} = 7.821$; $p = 0.003$; $\eta^2 = .43$) (Figure 2d). However, there were no differences in RR genotype ($F_{(2.14)} = 3.137$; $p = 0.074$; $\eta^2 = .30$) between groups (Figure 2c).

Figure 3 illustrates the effects of ACE and ACTN3 gene variants on the 30 m sprint test. In the DD genotype, sprinters and jumpers had lower time in 30 m when compared to endurance ($F_{(2,12)} = 8.886$; $p = 0.0043$; $\eta^2 = .59$) (Figure 3a), while the ID (Figure 3b), RR (Figure 3c), and RX (Figure 3d) genotypes did not shown significant difference.

Figure 4 shows the effects of ACE and ACTN3 gene variants on VO_{2max}. The endurance group with ID ($F_{(2,21)} = 8.925$; $p = 0.0016$; $\eta^2 = .45$) (Figure 4b) and RX ($F_{(2,18)} = 5.501$; $p = 0.013$; $\eta^2 = .37$) (Figure 4d) genotypes show higher aerobic fitness when compared to jumpers athletes. However, no changes in VO_{2max} were observed among the groups with DD (Figure 4a) and RR (Figure 4c) genotypes.

Discussion

Our main results showed that both ACE and ACTN3 gene variants seem to exert potential effects on the athletic performance of jumpers, sprinters, and endurance athletes, specifically in young talents of track and field. The DD homozygotes and RX heterozygotes, particularly at the sprinters and jumpers had better SJ scores than in endurance running. Indeed, the D allele has been associated with a greater percentage of fibers type IIb/x (i.e., glycolytic/fast-twitch type)[26], while R allele generates a functional α-actinin-3 that is almost exclusively expressed in fast glycolytic fibers (IIx), which appears to have an advantage for speed and power tasks[27,28]. A higher number of RX heterozygotes expressing the α-actinin-3 protein was observed at sprinters compared to endurance athletes[6]. Consistent with our hypothesis, the DD and RX variants showed better results for sprinters and jumpers in the achievement of power/strength tasks compared with endurance runners, as previously reported[17,18].

Although our study has an apparent limitation of the sample size, our findings are relevant since the presence of DD homozygotes showed favors SJ performance in sprinters and jumpers compared to endurance, while ID heterozygotes increase SJ scores only sprinters, but not to jumpers' athletes. The presence of the DD genotype may

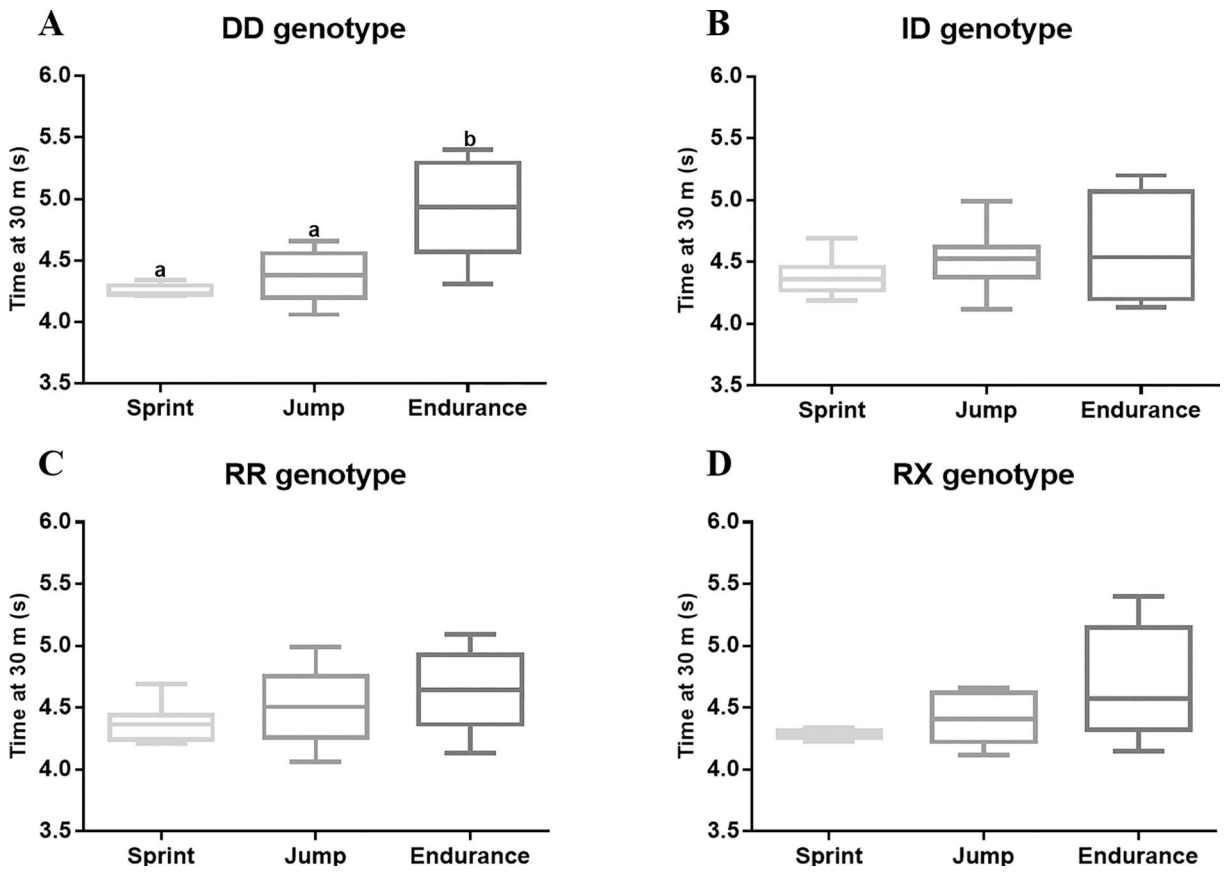

Figure 3 - Effect of ACE and ACTN3 gene variants on 30 m sprint in young talents. The DD genotype (a), ID genotype (b), RR genotype (c), and RX genotype (d). Different letters indicate significant differences between groups (one-way test (ANOVA) followed by Tukey's test, $p < 0.05$).

be likely for the optimal generation of strength in jumpers. Loturco et al. (2015)[29] showed that sprinters, jumpers, and throwers performed higher jumps than endurance athletes; therefore, differences in jump height reflect muscle fiber composition, neuromechanical properties, and training-specific adaptations across these athletes.

The SJ height was unchanged remain with RR homozygotes among sprinters, jumpers, and endurance runners. It is known that the performance of sprint/power athletes was associated with an elevated proportion of the R allele[27] and may be mainly related to the α-actinin-3, the most-highly protein specialized of the α-actinins family (in mammalian)[28]. However, studies showed that the RR genotype was more prevalent among sprinters/jumpers and weightlifters than the controls, it is considered essential for elite sprinting and jumping performance[30]. Thus, the RR genotype may be more related to specialized athletes (elite-level) due to training-related effects, but rather should not carry advantage for the non-specialized young population.

Besides, sprint performance of sprinters and jumpers with DD homozygotes showed a faster 30 m sprint time when compared to the endurance athletes. Sprinters are expected to have a metabolic advantage to carry out repeated contractions under a high metabolic flux. In this way, the D allele was found to demonstrate a higher percentage of muscle fiber type II[26], characterized by a possible advantageous effect in power sports, including sprinters with higher D-allele frequency in running ≤ 200 m[31]. Like in the current study, previous research did not report this finding involving the jumpers' group. On the other hand, there was no possibility to verify a change in sprint performance between groups with ID, RR, and RX genotypes. It was shown that the ID heterozygotes did not seem to favor the athletes during 30 m sprint tests[32], since the I allele in homozygosis or heterozygosis has shown a higher percentage of type I fibers/slow-twitch type fibers[26]. Despite jumpers' and sprinters' occurring naturally multi-joint movements in humans, differences in the angular velocities at the hip or knee joints showed distinct results for the same genotype (ID, RX, and RR)[27,32]. During sprint events, the rate of force development is influenced by multiple factors, such as muscle fiber types, synchronization of motor units, and tendon stiffness, which is a critical performance determinant in sports disciplines[33]. Thus, the use of tests with isokinetic (single-joint), jumps, and sprints (multi-joint) can better reveal the genotype-phenotype relationships[32].

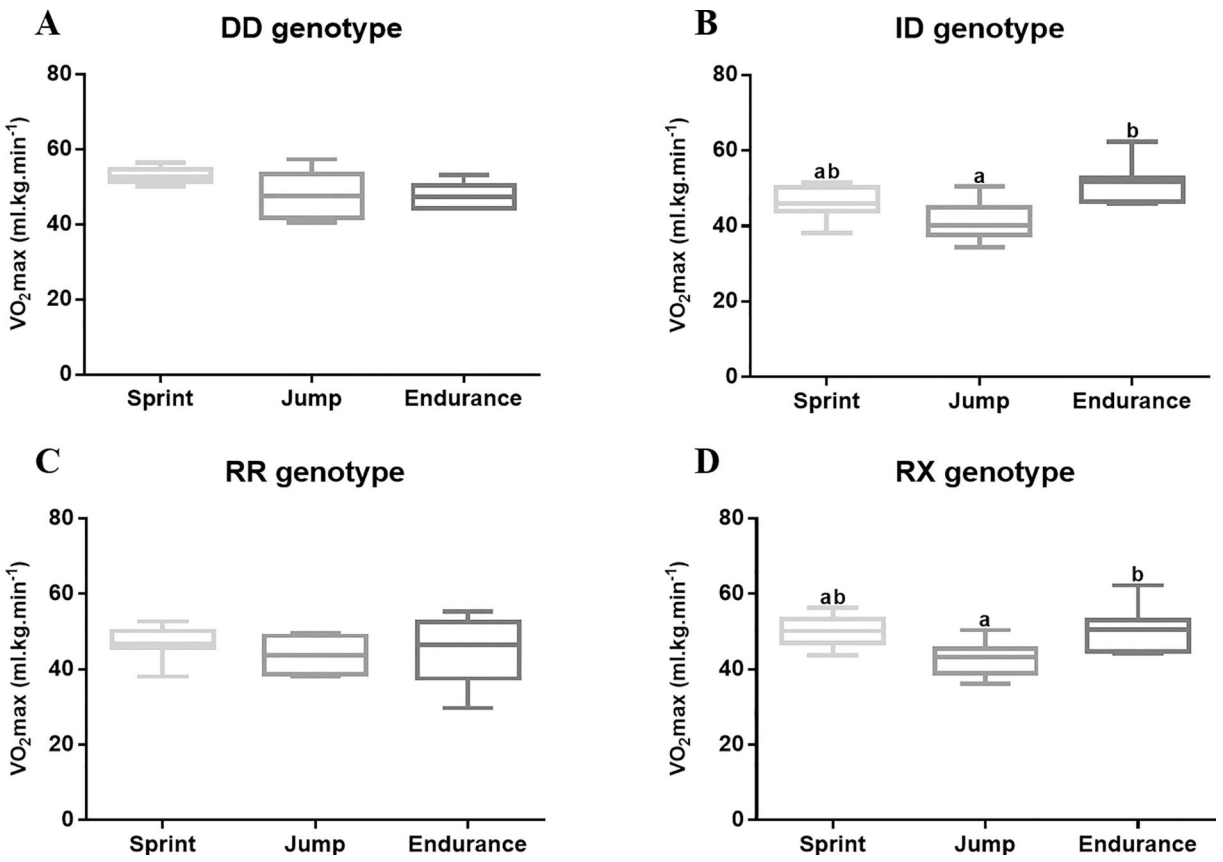

Figure 4 - Effect of ACE and ACTN3 gene variants on VO_{2max} in young talents. The DD genotype (a), ID genotype (b), RR genotype (c) and RX genotype (d). Different letters indicate significant differences between groups (one-way test (ANOVA) followed by Tukey's test, $p < 0.05$).

Both X and I alleles are related to endurance performance[31,34]. There has also evidence that an increased percentage of slow-twitch type I fibers are related to these alleles[26,35]. Particularly, the performance of elite distance runners has been associated with a linear trend of increasing I allele frequency by distance ran[31]. Findings showed in other cohorts an excess of the D allele in the short distance athletes with an excess of the I allele in the middle-distance athletes[36]. In our study, the presence of I (ID) and X (RX) alleles amongst the sprinters and endurance runners were associated with a better level of aerobic fitness (VO_{2max}). This data may be linked to an individual profile suitable to excel in both mixed (aerobic and anaerobic) and predominantly anaerobic sport disciplines. Higher aerobic fitness is an important requirement, but not determinant since VO_{2max} may be influenced by genetic and environmental variables with marked inter-individual response to training[37]. Studies have shown no difference between I and X alleles, and their respective genotypes with VO_{2max}[35,38], emphasizing our finding of ID and RX genotypes to VO_{2max}.

Conclusion

In summary, our pilot study concludes that both ACE DD and ACTN3 RX genotypes may benefit young talents in different sport disciplines that require sprint and power, while ACE ID and ACTN3 RX genotypes benefit endurance runners. Here, a priori, the D allele seems to be more important in the sports initiation, and it seems to present a definition of the sports phenotype. Whereas R allele seems to be a more "refined" or "specialized" gene, closely linked to the quality of the movement and that can be influenced by external factors, such as training.

Acknowledgments

The authors thank CAPES and CNPQ for scholarships.

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Manuscript received on March 4, 2021
Manuscript accepted on December 9, 2021