African Journal of Biochemistry Research Vol. 5(7), pp. 197-205, July 2011
Available online at http://www.academicjournals.org/AJBR
ISSN 1996-0778 ©2011 Academic Journals

Full Length Research Paper

Angiotensin-converting enzyme genotypes relationship with blood pressure, C-reactive protein and selected physical tests in Zulu South African cricketers

Trayana Djarova[1]*, Abigail Ramakoaba[1], Albert Basson[1], Sam Mugandani[2], Jeanne Grace[2], Jacque Cloete[3] and Gregory Watson[4]

[1]Department of Biochemistry and Microbiology, University of Zululand, KwaDlangezwa 3886, South Africa.
[2]Department of Biokinetics and Sport Science, University of Zululand, KwaDlangezwa 3886, South Africa.
[3]Department of Mathematical Sciences, University of Zululand, KwaDlangezwa 3886, South Africa.
[4]School of Biochemistry, Genetics and Microbiology, University of KwaZulu Natal, Scottsville 3209, South Africa.

Accepted 27 May, 2011

Zulu cricketers (n=14) and students (n=17) as controls were genotyped (blood spots) for angiotensin converting enzyme (ACE), gene by PCR amplification followed by agarose gel electrophoresis. Systolic and diastolic blood pressure (SBP and DBP) and grip strength (kg), knee extension and flexion (Nm/kg) were measured, systolic tension time (STT) and metabolic rates (MR) were calculated. After ANOVA, the association between these parameters and I/D gene polymorphism was probed using *Chi*^{2} maximum likelihood test and Fisher's exact test. ACE genotyping for the whole group displayed a complete absence of II genotype, 67.7% DD and 32.3% ID genotypes. The frequency of D allele was 83.8% and I allele 16.2%. In cricketers DD and ID genotypes were 50% each compared to controls-83% DD and 17% ID. No differences in grip strength and quadriceps/hamstring muscle strength between the groups were observed, but for the whole cohort 86% D allele frequency was associated with higher (greater than 43.3 kg) grip strength ($p<0.037$). In cricketers CRP (less than 3.0 mmol/l) was associated with 79% D allele frequency. SBP and DBP were significantly lower by 3.2 and 4.25 mmHg, whereas increased values of STT by 5.5%, and MR by 10.3% were found. Although, the number of participants in this study is small, it is concluded that in cricketers no over presentation of DD or ID genotypes was observed indicating a more balanced display of power and endurance required for the game.

Key words: Angiotensin converting enzyme (ACE) genotype, polymorphism, blood pressure, body mass index (BMI), lean body mass (LBM), fat mass, hand grip, quadriceps and hamstring muscle strength, Zulu cricketers.

INTRODUCTION

Researches have shown that genes play an important role in athletes' performance in various sports (Woods et al., 2002; Wolfarth et al., 2000; Williams and Folland, 2008; Bray et al., 2008; Ruiz et al., 2010). To date improvements in athletes' performance have been based mainly on the manipulation of physiological, physical, nutritional and psychological factors which have been referred to as nurture or environmental constrains (MacArthur and North, 2004; Davids and Baker, 2007). The general view is that there is an interactive influence of genetic and environmental factors on human physical performance. Therefore numerous studies have focused on the genetic make-up of the athletes and associations with fitness phenotypes, physical/physiological tests and molecular basis of adaptation to training (Rankinen et al., 2000; Calo and Vona, 2008; Lucia et al., 2010).

Angiotensin converting enzyme (ACE) polymorphism is the most investigated genetic variation linked to athletic status, physical performance, cardiovascular and muscle function, and trainability in athletes (Alvarez et al., 2000; Scanavini et al., 2002; Collins et al., 2004; Amir et al., 2007; Wang et al., 2008; Min et al., 2009; Ruiz et al.,

*Corresponding author. E-mail: tdjarova@pan.uzulu.ac.za or drdjarova@yahoo.co.uk. Tel: +2735 902 6100 or + 27826192989.

2010).

The ACE gene is located on the long arm of chromosome 17q23.3. It is made up of 26 exons and 25 introns, stretching over 21 kb. The polymorphism analyzed here consists of the presence (insertion, I allele) or absence (deletion, D allele) of a 287 bp Alu repeat sequence, resulting in three genotypes (homozygote DD and II and heterozygote ID) (Rigat et al., 1990; Wang et al., 2008; Wagner et al., 2006; Sipahi et al., 2006). Observations have been reported that individuals carrying ACE I allele are overrepresented in elite endurance athletes (Alvarez et al., 2000; Nazarov et al., 2001; Scanavini et al., 2002) and have been associated with higher percentage of slow twitch type I muscle fibres (Zhang et al., 2003), and have shown an enhanced response to training (Wang et al. 2008). However while the research results are not widely consistent (Scot et al., 2005; Amir et al., 2007) the general findings support the hypothesis that the I allele is associated with better performance in endurance events, while the D allele is associated with success in power events (Folland et al., 2000; Pescatello et al., 2006; Lucia et al., 2010)

The ACE gene is implicated in the rennin angiotensin aldosterone system (RAS). The local RAS is active in a variety of tissues, including lung, kidney, heart, vascular smooth muscle cells and skeletal muscle (Pieruzzi et al., 1995; Jones and Woods, 2003). Angiotensin I-converting enzyme is a key enzyme in the generation of angiotensin (AT)-II from vasoinactive angiotensin (AT)-I. Angiotensin II promotes peripheral vasoconstriction. It also stimulates aldosterone and vasopressin secretion which leads to increased blood volume and increased arterial blood pressure (Sipahi et al., 2006; Calo and Vona, 2008). Individuals who are homozygous for the D allele have been shown to have higher angiotensin-converting enzyme activity in serum and tissue than in those with the I allele (Rigat et al., 1990) but it is not known by what mechanism lower circulating levels of the enzyme could improve performance. ACE protein is also responsible for the degradation of bradykinin, respiratory drive and tissue oxygenation (Ostrander et al., 2009).

Studies involving the association of ACE gene or other muscle and metabolism related genes (example ACTN3, AMPD1, ADRB2, TNF) with blood biochemical parameters and markers of inflammation and exercise-induced oxidative stress have been rare and not studied enough (Lakka et al., 2006; Payne et al., 2007; Bray et al., 2008; Wang et al., 2008; Andonov et al., 2008; Tsianos et al., 2009; Milander et al., 2009). The association of ACTN3 and TNF gene polymorphism with C-reactive protein, uric acid and lactate in cricketers was reported (Djarova et al., 2011). Considering the role of ACE protein in the regulation of some inflammatory reactions and skeletal muscle efficiency (Woods et al., 2000), it is important to explore further the possible association of ACE polymorphism with the above mentioned markers in the same group of cricketers.

Modern cricketers are now exposed to greater physical and physiological demands. Heart rate could reach 190 beats/min and the predominant contribution from the oxygen-independent glycolysis to lactate can contribute to 60% of the total energy in multiple activities of short duration of less than 40 s which are typical for the cricket game (Noakes and Durandt, 2000).

Martens (2004) has identified the following as the estimated energy and muscular/cardiovascular fitness demands of cricket: low to moderate aerobic capacity, moderate anaerobic capacity, moderate strength and flexibility, low to moderate endurance and moderate to high speed. Fast bowling has been linked with a mesomorphic somatotype, greater percentage of type II muscle fibres and a superior phosphagenic and glycolytic metabolic pathways together with eccentric muscle strength. Speed of the ball at release was seen to determine success in bowling (Stuelcken et al., 2007). Hand grip strength was also adjudicated to be an acceptable indicator for good performance in cricket (Koley and Yadar, 2009). A shorter stature and isokinetic knee and shoulder strength were seen to be contributory to the success of batsman (Noakes and Durant, 2000; Nunes and Coetzee, 2007).

The aim of the study is to explore ACE I/D polymorphism, blood pressure and association with C-reactive protein and selected physical tests in Zulu South African cricketers.

MATERIALS AND METHODS

Experimental subjects

The participants of this study were 31 Zulu South African males (14 cricketers age 22.85±0.65 from the University of Zululand cricket team and 17 students age 22.64±0.66 as controls). All experimental subjects were volunteers and a written consent was obtained prior to the study. Experimental protocols were approved by the Ethic Committee of the Research Board of University of Zululand. The participants of the control group reported leisure physical activities once or twice weekly. Cricket players participated in regular 2 h training sessions 5-6 times weekly and played club matches in the Uthumgulu District, KZN over the weekend and inter-universities matches during the season.

Measurements of body mass index (BMI), fat percentage (Fat %), lean body mass (LBM) and fat mass (FM) were taken according to the procedures of the American College of Sports Medicine (Thompson et al., 2000). The evaluation testing procedure as suggested by Ashton and Myers, (2004) was used for the measurement of grip strength. The IsoKnee α was applied to determine the relative strength of the quadriceps (knee extension) and hamstring (knee flexion) muscles as suggested by Coetsee (1995). The speed of rotation was set at 60° for the measurement of the peak muscular strength. A warm-up routine of two to three sets of 6 (six) repetitions interspaced by 30 s rest followed by 3 (three) maximum. The subject was allowed to recover for a few minutes. Data was recorded with the subject performing maximal knee extension and knee flexion for the duration of 10 s.

The estimates of daily energy requirements and metabolic rate (MR) were done using Cunningham equation (Thompson and Manore, 1996). The equation estimates metabolic rate at rest which

Table 1. ACE genotype and allele frequency (%) in cricket players and controls.

Group	Genotype frequency in % and number in brackets)					Allele frequency in %	
	DD		ID		II	D	I
Cricket players (n=14)	50.0	(7)	50.0	(7)	Null	75.0 (10.5)	25.0 (8.5) [a]
Controls (n=17)	82.4	(14)	16.7	(3)	Null	91.2 (15.5)	8.8 (1.5) [a]
Total (31)	67.7	(21)	32.3	(10)	Null	83.8 (26)	16.2 (5.0)[a]

[a]p= 0.004 Fisher's test - two tailed based on %.

is multiplied by an activity factor (within range 1.2 to 1.9) to establish mean daily energy requirements. This estimation has been shown to be the best energy requirement prediction equation for metabolic rate in athletic population (Watson et al., 2005).

All participants were advised not to change their dietary habits and to refrain from physical exercise 24 h before blood sampling. Blood samples were collected at rest from the antecubital vein into vacutainers and analysed in the accredited Lancet laboratory at Bay Hospital, Richards Bay according to the South African standards of good laboratory practice. The Dimension Xpanda (Siemens, Germany) equipment was used for the determination of C-reactive protein (CRP, range 0-8 mg/L), uric acid (UA, range 0.26-0.45 mmol/L and lactate (LA, range 0.63-2.4 mmol/L).

Genotyping

Blood spots were collected on FTA® Classic cards according to the manufacturer's instructions (Whatman International, UK). Samples were prepared by punching 1.2 discs from the cards and washing with FTA® purification reagent and TE (10 mM Tris-HCl, 0.1 mM EDTA, pH 8.0) according to the manufacturer's instructions. PCR was then performed directly from the dried disc. The detection of the insertion (I) and deletion (D) alleles of the *ACE* gene was performed by a modified method of Alvarez and Coto (1998). The primer sequences were ACE F (forward): 5´-CTGGAGACCACTCCCATCCTTTCT -3´ and ACE R (reverse): 5´-GATGTGGCCATCACATTCGTCAGAT -3´. PCR reactions were performed using the SensiMix™ dT kit according to the manufacturer's instructions (Quantace, UK). The final reaction mixtures contained 1× SensiMix (with a final Mg^{2+} concentration of 3 mM) and 200 nM of each of each primer. 20 µl of the PCR mix was added to a single dried disc in a thin-walled 200 µl PCR tube. All amplifications were performed in a Rotor-Gene 6000 (Corbett Research, Australia) using the following conditions: activation step 95°C for 10 min. followed by 40 cycles of 95°C for 10 s, 60°C for 20 s 72°C for 20 s. 1 µl 6× loading buffer was added to 5 µl of each PCR reaction which was then loaded and analysed in a 2% (w/v) agarose 1× TBE gel. All genotypes were determined in duplicate.

Statistical analysis

The Student's *t*-test was used to analyze the statistical difference in the blood biomarkers and physical characteristics between the cricket players and the control group. The results are presented as mean ± SEM. Statistical significance was accepted at $p<0.05$. Statistical analysis for the genotype associations was done using GenStat Discovery Edition 3. The distribution of some variables was skewed; hence these variables were transformed for the Analysis of Variance (Unbalanced design). For the association tests, CRP levels were categorized as less than 3 mg/L (low) and less than 3 mg/l (high) according to Pearson et al. (2003). Other variables were categorized according to their median (M). After ANOVA the association was examined using Chi^2 maximum likelihood test and Fisher's exact test.

RESULTS

ACE genotyping and allele frequencies are shown in Table 1. ACE genotyping showed a complete absence of II genotype (Figure 1). For the whole group 67.7% DD and 32.3% ID genotypes were observed (Figure 2) In cricketers, DD and ID genotypes were 50% each compared to controls – 83% DD and 17% ID (Figure 2). The total frequency of 83.8% D allele for the cohort was significantly higher ($p=0.004$) compared to 16.2% I allele. It was also found that in cricketers 25% ACE I allele frequency was higher ($p=0.004$) than 8.8% in controls, and 75% D allele frequency was lower ($p=0.004$) compared to 91.2%, respectively (Figure 3 and Table 1).

The blood pressure results are shown in Table 2. In cricket players SBP was lower by 3.2 mmHg ($p<0.05$) and DBP by 4.25 mmHg ($p<0.001$), where the values of STT were increased by 5.5% ($p<0.05$) compared to controls. No differences in heart rate and pulse pressure and no associations between blood pressure and allele frequencies were noted.

Cricketers have shown higher ($p<0.05$) basic metabolic rate and increased values ($p<0.001$) of metabolic rate by 10.3% and energy requirements by 14% (Table 3).

C-reactive protein (Table 4) in controls is much higher ($p<0.001$) than in cricketers, but still within the reference range of 0-8 mg/L accepted by Lancet Laboratory, South Africa. The results in cricketers have shown that 79% D allele frequency was associated ($p<0.001$) with lower CRP levels (<3.0 mg/L). Uric acid (<0.30 mmol/L) was associated ($p=0.001$) with 43% D allele frequency.

BMI, LBM and FM were higher ($p<0.001$) in cricket players (Table 5). High D allele frequency (91-94%) were associated with BMI and FM in cricketers ($p=0.001$) and in controls ($p=0.029$), where LBM has shown an association with 71% D allele ($p<0.041$) and with 94% D allele ($p<0.029$) respectively.

No differences in grip strength and the strength of the quadriceps (knee extension) and hamstring (knee flexion) muscles between the groups were observed (Table 6).

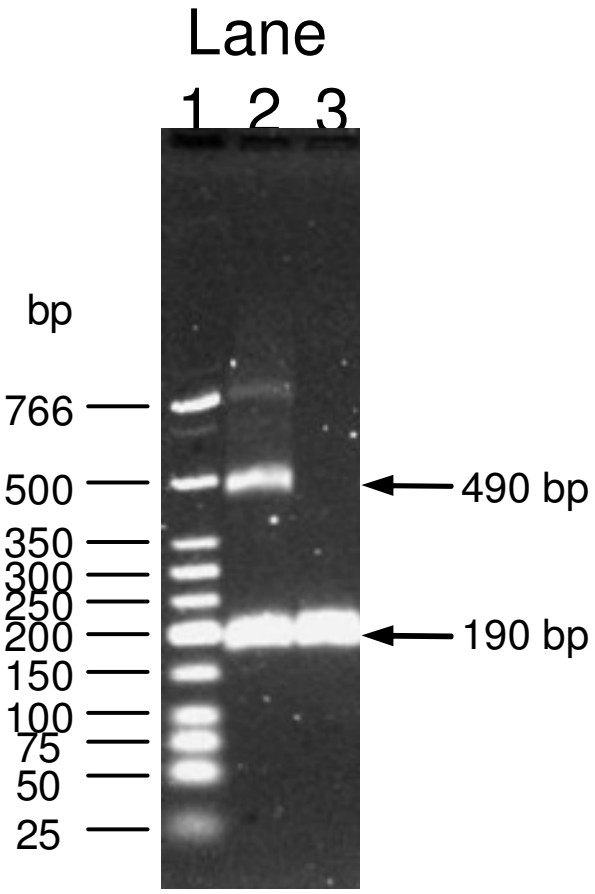

Figure 1. Analysis of ACE ID and DD genotypes. Amplified fragments were resolved in 2% (w/v) agarose, 1 ×TBE gels. Lane 1 - Low molecular weight DNA ladder (New England Biolabs); Lane 2 - ID genotype; Lane 3 - DD genotype. II genotype is absent.

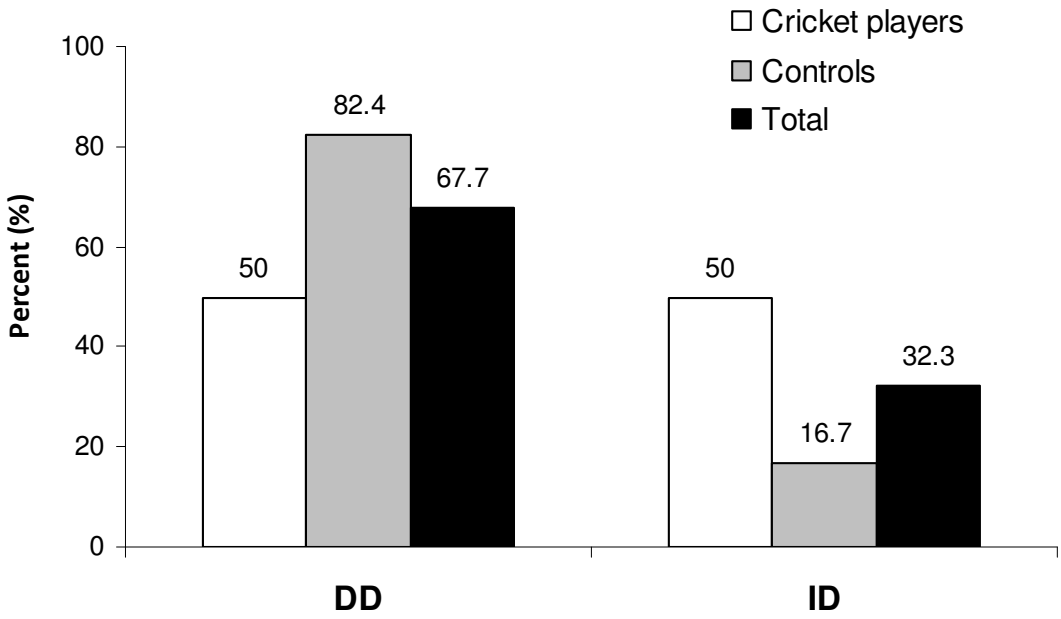

Figure 2. ACE DD and ID genotype frequencies (%) in cricketers, controls and the whole group.

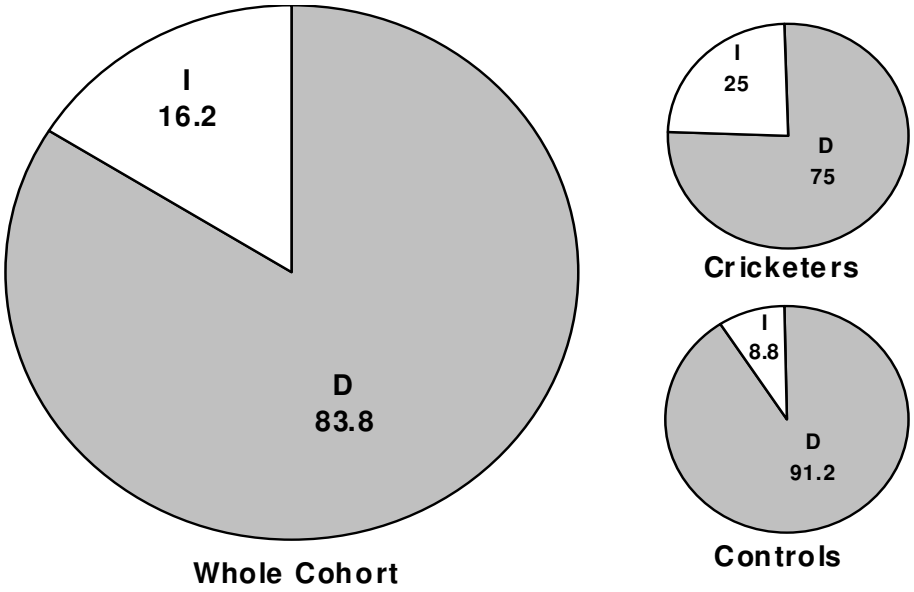

Figure 3. D and I allele frequencies (%) for the whole cohort, cricketers and controls.

Table 2. Systolic and diastolic blood pressure (mmHg) pulse pressure, heart rate (beats/min) and systolic tension time in cricket players and control group (mean ± SEM).

Parameters	Cricket players	Control group
Systolic blood pressure mmHg	120.81 ± 2.03	123.75 ± 1.07*
Diastolic blood pressure mmHg	76.88 ± 1.4	81.13 ± 1.31**
Pulse pressure mmHg	43.00 ± 1.29	42.00 ± 2.01
Heart rate beats/min	60.75 ± 0.85	59.80 ± 1.11
Systolic tension time (SBP x HR)	7654.27 ± 147.59	7253.60 ± 124.48*

*p < 0.05; *p > 0.0001.

Table 3. Basic metabolic rate, metabolic rate and energy requirements (kilojoules) in cricket players and control group (mean ± SEM).

Parameters	Cricket players	Control group
Basic metabolic rate	1502.40 ± 25.91	1480.31 ± 13.34*
Metabolic rate	2246.99 ± 102.24	2035.40 ± 18.34**
Energy requirements (kilojoules per food intake)	9737.41 ± 76.79	8520.18 ± 76.79**

*p < 0.05; *p > 0.0001.

Table 4. C-reactive protein (CRP), uric acid (UA) and lactate (LA) blood levels at rest in cricket players and control group (mean ± SEM) and ACE association tests.

Biomarkers	Cricket players	Control group
C-reactive protein (mg/L)	1.81 ± 0.37 [a,]	5.81 ± 0.51** NS
Uric acid (mmol/L)	0.31 ± 0.008 [b]	0.29 ± 0.007* NS
Lactate (mmol/L)	1.55 ± 0.08 NS	1.95 ±. 0.11** NS

Student's *t*-test: *p<0.05 control group vs. cricket players; **p<0.001 control group vs. cricket players.
Association tests: [a]p=0.001 CRP <3 mg/L in cricketers – ACE D allele frequency (79%), I allele (21%); [b]p=0.001 UA <0.30 mmol/L in cricketers – ACE D allele frequency (43%), I allele (57%); NS – no significant association tests.

Table 5. Physical characteristics of cricket players and control group (mean ± SEM) and ACE association tests.

Physical characteristics	Cricket players	Control group
Weight (kg)	68.68 ± 2.54	61.00 ± 1.61 **
Stature (cm)	175.08 ± 1.25	170.58 ± 0.33 **
Body mass index-BMI (kg/m^2)	22.40 ± 0.81 [a,]	20.79 ± 0.36 **, [c]
Lean body mass-LBM (kg)	61.81 ± 2.01 [b]	55.41 ± 1.49 **,C
Fat mass-FM (kg)	6.87 ± 0.54 [a,]	5.59 ± 0.22 **,C
Fat %	9.84 ± 0.39 NS	9.13 ± 0.32 * NS

Student's *t*-test: *p< 0.05 control group vs. cricket players; **p<0.001 control group vs. cricket players.
Association tests for BMI, LBM and FM: [a]p=0.001 BMI below 22.4 kg/m^2 , and FM below 6.9 kg – ACE D allele frequency (91%), I allele frequency (9%) in cricketers; [b]p=0.041 LBM below 61.8 kg – ACE D allele frequency (71%), I allele frequency (29%) in cricketers; [c]p=0.029 BMI below 22.4 kg/m^2 , LBM below 55.4 kg and FM below 5.6 kg – ACE D allele frequency (94%), I allele frequency (6%) in controls; NS – no significant association tests.

Table 6. Grip strength (kg), quadriceps strength (knee extension - Nm/kg) and hamstring strength (knee flexion - Nm/kg) of cricket players and control group (mean ± SEM) and ACE association tests.

Physical characteristics	Cricket players	Control group
Grip strength – L (kg)	44.00 ± 2.12 NS	42.57 ± 1.97 NS
Grip strength – R (kg)	45.79 ± 2.25 NS	45.11 ± 1.84 NS
Knee extension – L (N/kg)	3.73 ± 0.11 [a,]	3.71 ± 0.13 [e,]
Knee extension – R (N/kg)	3.63 ± 0.08 [b]	3.59 ± 0.12 [e]
Knee flexion –L (N/kg)	2.04 ± 0.08 [c]	2.08 ± 0.88 [f]
Knee flexion –R (N/kg)	2.00 ± 0.02 [d]	2.00 ± 0.08 [f]

L = left; R = right. Association tests: NS – no significant association tests for grip strength (L) and (R) per group of cricketers and controls. p=0.037 for the whole cohort grip strength (L) and (R) above 44.3 kg – ACE D allele frequency (86%) and I allele frequency (14%). [a]p=0.010 Knee extension (L) .above 3.7 Nm/kg ACE D allele frequency (86%) and I allele frequency (14%) in cricketers. [b]p=0.014 Knee extension (R) above 3.6 Nm/kg – ACE D allele frequency (79%); I allele frequency (21%) in cricketers. [c]p=0.014 Knee flexion (L) above 2.0 Nm/kg – ACE D allele frequency (78%) and I allele frequency (22%) in cricketers. [d]p=0.014 Knee flexion ® above 2.1 (Nm/kg) –ACE D allele frequency (83%) and I allele frequency (175) in cricketers. [e]p=0.001 Knee extension (L) above 3.7 Nm/kg and (R) above 3.6 N/kg – ACE D allele frequency (83%) and I allele frequency (17%) in controls.[f]p= 0.001 Knee flexion (L) and (R) above 2.0 Nm/kg – D allele frequency 87% and I allele frequency (13%) in controls

Knee extension L (>3.73 Nm/kg) and R (>3.63 Nm/kg) was associated with D allele frequency of 86% (p=0.010) and 79% (p=0.014). Knee flexion L (>2.04 Nm/kg) and R (>2.0 Nm/kg) was associated (p=0.014) with D allele frequency of 78% and 83%. For the whole cohort (Table 7) 86% D allele frequency was associated (p=0.037) with grip strength L (>43.3 kg) and R (R>45. 5 kg).

DISCUSSION

In our study a complete absence of ACE II genotype was established for the first time in Zulu South Africans. The genotype distribution for the whole cohort was skewed (67.7% DD and 32.3% ID). Low frequency of II genotype was reported in African Americans, Kenyans, Jamaicans (Scott et al., 2005), Nigerians (Woods, 2009) and Xhosa South Africans (Payne et al., 2007). The ACE distribution in Caucasian Europeans (Woods, 2009) was found to be in ratio 1:2:1 (e.g. 26% DD, 50%ID, and 24% II in British males).

Collins et al. (2004) tested a mixed group of South African-born athletes participating in Ironman triathlons and observed genotype frequencies of 24.3% DD, 54% ID and 21.6% II compared to 32.5% DD, 50.6% ID and 16.95% II in controls, pointing out that significantly higher 51.5 % ACE I allele frequency was found in the fastest South African finishers. The frequency of I allele was higher in Lithuanian elite athletes than in controls (Gineviciene et al., 2010). On the other hand, a large study of East African distance runners did not find any association between ACE genotypes and elite endurance athletic status (Scott et al., 2010). In the present study no over presentation of DD and ID genotypes was displayed in Zulu cricketers.

Our findings of high D allele frequency in Zulu South Africans are in line with the trend reported in Afro-Caribbean people (Berley et al., 1996), Nigerians (Woods, 2009) and elite Taekwondo athletes of Turkish and Azerbaijan origin (Gunay et al., 2010). The excess of D allele was represented more in athletes participating in power-oriented and short-distance/high intensity events (Myerson et al., 1999; Woods et al., 2000; Nazarov et al., 2001; Cerit et al., 2006; Charbonneau et al., 2008).

Previously, we used the same cohort to investigate the

Table 7. ACE D and I allele frequency (%) association with left (L) and right (R) grip strength in the whole cohort.

Physical test		Whole cohort		
		D (%)	I (%)	P
Grip Strength (L)	< M	92	8	-
	> M	81	19	-
Total	> M	86	14	0.037
Grip Strength (R)	< M	92	8	-
	> M	81	19	-
Total	> M	86	4	0.037

association of ACTN3 gene polymorphism and CRP, uric acid and lactate. Therefore in this study after performing ACE genotyping we conducted the association tests using the same results of the above mentioned biomarkers.

We found that the high frequency of power linked ACE D allele is also associated with lower CRP and uric acid levels in cricketers. It is important to mention that this is in concordance with our previous findings of strong association in the same cohort between these biomarkers and another power-related R allele of the ACTN3 gene (Djarova et al., 2011). C-reactive protein might be involved via cytokines in triggering metabolic signaling pathways in the exercising muscles that could be under genetic control by both genes.

It is important to emphasize that associations of the same trend as the above mentioned between high ACE D allele and high ACTN3 R allele frequencies, and BMI, LBM and FM were also found. ACE I/D polymorphism associations with BMI and body fat have been reported (Thompson et al., 2007) suggesting that it may affect adherence to exercise training.

In sports events like cricket requiring short power/sprint bursts, considering the fact that both ACE D and ACTN3 alleles have shown similarity in the association tests is a finding of interest that needs further studies. The other similarity found was the complete absence of ACE II genotype and ACTN3 XX genotype.

When comparing cricketers to control subjects we established no differences in heart rate, lower BP and higher SST, metabolic rate and energy requirements. The I/D polymorphism may play a role in enhanced performance but this is not mediated by differences in the heart rate/VO_2 relationship to training (Woods, 2009). Lower blood pressure and higher systolic tension time at rest indicate better cardiac efficiency in cricketers. Despite the interaction between ACE genotypes and serum ACE activity and the fact that D allele has been related to higher circulating/tissue ACE levels and enhanced performance, no associations between I and D alleles and BP have been reported (Bloem et al., 1996; Ostrander et al., 2009). Significantly higher estimated energy requirements were noted in cricketers. This corresponds to the findings of Noakes and Durandt (2000) that the energy demands of different cricket activities varied from 760 kJ/h in fielding to 1064 kJ/h in bowling and 1368 kJ/h in batting.

Strength, flexibility and speed parameters are among many factors contributing to the success in cricket (Nunes and Coetsee, 2007). The high D allele frequency association with grip strength that was established in the whole cohort in our study is in accordance with the findings that the D allele is related to the power/sprint output (Ruiz et al., 2010). Associations between the higher values of quadriceps/hamstring strength in cricketers and ACE D allele frequency has been observed for the first time. In batting and especially in fast bowling the trunk must flex, extend and rotate within a short period. The knee circumvents through flexion, rotation and extension. The bowling arm circumvents through extension, abduction, external rotation, thrusting flexion and internal rotation (Stuelcken et al., 2007). The average running sprint between the wickets was found to be 18.7 km/h which reflects high intensity work bouts (Christie and King, 2008).

The interpretation of association studies has always been controversial especially when the limitation is the small sample. Genetic studies need large population samples, but it is difficult to reconcile this premise with the scarce number of world-class champions or a given ethnicity and sport event (Ruiz et al., 2010). The analysis of a single sport discipline and association with I/D ACE gene polymorphism has been done in groups of athletes from 25 elite climbers, 37 swimmers and 27 up to 291 runners (Woods, 2009).

The unique demands of cricket may require specific physical characteristics and genetic traits play a substantial role. The perspective is to consider individual genetic endowment and develop training programmes that allow it to be optimized (Ostrander et al., 2009; Djarova et al., 2011). This study also might provide insight in talent identification and nurturing of young South African cricketers of various ethnicities.

Although, the number of participants in this study is small, it is concluded that ACE I/D genotyping has shown a complete absence of II genotype. Zulu cricket players

display a balanced DD and ID genotypes distribution in conjugation with significantly higher D allele frequencies associated with physical tests and beneficial differences in blood pressure and systolic tension time compared to controls. This could be considered as a competitive advantage in the cricket training and performance.

REFERENCES

Alvarez R, Terrados N, Ortolano R, Iglesias-Cubero G, Reguero JR, Batalia A, Cortina R, Fernandez-Garcia B, Rodriguez C, Braga S, Alvarez V, Coto E (2000). Genetic variation in the rennin-angiotensin system and athletic performance. Eur. J. Appl. Physiol., 82: 117-120.

Alvarez V, Coto E (1998). Angiotensin-converting enzyme and angiotensin II receptor 1 polymorphisms: association with early coronary disease. Cardiovasc. Res., 40: 375-379.

Amir O, Amir R, Yamin R, Attias E, Enion N, Sagiv M, Meckel Y (2007). The ACE deletion allele associated with Israeli elite endurance athletes. Exp. Physiol., 92: 881-886.

Andonov S, Saraeva R, Andonova S, Kaneva R, Gigova V, Stefanov L, Kremenski I, Atanassov P (2008). Polymorphism of ACTN3, ACE and AMPD1 genes and physical performance in Bulgarian sub-elite athletes. European Human Genetics Conference, Barcelona, 31 May–3 June, Supplement 2. Eur. J. Hum. Genet., pp. 288-289.

Ashton LA, Myers S (2004). Serial grip strength testing-Its role in assessment of wrist and hand disability. Int. J. Surg., 5: 1528-1642.

Bloem LJ, Manatunga AK, Prat JH (1996). Racial differences in the relationship of an angiotensin I–converting enzyme gene polymorphism to serum angiotensin I–converting enzyme activity. Hypertension, 27: 62-66.

Bray MS, Hagbero JM, Perusse L, Rankinen T, Roth SM, Wolfarth B, Bouchard C (2008). The human gene map for performance and health-related fitness phenotypes: The 2006-2007 update. Med. Sci. Sports Exerc., 41(1): 34-72.

Calo CM, Vona G (2008). Gene polymorphisms and elite athletic performance. J. Anthropol. Sci., 86: 113-131.

Cerit M, Colakoglu M, Erdogan M, Berdeli A, Cam FS (2006). Relationship between ace genotype and short duration aerobic performance development. Eur. J. Appl. Physiol., 98: 461-465.

Charbonneau DE, Hanson ED, Ludlow AT, Delmonico MJ, Hurley BF, Roth SM (2008). ACE genotype and the muscle hypertrophic and strength responses to strength training. Med. Sci. Sports Exerc., 40: 677-683.

Christie CJ, King GM (2008). Heart rate and perceived strain during batting in a warm and cold environment. Int. J. Fitness, 4(1): 33-38.

Coetsee MF (1995). Isoknee α Instruction Manual, Isokinetic Equipment cc Publisher, South Africa, pp. 1-163.

Collins M, Xenophontos SL, Cariolou MA, Mokone GG, Hudson DE, Anastasiades L, Noakes TD (2004). The ACE gene and endurance performance during the South African Ironman Triathlons. Med. Sci. Sports Exerc., 36: 1314-1320.

Davids K, Baker J (2007). Genes environment and sports performance. Sports Med., 37: 901-980.

Djarova T, Watson G, Basson A, Grace J, Cloete J, Ramakoaba A (2011). ACTN3 and TNF gene polymorphism association with C-reactive protein uric acid, lactate and physical characteristics in young African cricket players. Afr. J. Biochem. Res., 4: 22-27.

Folland J, Leach B, Little T, Howker K, Myerson S, Montgomery H (2000). Angiotensin converting enzyme genotype affects the response of human skeletal muscle to functional overload. Exp. Physiol., 85: 75-579.

Gineviciene V, Pranckeviciene E, Millasius K, Kuchinskas V (2010). Relating fitness phenotypes to genotypes in Lithuanian elite athletes. Acta Medica Lithuanica, 17: 1-10.

Gunay M, Ulkuer MK, Celenk C, Bezci S, Gokdemiir K, Gevat C, Kesici T (2010). Angiotensin-converting enzyme polymorphism in elite Taekwondo athletes of Turkish and Azzerbaijan Taekwondo teams. Ovidia University Annals, Series Physical Education and Sports/Science, Movement Health, 10: 165-168.

Jones A, Woods DR (2003). Skeletal muscle RAS and exercise performance. Int. J. Biochem. Cell Biol., 35: 855-866.

Koley S, Yadav MK (2009). An association of hand grip strength with some anthropometric variables in Indian cricket players. Facta Universitatis, Phy. Edu. Sport, 7: 113-123.

Lakka HM, Lakka TA, Rankinen T, Rice T, Rao DC, Leon AS, Skinner JS, Bouchard C (2006). The TNF-α G308A polymorphism is associated with C-reactive protein levels: The HERITAGE Family Study. Vascul. Pharmacol., 44: 377-383.

Lucia A, Moran M, Zihong, H, Ruitz R (2010). Elite Athletes: Are the Genes the Champions. Int. J. Sports Physiol. Perform., 5: 93-102.

MacArthur DG, North KN (2004). A gene for speed? The evolution function of alpha-actinin 3 gene. Bio. Essays, 26: 785-789.

Martens R (2004). Successful Coaching (3rd edition). Champaign Illinois, USA, Human Kinetics.

Milander L, Stein DJ, Collins M (2009). The interleukin-6, serotonin transporter, and monoamine oxidase A genes and endurance performance during the South African Ironman Triathlon. Appl. Physiol. Nutr. Metab., 34: 858-865.

Min S-K, Takahashi K, Ishigami H, Hiranuma K, Mizuno M, Ishii T, Kim C-S, Nakazato K (2009). Is there a gender difference between ACE gene and race distance? Appl. Physiol. Nutr. Metab., 34: 926-932.

Myerson S, Hemingway H, Budget R, Martin J, Humphries S Montgomery H (1999). Human angitensin I-converting enzyme gene and endurance performance. J. Appl. Physiol., 87: 1313-1316.

Nazarov IB, Woods DR, Montgomery VA, Shneider OV, Kasakov VT, Tomilin NV, Rogozkin VA (2001). The angiotensin converting enzyme I/D polymorphism in Russian athletes. Eur. J. Hum. Genet., 9: 797-801.

Noakes TD, Durandt JJ (2000). Physiological requirement of cricket. J. Sports Sci., 18: 919-929.

Nunes T, Coetsee B (2007). The contribution of isokinetic strength parameters to the performance of cricket batsmen. Isokinetic Exerc. Sci., 15: 233-244.

Ostrander EA, Huson HJ, Ostrander GK (2009). Genetics of athletic performance. Annu. Rev. Genomics Hum. Genet., 10: 407-429.

Payne JR, Dhamrait SS, Gohlke P, Cooper J, Scott, RA, Pitsiladis YP, Humphries SE, Rayner B, Montgomery HE (2007). The impact of ACE genotype on serum ACE activity in a Black South African male population. Ann. Hum. Genet., 71: 1-7.

Pescatello LS, Kostek MA, Gordish-Dressman H, Thompson PD, Seip RI, Price TB, Angelopoulos TJ, Clarkson PM, Gordon PM, Moyna NM, Visich PS, Zoeller RF, Devaney JM, Hoffman EP (2006). ACE ID genotype and the muscle strength and size response to unilateral training. Med. Sci. Sports Exerc., 38: 1074-1081.

Pieruzzi F, Abassi A, Keiser HR (1995). Expression of rennin-angitensin system compounds in heart, kidneys and lungs of rats with experimental heart failure. Circulation, 92: 3105-3112.

Rankinen T, Perusse L, Gagnon J, Changon YC, Keon AC, Skinner JS, Wilmore JH, Rao DC, Bouchard C (2000). Angiotensin-converting enzyme ID polymorphism and fitness phenotype in the HERITAGE family study. J. Appl. Physiol., 88: 1029-1035.

Rigat B, Hubert C, Athenc-Gelas F, Cambien F, Corvol P, Soubrier F (1990). An insertion/deletion polymorphism in the angiotension I-converting enzyme gene accounting for half the variance of serum levels. J. Clin. Invest., 86: 1343-1346.

Ruiz JR, Arieta D, Buxens A, Arieda M, Gomez-Galego F, Santiago C, Yvert T, Moran M, Lucia A (2010). Can we identify a power-oriented polygenic profile? J. Appl. Physiol., 108: 561-566.

Scanavini D, Bernardi F, Castoldi E, Conconi F, Mazzoni G (2002). Increased frequency of the homozygous II Ace genotype in Italian Olympic endurance athletes. Eur. J. Hum. Genet., 10: 576-577.

Scott RA, Irving R, Irwin L, Morrison E, Charlon V, Austin K, Tladi D, Deason M, Headley S, Kolkhorst FW, Yang N, North K, Pitsiladis Y (2010). ACTN3 and ACE genotypes in elite Jamaican and US sprinters. Med. Sci. Sports Exerc., 42(1): 107-112.

Scott RA, Moran C, Wilson RH, Onywera V, Boit MK, Goodwin WH, Gohike P, Montgomery H, Pitsiladis YP (2005). No association between angiotensin converting enzyme (ACE) gene variation and endurance athlete status in Kenyans. Comp. Biochem. Physiol. Mol. Intregr. Physiol., 141: 169-175.

Sipahi T, Budak M, Sen S, Ay A, Seneqr S (2006). Association between

Ace gene insertion (I) / deletion (D) polymorphism and primary hypertension in Turkish patients of Trakya region. Botechnol. Biotechnol. Eq., 20: 104-108.
Stuelcken M, Pyne D, Sinclair P (2007). Anthropometric characteristics of elite cricket fast bowlers. J. Sports Sci., 25: 1587-1597.
Thompson J, Manore MM (1996). Predicted and measured resting metabolic rate of male and female endurance athletes. J. Am. Diet Assoc., 96: 30-34.
Thompson J, Raitt J, Hutchings, I, Bjargo E, Loset A, Grocott M, Montgomery H, Caudwell E (2007). Angiotenzin-converting enzyme genotype and successful ascent to extreme high altitude. Extreme Everest Research Group. High Alt. Med. Biol., 8: 278-283.
Thompson WR, Gordon NF, Pescatello LS (2000). Guidelines for exercise training and prescription. American College of Sports Medicine. 6th Ed. Philadelphia, PA. Williams and Wilkins.
Tsianos GI, Evangelou E, Boot A, Zillikens MC, van Meurs JNJ, Uitterlinden AG, Ioannidis JPA (2009). Associations of polymorphism of eight muscle- or metabolism-related genes with performance of Mount Olympus marathon runners. J. Appl. Physiol., 108: 569-574.
Wagner H, Thaller S, Dahse R, Sust M (2006). Biomechanical muscle properties and angiotensin-converting enzyme gene polymorphism: a model-based study. Eur. J. Appl. Physiol., 98: 507-515.
Wang P, Fedoruk MN, Rupert JL (2008). Keeping pace with ACE. Sports Med., 38: 1065-1079.
Watson TA, Callister R, Taylor RD, Sibbritt DW, MacDonald-Wicks LK, Garg ML (2005). Antioxidant restriction and oxidative stress in short-duration exhaustive exercise. Med. Sci. Sports Exerc., 37: 63-71
Williams AG, Folland JP (2008). Similarity of polygenic profiles limits the potential for elite human physical performance. J. Physiol., 1(586): 113-121.
Wolfarth B, Rivera MA, Oppert JM, Bowlay MR, Dionne FT (2000). A polymorphism of the alpha 2a – adrenoreceptor gene and endurance athlete status. Med. Sci. Sports Exerc., 32: 1709-1712
Woods D (2009). Angiotensin-converting enzyme, rennin-angiotensin system and human performance. In Collins M (Ed) Genetics and Sports. Med. Sport Sci. Basel, Karger, 54: 72-87.
Woods DR, Brull D, Montgomery HE (2000). Endurance and the ACE I/D polymorphism. Sci. Prog., 84: 317-336.
Woods DR, World M, Rayson MP, Williams AG, Jubb M, Jamshidi Y, Hayward M, Mary DASG, Humphries SF, Montgomery HF (2002). Ednurance enhancement related to the human angiotensin I-converting enzyme I-D polymorphism is not due to differences in the cardiorespiratory response to training. Eur. J. Appl. Physiol., 86: 240-244.
Zhang B, Tanaka H, Shono N, Miura S, Kkiyomaga A, Shindo M, Saku K (2003). The lallele of the angiotensin-converting enzyeme gene is associated with an increased percentage of slow twitch type I fibres in human skeletal muscle. Clin. Genet., 63: 139-144.