

ACE ID genotype and leg power in Rugby Union players

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Aim. The present study examined the hypothesis that there were no significant differences between forwards and backs in the elements of leg power between the ID and DD genotypes of the ACE (I/D) gene in *developing* young adult Rugby Union players.

Methods. Sixty-eight players were recruited to identify the distribution of genotypes between forwards and backs. Fifty-eight players were investigated for leg power. Forwards (n=28) comprised 15 ID and 13 DD genotypes, and backs (n=30) 19 ID and 11 DD genotypes. Leg power was measured on a force platform using a counter movement jump; the parameters of interest were peak and relative force, peak and relative power, displacement and velocity. The three-primer polymerase chain reaction was used to assay the region of interest for I and D variants of the ACE gene. The distribution of genotypes was determined by chi-square and comparisons between forwards and backs made using the independent t-test.

Results. No significant differences were identified in the distribution of genotypes between forwards and backs ($\chi^2=2.2$, $P=0.336$). However, significant differences were identified between forwards and backs in a number of components of leg power. Backs had significantly larger values than forwards for relative force (1.50 vs. 1.30 Wt%, $P=0.001$) and relative power (27.1 vs. 24.3 W.kg⁻¹, $P=0.034$) for the ID genotype, whereas backs had significantly larger values than forwards for displacement (0.42 vs. 0.38 m, $P=0.049$) and velocity (2.76 vs. 2.55 m.s.⁻¹, $P=0.007$) for the DD genotype.

Conclusions. The characteristics of leg power identified will enhance the functional requirements of players according to playing position and commitment.

KEY WORDS: ACE (I/D) gene - Leg - Power - Rugby Union Players.

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In the modern game of Ruby Union football, fitness has become increasingly important, particularly the components of strength and power. In general terms, strength is defined as the force that a muscle or muscle group can generate; power on the other hand, is the rate at which work is performed.¹

Functionally, rugby union players are classified into one of two discrete playing units; forwards (n=8) or backs (n=7). The distinction arises as a result of their contrasting playing commitment. Although players have individual performance responsibilities, they are, nevertheless, expected to have the basic skills required to compete in aspects of general play.

Our original study of rugby union players² was undertaken to identify the distribution of polymorphisms of the ACE (I/D) gene in a homogeneous group of *developing* young adult players. The findings of the study showed that there were no significant differences between rugby players (n=109) and controls (n=108), ($\chi^2=2.4$, $P=0.31$, power = 0.49); forwards (n=56) and backs (n=53), ($\chi^2=2.6$, $P=0.279$, power=0.51); or individual playing positions (n=109), ($\chi^2=4.5$, $P=0.607$, power=0.93).

A polymorphism of the human gene encoding ACE (17q23)³ consists of the presence (insertion, I allele) or absence (deletion, D allele) of a 287 amino acid

base-pair fragment found within intron 16.^{4, 5} Individuals carry two versions of the ACE allele, therefore three ACE genotypes are identified: individuals homozygous for the insertion (II) and deletion (DD) alleles, and individuals heterozygous for the ID allele. The presence of the I allele is associated with low levels of ACE activity in serum and tissues, whereas the D allele is associated with increased levels of ACE activity.⁶

A number of studies have suggested that the I allele is associated positively with enhanced endurance performance. These sports include, for example, mountaineering,^{7, 8} long-distance swimming,⁹ triathlons,¹⁰ middle/long distance running,^{11, 12} and rowing.¹³ Where admixtures of athletes from different endurance events have been used, likewise, an over-representation of the I allele has frequently been observed.¹⁴ On the other hand, there are studies which have failed to identify a definitive relationship between the I allele and endurance performance.¹⁵⁻¹⁷

The D allele is thought to be associated with anaerobic performance; for example short distance sprinting events¹² and sprint swimming.¹⁸ However, it has recently been shown¹⁹ that elite Israeli marathon runners have a greater proportion of the DD genotype than elite sprinters; and top level professional 3 wk-tour cyclists a greater distribution of the D allele and DD genotype than other endurance athletes.²⁰

Several investigations have demonstrated a positive relationship between the ACE (I/D) genotype and strength/strength gains.²¹⁻²³ Others have found little influence of the ACE I/D gene on muscular size, endurance and strength.²⁴

The null hypothesis of the present study, therefore, was that there were no significant differences between forwards and backs in the elements of leg power between the ID and DD genotypes of the ACE (I/D) gene in *developing* young adult rugby union football players.

Materials and methods

Study design and participants

A cross-sectional case-control study was employed using a candidate gene approach. A university-based group of 68 young adult male *developing* players, of mean age 20.4 ± 1.9 yr, all Caucasian, were participants in the study. Players were members of the University Rugby Centre of Excellence. Forwards ($n=35$) comprised 7 II, 15 ID and 13 DD genotypes, and backs

($n=33$) 3 II, 19 ID and 11 DD genotypes. A statistically insufficient numbers of players with the II genotype completed the counter movement jump (CMJ) (7 forwards and 3 backs) thus the study ($n=58$) proceeded with the analysis of ID ($n=34$) and DD genotypes ($n=24$).

Jones *et al.*²⁵ outlined a gene-environmental model which was used as a prototype for the study. A homogeneous group of *developing* young adult players participating in similar skill and conditioning programmes, and having comparable lifestyle conditions were screened for participation in the study from a larger cohort of players by the Director of Rugby. The study was approved by the University Ethics Committee and written informed consent obtained from individuals.

Leg power

Leg power was selected as the experimental variable since the characteristics of the lower limbs in rugby union players are crucial for effective performance. Each participant performed three separate counter movement jumps (CMJ) using a piezo-electric 0.9×0.6 m force platform (Kistler 9287BA, Germany) utilizing the protocol described by Linthorne.²⁶ Individual practice trials were allowed before the final test measurements were taken. Instrumentation was calibrated and body weight measured on the force platform before the commencement of each jump. Kistler BioWare software (version 3.2.6) was used to analyse the data. The maximal CMJ was selected for analysis.

Anthropometry and body composition

Stature was measured to the nearest 0.1 cm using a Harpenden stadiometer and body mass to the closest 0.1 kg on a digital weighing scale. Lean leg tissue mass (LLTM) was measured by dual-energy X-ray absorptiometry (DXA) using the Hologic QDR Discovery A (S/N 70902) fan-beam model. Calibration was checked on a daily basis using external phantoms. The digital image of each subject was partitioned into regional anatomical segments comprising the head, right and left arms, trunk, and right and left legs using Hologic software (version 12.4:3). The quality of images and analyses was confirmed by a second independent experienced observer. The sum of right and left leg lean tissue mass (LTM) provided an estimate of the total leg LTM.

TABLE I.—Summary statistics of ID and DD genotypes for height and body mass (n=58) and lean leg tissue mass (n = 48) for forwards (F) and backs (B).

	ID genotype			DD genotype		
	Forwards	Backs	P	Forwards	Backs	P
Height (cm)	186.4±8.3	180.1± 6.1	0.015*	184.3±6.8	178.4±6.7	0.045*
Body mass (kg)	104.1± 13.4	84.5±5.9	0.0001*	98.4±9.2	80.6±4.9	0.0001*
LLTM (kg)	26.8±2.6	23.5±2.1	0.0001*	25.8±2.4	23.4±2.9	0.084

*Significant at given level. LLTM: lean leg tissue mass. Height and body mass: ID genotype (F = 15, B = 19), DD genotype (F = 13, B = 11). Lean leg tissue mass: ID genotype (F = 14, B = 17), DD genotype (F = 10, B = 7).

ACE genotype

Buccal cell DNA extraction was carried out (Qiagen, QIAamp DNA Micro Kit) on 5 ml saliva samples. Three-primer polymerase chain reaction (PCR) was used to assay the region of interest for I and D variants of the ACE gene.

Primer sequences:

— Forward

5' - GACTCTGTAAGCCACTGCTGGAG - 3'

— Reverse

5' - TCGCCAGCCCTCCCATGCCATAA - 3'

— Internal Forward

5' - TGGGACCACAGCGCCCGCCACTAC - 3'

Twenty-five ng of template DNA, 25 pmoles of each oligonucleotide, 0.2 mM of each dNTP, 0.5 units of Ampli Taq Gold DNA Polymerase and 1X PCR Buffer (15 mM MgCl₂) were mixed in a total volume of 25 µl. Cycling conditions were 94°C for 12 minutes, followed by 35 cycles of 94°C denaturing for 30s, annealing at 60°C for 30 s, extension at 72°C for 30 s, with a final extension period of 10 min at 72°C. A 5 µl sample of PCR product was mixed with 5 µl of Gel Loading Buffer and loaded into the well of a 2% agarose gel and electrophoresis run at 100v.

Statistical analysis

Descriptive statistics are presented as means and SDs. Levene's test was used to check homogeneity of variance. Pearson's chi-square was employed to compare the genotype distribution of players using the available genotype data for forwards (n=35) and backs (n=33). Comparison of ID and DD genotypes for power variables between forwards (n=34) and backs (n=24) were made using an independent t-test. The technical error of measurement (TEM) was employed to identify the degree of reliability of the parameters of the

CMJ. For force and power the TEM was 2.5N and 2.2W respectively; for displacement and velocity 0.03m and 0.15m.s⁻¹ respectively. Computations were carried out with SPSS versions 12 and 15. A value of p ≤ 0.05 was considered significant.

Results

There were no significant differences between the distribution of genotypes of forwards and backs. For forwards the genotype proportions were 0.20, 0.43 and 0.37 for II, ID and DD respectively, and for backs 0.10, 0.57 and 0.33 respectively ($\chi^2 = 2.2$, 2 df, P=0.34). Allele frequencies for forwards were I=0.41 D=0.59 ($\chi^2 = 1.63$, p = 0.20) and for backs I=0.38 D=0.62 ($\chi^2 = 2.92$, p = 0.09).

Table I outlines the significant differences for height, body mass and lean leg tissue mass in ID and DD genotypes. Height was significantly greater in forwards than backs for both ID (P=0.015) and DD (P=0.045) genotypes, as was body mass (both P=0.0001). Lean leg tissue mass was significantly greater in forwards for the ID genotype only (P=0.0001).

Significant differences occurred between forwards and backs in the elements of leg power; the variables concerned were, relative force, relative power, displacement and velocity (Table II).

Absolute force, although descriptively larger in forwards than backs in the ID (1329 vs. 1251 N) and DD genotypes (1260 vs. 1119 N) was not significant (P=0.261 and P=0.071 respectively). Relative force, on the other hand, was significantly greater in backs than forwards in the ID genotype (1.5 vs. 1.3 Wt %, P=0.001); values in the DD genotype were similar in both groups (1.30 vs. 1.38 Wt %) and not significant (P=0.289).

TABLE II.—Comparison of elements of leg power for ID and DD genotypes for forwards (F) and backs (B).

	ID genotype			DD genotype		
	Forwards	Backs	P	Forwards	Backs	P
Force (N)	1329±209	1251± 185	0.261	1260±175	1119±190	0.071
Force (Wt %)	1.30±0.2	1.50±0.2	0.001*	1.30±0.2	1.38±0.2	0.289
Power (W)	2542±525	2303±336	0.118	2277±393	2146±325	0.386
Power (W. kg ⁻¹)	24.3±3.9	27.1±3.6	0.034*	23.1±5.0	26.3±3.7	0.092
Power (W. kg ⁻¹ LM)	91.9±11.8	97.9±12.6	0.189	89.70±18.2	94.10±8.7	0.565
Displacement (m)	0.39±0.1	0.42±0.1	0.110	0.38±0.1	0.42±0.1	0.049*
Velocity (m.s ⁻¹)	2.62±0.15	2.73±0.20	0.101	2.55±0.19	2.76±0.13	0.007*

*Significant at given level. ID genotype (F = 15, B = 19), DD genotype (F = 13, B = 11) genotypes. Power (W. kg⁻¹ LM) ID genotype (F = 14, B = 17), DD genotype (F = 10, B = 7).

Absolute power, in either genotype, was not significant between forwards and backs (Table II), although as expected forwards were descriptively more powerful than backs in both ID (2542 vs. 2303 W, $P=0.118$) and DD (2277 vs. 2146 W, $P=0.386$) genotypes. In relative terms, backs had larger power values than forwards in the ID genotype (27.1 vs. 24.3 W. kg⁻¹, $P=0.034$), but not the DD genotype (26.3 vs. 23.1 W. kg⁻¹, $P=0.092$). Power expressed in relation to LLTM was unable to discriminate between forwards and backs in either the ID ($P=0.189$) or the DD genotype ($P=0.565$).

Displacement was significantly larger in the DD genotype of backs compared to forwards (0.42 vs. 0.38 m, $P=0.049$), while velocity was significantly faster in backs than forwards in the DD genotype (2.76 vs. 2.55 m.sec⁻¹, $P=0.007$).

Discussion

The present study has shown that there were significant differences between forwards and backs relative to ID and DD genotypes in a number of the elements of leg power; these were relative force, relative power, displacement and velocity.

In general, there is a lack of information regarding the influence of the ACE (I/D) gene in team games. Of the games reported in the literature, field-hockey ($n=53$) had a genotype (II, ID and DD) of 0.28, 0.47 and 0.25 ($P=0.768$); ice-hockey ($n=34$) one of 0.24, 0.50 and 0.26 ($P=0.49$).¹¹ The genotype of elite Italian association football players ($n=28$) was 0.14, 0.43 and 0.43 ($P=0.89$)²⁷ and handball ($n=15$) 0.20, 0.67 and 0.13.¹⁴ For comparison, the present group of forwards had a

genotype of 0.20, 0.43 and 0.37, and backs one of 0.10, 0.57 and 0.33 ($P=0.34$). The advantage of team games where genotypes are in Hardy-Weinberg equilibrium, is an increased size of the gene pool available for player selection, and the knowledge that environmental influences are fundamental for the development of skill, technique and playing performance.

Many of the performance activities undertaken by forwards and backs have been described by Roberts *et al.*²⁸ in their study of elite English rugby union players. Using time-motion analysis, they found that backs covered a greater overall distance than forwards, which incorporated greater distances in walking and high-intensity running. Forwards, on the other hand, engaged more frequently than backs in high-intensity activities, particularly those which included elements of static exertion, such as the scrummage.

The present study shows that both forwards and backs have the allele capability for endurance and power, although there tends to be an emphasis towards power. Because of the intermittent and varied nature of rugby union, the metabolic and physiological requirements involved will necessitate the delivery of a functional admixture of aerobic, anaerobic, strength and power resources.

Height and body mass were significantly greater in forwards than backs in both ID and DD genotypes, and LLTM significantly larger in forwards in the ID genotype only (Table I). These dimensions reflect the absolute nature of the playing requirement.

Because of their greater overall size, forwards demonstrated larger absolute amounts of force and power than backs (Table II); in a contact game such as rugby union these characteristics are necessary for securing and maintaining ball possession. When val-

ues are expressed relative to size, however, backs are found to have greater force and are more powerful than forwards. This was the finding for both force and power for the ID genotype, but not the DD genotype.

Displacement (Table II) was significantly greater in backs than forwards for the DD genotype and likewise velocity significantly greater in backs than forwards for the DD genotype. Displacement, enhanced by velocity, is advantageous in activities such as jumping to obtain possession of the high ball during attack and defense, and at restarts such as the line-out. It would also be beneficial at plays such as scrummages and rucks, where there is a reasonably close functional relationship between the application of leg power and the modality of the measurement of leg power. Greater velocity infers an increased capability for power and is particularly advantageous in running skills incorporating speed, acceleration, deceleration, and change of direction, all of which are distinguishing features of backs.

Using isometric and dynamic training practices, Folland *et al.*²¹ examined the effect of ACE (I/D) activity on changes in strength of the quadriceps muscle in untrained recreationally active males. No association was found between strength and genotype in the untrained state. Following training, there was a significant interaction between the ACE (I/D) genotype and isometric training, strength gains being greatest in individuals with the D allele (II=9%, ID=17.6%, DD=14.9%, $P<0.05$). The interaction between the ACE (I/D) genotype and training was consistent across all strength measures and both types of training (ID $P>$ DD $>$ II). The results of the present study (Table II) may be a reflection of this effect when backs are compared with forwards for relative force (1.50 vs. 1.30 W%, $P=0.001$) and relative power (27.1 vs. 24.3 W. kg⁻¹, $P=0.034$) for the ID genotype.

The most interesting feature of the Pescatello *et al.* study.²³ was that the fact that the ID genotype was associated primarily with cross-education and learning effects, rather than direct changes in the size and strength of muscle tissue arising from resistance training. This phenomenon would certainly provide beneficial learning effects in the training of performance skills, and may contribute covertly to the development of power.

Zhang *et al.*²⁹ found that ACE II subjects had a significantly greater percentage of type I fibres (50.1 vs. 30.5 %, $P<0.01$) and a corresponding lower percent-

age of fast-twitch type IIb fibres (16.2 vs. 32.9%, $P<0.01$) than DD subjects. In the vastus lateralis muscle of South African rugby players (Jardine *et al.*³⁰) the distribution of type IIb fibres in forwards and backs was found to be essentially the same (52.6 vs. 56.8 %, $P>0.05$). It is thought that Ang II may be influential in the redirection of blood flow from type I to type II fibres, thereby providing a temporary advantage during power-related activities.

Strength is the basic component that influences the dimension of power. Although increases in maximal strength are related to increases in relative strength and power, it is important to appreciate that power is not simply a by-product of strength. Strength needs to be adapted functionally in order to enhance the capability of power. The nature of the activities which facilitate these adaptations form part of the design of conditioning and training routines.

Conclusions

The present study has shown that there were significant differences between forwards and backs in association with ID and DD genotypes of the ACE (I/D) gene in a number of the elements of leg power. Backs had significantly larger values than forwards for relative force and relative power for the ID genotype, whereas backs had significantly larger distances than forwards for displacement and greater velocities for the DD genotype.

References

1. Knuttgen, HG, Komi P.V. Basic definitions for exercise. In: P.V. Komi (editor) *Strength and Power in Sport*. London: Blackwell Scientific Publications; 1992: 3-6.
2. Bell W, Colley JP, Gwynne JR, Llewellyn L, Darlington SE, Evans WD. Polymorphisms of the Angiotensin-Converting Enzyme ACE (I/D) and their distribution in developing young adult Rugby Union players. In: T. Reilly and G. Atkinson (eds). *Contemporary Sport, Leisure and Ergonomics*. London: Routledge; 2009; 161-173.
3. Hubert C, Houot A-M, Corvol P, Soubrier, F. Structure of the angiotensin I-converting enzyme gene. Two alternate promoters correspond to evolutionary steps of a duplicated gene. *J Biol Chem* 1991; 266: 15377-15383.
4. Rigat B, Hubert C, Alhenc-Gelas F, Cambien F, Corvol P, Soubrier F. An insertion/deletion polymorphism in the angiotensin I-converting enzyme gene accounting for half of the variance of serum enzyme levels. *J Clin Invest* 1990; 86: 1343-1346.
5. Tiret L, Rigat B, Visvikis S, Breda C, Corvol P, Cambien F, *et al.* Evidence, from the combined segregation and linkage analysis, that a variant of the angiotensin I-converting enzyme (ACE) gene controls plasma ACE levels. *Am J Hum Genet* 1992; 51: 197-205.

6. Danser AHJ, Maarten ADH, Schalekamp MD, Bax WA, van den Brink AM, Saxena PR, *et al.* Angiotensin-converting enzyme in the human heart. Effects of the deletion/insertion polymorphism 1995; *Circulation*; 92: 1387-1388.
7. Montgomery HE, Marshall R, Hemingway H, Myerson S, Clarkson P, Dollery C, *et al.* Human gene for physical performance. *Nature* 1998; 393: 221-222.
8. Tsianos G, Eleftheriou KI, Hawe E, Woolrich L, Watt M, Watt I, *et al.* Performance at altitude and angiotensin I converting enzyme genotype. *Eur J Appl Physiol* 2005 93:630-633.
9. Tsianos G, Sanders J, Dhamrait S, Humphries S, Grant S, Montgomery HE. The ACE gene insertion/deletion polymorphism and elite endurance swimming. *Eur J App Physiol* 2004; 92:360-362.
10. Collins M, Xenophontos SL, Cariolou MA, Makone GG, Hudson DE, Anastasiades L, *et al.* The ACE gene and endurance performance during the South African Ironman Triathlons. *Med Sci Sports Exerc* 2004; 36: 1314-1320.
11. Myerson S, Hemingway H, Budget R, Martin J, Humphries S, Montgomery HE. Human angiotensin I-converting enzyme gene and endurance performance. *J Appl Physiol* 1999; 87: 1313-1316.
12. Nazarov I.B, Woods DR., Montgomery HE, Shneider OV, Kazakov VI., Tomilin NV, *et al.* The angiotensin converting enzyme I/D polymorphism in Russian athletes. *Eur J Hum Genet* 2001; 9: 797-801.
13. Gayagay G, Yu B, Hambly B, Boston T, Hahn A, Celermajer DS, *et al.* Elite endurance athletes and the ACE I allele – the role of genes in athletic performance. *Hum Genet* 1998; 103: 48-50.
14. Alvarez R, Terrados N, Ortolano R, Iglesias-Cubero G, Reguero J.R, Batallo A, *et al.* Genetic variation in the renin-angiotensin system and athletic performance. *Eur J of Appl Physiol* 2000; 82: 117-120.
15. Taylor RR, Mamotte CDS, Fallon K, van Bockxmeer FM. Elite athletes and the gene for angiotensin-converting enzyme. *J Appl Physiol* 1999; 87: 1035-1037.
16. Rankinen, T, Wolfarth B, Simoneau JA, Maier-Lenz D, Rauramaa R, Rivera MA, *et al.* No association between angiotensin-converting enzyme ID polymorphism and elite endurance athlete status. *J Appl Physiol* 2000; 88: 1571-1575.
17. Scott RA, Moran C, Wilson RH, Onywera V, Boit MK, Goodwin WH, *et al.* No association between angiotensin converting enzyme (ACE) gene variation and endurance status in Keyans. *Comp Biochem Physiol* 2005. Part A 141, 169-175.
18. Woods DR, Hickman M, Jamshidi Y, Brull D, Vassiliou V, Jones A, *et al.* Elite swimmers and the D allele of the ACE I/D polymorphism. *Hum Genet* 2001; 108: 230-232.
19. Amir O, Amir R, Yamin C, Attias E, Eynon N, Sagiv M, *et al.* The ACE deletion allele is associated with Israeli elite endurance athletes. *Exp Physiol* 2007; 92.5: 881-886.
20. Lucia A, Gomez-Gallego F, Chicharro JL, Hoyos J, Celeya K, Cordova A, *et al.* Is there an association between ACE and CKMM polymorphisms and cycling performance status during 3-week races? *Int J Sport Med* 2004; 25: 442-447.
21. Folland J, Leach B, Little T, Hawker K, Myerson S, Montgomery H, *et al.* Angiotensin-converting enzyme genotype affects the response of human skeletal muscle to functional overload. *Exp Physiol* 2000; 85: 575-579.
22. Colakoglu M, Cam FS, Kayitken B, Cetinoz F, Colakoglu S, Turkmen M, *et al.* ACE genotype may have an affect on single versus multiple set preferences in strength training. *Eur J Appl Physiol* 2005; 95: 20-26.
23. Pescatello LS, Kostek MA, Gordish-Dressman, H, Thompson PD, Seip RL, Price TB, *et al.* ACE ID genotype and the muscle strength and size response to unilateral resistance training. *Med Sci Sport Exer* 2006; 38: 1074-1081.
24. Sonna LA, Sharp MA, Knapik JJ, Cullivan M, Angel KC, Patton JF, *et al.* Angiotensin-converting enzyme genotype and physical performance during US army basic training. *J Appl Physiol* 2001; 91: 1355-1363.
25. Jones A, Montgomery HE, Woods DR. Human performance: a role for the ACE genotype. *Exerc Sport Sci Rev* 2002; 30: 184-190.
26. Linthorne LP. Analysis of standing vertical jumps using a force platform. *Amer Assoc Physics Teachers* 2001; 69: 1198-1204.
27. Fatini C, Guazzelli R, Manetti P, Battaglini B, Gensini F, Vono R, *et al.* RAS genes influence exercise-induced left ventricular hypertrophy: an elite athletes study. *Med Sci Sports Exerc* 2000; 32: 1868-1872.
28. Roberts SP, Trewartha G, Higgitt RJ, El-Abd J, Stokes, KA. The physical demands of elite English Rugby Union. *J Sports Sci* 2008; 26: 825-833.
29. Zhang B, Tanaka H, Shono N, Miura S, Kiyonaga A, Shindo M, *et al.* The I allele of the angiotensin-converting enzyme gene is associated with an increased percentage of slow-twitch type I fibres in human skeletal muscle. *Clin Genet* 2003; 63: 139-144.
30. Jardine MA, Wiggins TM, Myberg KH, Noakes TD. Physiological characteristics of rugby players including muscle glycogen content and muscle fibre composition. *S Afr Med J* 1988; 73: 529-532.