Eur J Appl Physiol (2006) 98:507–515
DOI 10.1007/s00421-006-0293-7

ORIGINAL ARTICLE

Biomechanical muscle properties and angiotensin-converting enzyme gene polymorphism: a model-based study

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Accepted: 16 August 2006 / Published online: 28 September 2006

Abstract Previous studies reported an association of angiotensin-converting enzyme (ACE) I/D gene polymorphism with physical performance. The study was based on the hypothesis that certain individual biomechanical muscle properties could be associated with ACE genotype and that they could influence athletes' physical performance. Movement-independent individual biomechanical muscle properties of 62 sports students were determined by applying a mathematical model to experimental data. Subjects exerted concentric and isometric contractions at a leg-press. The model was based on a Hill-type muscle model, a function describing the geometrical arrangement of human leg extensor muscles, and an exponential function describing muscle activation. Mouthwash samples were taken to determine the ACE genotypes. Several combinations of experimentally determined biomechanical properties served as input variables for a discriminant analysis. We were able to show that individual biomechanical muscle properties correlated with ACE I/D gene polymorphism. With a combination of certain individual muscle parameters based on a Hill-type muscle model, we were able to separate three individual ACE genotypes (II, ID, DD) in a significant way ($P < 0.03$) and correctly classify 89% of the cases using a discriminant analysis. We conclude that local biomechanical muscle properties are influenced by ACE genotype.

Keywords ACE · Force velocity · Biomechanics · Sport · Muscle

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Introduction

Human physical performance and fitness phenotype are the products of genetics and environmental stimuli. A large number of genetic variants are likely to interact with diverse environmental stimuli to yield finally a range of variable intermediate sporting phenotypes. Recent studies highlighted an association of physical performance with human angiotensin-converting enzyme (ACE; dipeptidyl carboxypeptidase 1) gene locus.

Humans possess one of three possible combinations of the ACE gene polymorphism (II, ID or DD). This polymorphism is due to a variation in the structure of the ACE gene (Rigat et al. 1990; Tiret et al. 1992; Danser et al. 1995), i.e. a 287 bp Alu repeat sequence can be present (insertion, I allele) or absent (deletion, D allele).

The I allele has been found with increased frequency in elite distance runners (Myerson et al. 1999; Nazarov et al. 2001), high-altitude mountaineers (Montgomery et al. 1998), Olympic rowers (Gayagay et al. 1998) and professional athletes from mixed sporting disciplines (Alvarez et al. 2000). Furthermore, a significant interaction between ACE genotype and isometric training

was reported with greater strength gains shown by subjects with a D allele (Folland et al. 2000). In chronic obstructive pulmonary disease, the deletion allele is associated with greater quadriceps strength (Hopkinson et al. 2004). Other studies using heterogeneous cohorts of elite athletes failed to find an excess of the I allele (Karjalainen et al. 1999; Taylor et al. 1999; Rankinen et al. 2000). Nevertheless, recent data support a role for ACE in the regulation of human skeletal muscle strength, but do not confirm a role in altering the response to short-term training (Williams et al. 2005).

How can sporting phenotypes be influenced by the ACE gene polymorphism?

ACE genes exist in tissues where they influence tissue growth and fat metabolism, as well as an I allele-dependent increase in skeletal muscle glucose metabolism (Montgomery et al. 1999). Differences in sporting phenotypes might be in part indirectly mediated; e.g. the D allele is associated with physiological cardiac growth. Since cardiovascular performance is a key element in athletic success, links have been suggested between the ACE genotype and an anabolic response to exercise training, endurance and muscle performance. Enhanced endurance performance has been reported to be linked to lower enzyme activity, which is associated with the longer I allele (Montgomery et al. 1998; Woods et al. 2000) and the ability of the cardiorespiratory system to deliver oxygen to exercising muscles (reviewed in Woods et al. 2000).

More evidence comes from recent studies (Montgomery et al. 1998; Williams et al. 2000) that differences in muscular mechanical efficiency and not cardiorespiratory fitness in general might account for an enhanced muscle performance associated with the I allele. The ACE gene has been identified in skeletal muscle. It degrades (vasodilator) kinins and yields (pressor) Ang II that has been shown to be a necessary mediator of the growth response of muscles to mechanical load. This may explain that the insertion variant of the ACE gene (I allele) is associated with elite endurance athletic performance and the deletion variant (D allele) with power- and strength-related sports.

However, to date, the role for altered skeletal muscle performance characteristics in mediating such genetic associations has gone untested. From this, we designed our study based on the hypothesis that certain individual muscle properties such as the individual shape of the force–velocity relation could be associated with ACE genotype.

A non-invasive direct measurement of human individual muscle properties is methodically impossible and scaling data from animal studies to humans (Herzog 1994) can determine only standard muscle properties but not differences between human individuals. Therefore, a model-based method had been established (Sust 1996) to determine individual muscle properties based on non-invasive measurements.

If a human decides to perform a voluntary muscle contraction with maximum voluntary effort, the contraction dynamics are determined by the skeletal system, neuromuscular activation dynamics and individual muscle properties. Muscle forces will be transferred through joints and bones into external forces. Hence, internal muscle forces can be calculated from measured external forces and a geometrical model. We used a well-established computer algorithm, which searched for a set of muscle properties by minimizing the difference between simulated and measured forces. This method had been successfully validated on single muscle contractions (Wagner et al. 2005). The method requires contractions with maximum voluntary effort, but it is not restricted to young or physically educated people. Therefore, we do not need a 'normalized' group of subjects. The following individual muscle properties were determined individually: maximum isometric force f_{iso}, maximum contraction velocity v_{max}, maximum power output p_{max}, and a parameter S describing activation dynamics.

The first objective was to investigate whether individual biomechanical muscle properties, which contribute to a certain physical performance phenotype, are associated with ACE genotype. Furthermore, our purpose was to separate between ACE genotypes (II, ID, DD) from local biomechanical muscle properties, using a discriminant analysis.

Materials and methods

Subjects

Sixty-two Caucasian sports students participated in the study. Our cohort represented 29 male volunteers, mean age (±SD) 22.5 years (±2.4), height 1.81 m (±0.05), body mass 76.1 kg (±9.6), and 33 female volunteers of age 20.4 years (±1.9), height 1.70 m (±0.07) and body mass 60.2 kg (±7.7). Informed written consent to participate was obtained from all the subjects.

ACE genotyping

ACE genotype was determined from 0.9% saline mouthwash samples. Cells from 0.1 ml wash sample were digested by Proteinase K treatment (Falk et al. 1997). ACE I/D polymorphism was genotyped by PCR with previously published primer pairs and conditions (Tiret et al. 1992) in a 10-µl aliquot of each lysate followed by agarose-gel electrophoresis. As ACE D allele was preferentially amplified, ID genotypes could be mistyped as DD. Thus, all DD samples from a standard amplification procedure were confirmed by an insertion-specific second amplification in the presence of a positive (ID/II) control (Shanmugam et al. 1993).

Determination of individual muscle properties

Experiments started with a non-invasive determination of anthropometrical characteristics which could be measured with a tape measure (Tables 1, 2). After a short warm up of leg musculature, the unshod subjects sat down on a leg-press with a slope of 21° (Tetra GmbH, Ilmenau, Germany, Fig. 1). To determine individual muscle properties, four contractions were carried out, two dynamic contractions with a supplementary load (m = 65 kg) followed by two isometric contractions. Initial knee angle was fixed at about 90°. The subjects were asked to perform contractions with maximum voluntary effort. The leg-extension was measured with an incremental encoder (ASM, Unterhaching, Germany, 9.9914 pulses/mm). Simultaneously, external forces were measured with two separate self-produced force-platforms (range 0–2,000 N, frequency response 500 Hz, capture rate 1,000 Hz).

Model calculations

The model consisted of two massless segments (thigh and shank) and a geometric model G describing the gearing of muscle force f_m into external force F beneath the feet (1).

$$F = G \times f_m \tag{1}$$

We wrote the geometric model as $G(X) = \frac{r \times \sin\beta}{l_0 \times l_u \sin\alpha} X \text{with} X = \sqrt{l_0^2 + l_u^2 - 2 l_0 l_u \cos\alpha}$ and $\alpha = 2\beta + \arcsin\left(\frac{r}{k_0}\sin\beta\right) + \arcsin\left(\frac{r}{k_u}\sin\beta\right)$

Muscle forces were described as a product of muscle activation $A(t)$ and Hill-type force–velocity relation f_H (2).

$$f_m = A \times f_H \tag{2}$$

An activation function described the level of muscular activation and its values were limited within an interval [0,1]. For the special case of muscle contractions under maximum voluntary effort (Sust et al. 1997a), the activation could be described by a simple exponential function (3).

$$A = 1 - e^{-S \cdot (t - t_0)} \tag{3}$$

with S—activation parameter, t_0—starting of activation.

The Hill-type force–velocity relation could be described as follows (4):

$$f_H = \frac{c}{v_m + b} - a \tag{4}$$

with v_m—muscle contraction velocity and a, b, c constants describing the shape of the force–velocity relation. Substituting $c = (f_{iso} + a)b$, we obtain another common expression of Hill's equation (Kohler and Boutellier 2005): $f_H + a = ((f_{iso} + a)\, b)/(v + b)$.

These properties could be transferred into physiological parameters f_{iso}—maximum isometric force, v_{max}—maximum contraction velocity and p_{max}—maximum power output, and vice versa. Maximum power

Table 1 Anthropometric characteristics

Symbols	Definitions
l_0	Length of the thigh: distance between Articulatio genus and Trochanter major
l_u	Length of the shank: distance between Articulatio genus and Malleolus fibularis
k_0	Extensor muscle length from Spina illiaca ant. inf. to mid-patella
k_u	Length of the patella tendon from mid-patella to Tuberositas tibiae
r	Distance between the centre of rotation and mid-patella girth of the shank around mid-patella divided by 2π
α	Flexion angle of the leg
β	Angle between patella tendon and r

Symbols and definitions for the geometric model according to Sust (1996)

Table 2 Distribution of genotypes (II, ID and DD) for male, female and all subjects

	II	ID	DD
Males (29)	7 (24.1%)	10 (34.5%)	12 (41.4%)
Females (33)	14 (42.4%)	6 (18.2%)	13 (39.4%)
All (62)	21 (33.9%)	16 (25.8%)	25 (40.3%)

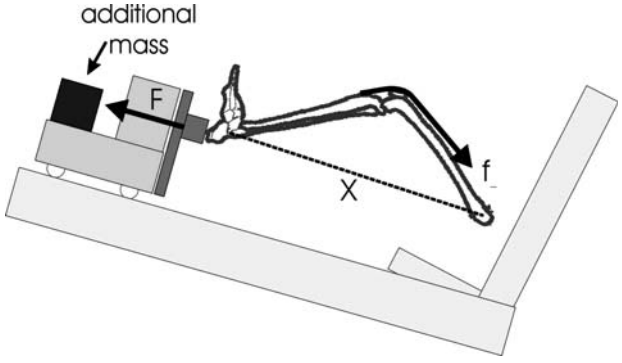

Fig. 1 Schematic representation of the leg-press. A mass is fixed on a rolling car, which can be pushed up a slope. The forces F exerted versus the rolling car can be determined by force-plates. The leg-extension X is measured by an incremental device simultaneously. The muscle force f_m can be calculated from the measured force F and the geometric function $G(X)$, depending on the leg-extension X. For isometric contraction the rolling car can be bolt together with the slope device

p_{max} was denoted by optimum force f_{opt} and optimum contraction velocity v_{opt}, resulting in $p_{max} = f_{opt} \times v_{opt}$. A more detailed discussion of the connections between Hill's properties and other physiological parameters, i.e. maximum efficiency, endurance and fibre composition, was given in Thaller and Wagner (2004).

As it was not possible to measure individual muscle properties of humans directly, they were determined with a non-linear regression method using experimentally measured concentric and isometric contractions with maximum voluntary effort. A modified Levenberg–Marquardt algorithm embedded in a software package (JOP Kinematics) altered a set of muscle properties in a step-wise fashion to minimize square deviation between measured and simulated forces (Weiss et al. 1995; Sust et al. 1997a, b; Schimmel et al. 2001; Wagner and Blickhan 2003). The results of this method were individual muscle properties of subjects. Initial position of the leg did not play a role while using anthropometric data of subjects to transform external forces and velocities into internal muscle forces and velocities (1).

Mean values and standard deviations of muscle parameters were calculated for male and female subjects (Tables 3, 4). Searching for an association between muscle properties in male and female subjects and their ACE genotypes, we first calculated mean values and standard deviations of muscle properties of single genotypes. Furthermore, we tested the differences using t tests and variance analysis (SPSS).

Discriminant analysis

We calculated mean values and standard deviations of determined and measured subject-specific parameters, and we performed a discriminant analysis (software SPSS). Discriminant analysis is a linear method and so it regards only linear combinations of parameters. The above-mentioned physiological quantities f_{iso}, v_{max} and p_{max}, however, are non-linear combinations of Hill constants a, b and c (4). So it seemed reasonable to use other non-linear combinations of experimentally determined values as input variables for an analysis. To put all information of muscle properties into a discriminant analysis, we tried to find as many non-linear combinations as possible, which were not, or only slightly, correlated with each other. Some of them had an obvious physiological meaning; others just reflect non-linear influences of basic properties.

We calculated a parameter $e = p_{max}/c$ which represents the efficiency e of a muscle, since Hill's parameter c describes the maximum physiological energy consumption and p_{max} the maximum mechanical power output (Thaller and Wagner 2004).

Furthermore, we used normalized parameters $a_n = a/A_c$, $b_n = b/l_0$ and $c_n = c/(A_c\, l_0)$, where l_0 denoted the length of thigh. The cross-sectional area A_c was calculated as the square of the girth of the thigh divided by 4π. Parameter b_n correlated with the area ratio of fast twitch fibres in muscles (Sust et al. 1997b). Analogously, normalized quantities f_{ison}, v_{maxn} and p_{maxn} were defined. Furthermore, we calculated a value $b_n S/v_{max}$, which, for simplicity, was denoted by T:

$$T = \frac{b_n \times S}{v_{max}} \tag{5}$$

T is a combination of all velocity-related parameters, normed to the muscle length.

To classify subjects into three genotypes by a discriminant analysis, two discriminant functions were determined (software SPSS) in such a way that coordinates in the discriminant space spanned by these functions separated the genotypes II, ID and DD. By evaluating the canonical discriminant functions for each subject, individual coordinates in discriminant space were calculated. The significance of the discri-

Table 3 Distribution of muscle-skeletal properties of all male subjects and regarding the ACE genotype

Male								
	All		II		ID		DD	
	Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD
f_{iso} (N)	7,434	1,874	7,393	2,789	6,826	1,263	7,965	1,658
v_{max} (m/s)	2.99	1.18	2.46	0.72	3.43	1.56	2.93	0.96
p_{max} (W)	1,361	312	1,343	353	1,300	340	1,422	277
a (N)	1,230	847	1,568	951	864	371	1,339	1,006
b (m/s)	0.43	0.19	0.51	0.26	0.39	0.13	0.41	0.18
c (W)	3,697	1,836	4,375	2,107	2,973	1,095	3,904	2,089
S (1/s)	10.89	3.00	11.270	4.269	10.334	3.017	11.135	2.266
e	0.42	0.126	0.362	0.152	0.455	0.086	0.423	0.134
T (1/ms)	4.35	4.30	7.274	7.481	2.807	1.555	3.923	2.565
Height (cm)	181	5	179	6.5	178	3.5	184	4.9
Mass (kg)	76.1	9.6	73.47	8.86	72.34	8.47	80.91	9.58

The values are given as mean and standard deviation SD within the groups

Table 4 Distribution of muscle-skeletal properties of all female subjects and regarding the ACE genotype

Female								
	All		II		ID		DD	
	Mean	±SD	Mean	±SD	Mean	±SD	Mean	±SD
f_{iso} (N)	4,405	921	4,481	955	3,772	564	4,614	941
v_{max} (m/s)	2.66	1.04	2.68	0.97	2.25	0.68	2.83	1.25
p_{max} (W)	805	179	812.5	166.7	667.8	100.3	861.2	194.5
A (N)	822	427	822.5	455.2	827.4	425.1	818.0	433.0
b (m/s)	0.44	0.18	0.46	0.26	0.44	0.09	0.42	0.09
c (W)	2,290	928	2,412.5	1,273.5	2,018.1	558.2	2,284.5	595.6
S (1/s)	10.42	5.22	11.58	7.75	8.7	0.97	9.94	1.95
e	0.387	0.118	0.39	0.13	0.35	0.10	0.40	0.12
T (1/ms)	5.62	7.68	7.17	11.49	4.60	1.99	4.42	2.76
Height (cm)	170	7	170.5	5.6	164.3	4.1	172.5	7.5
Mass (kg)	60.2	7.7	61.38	5.78	51.00	4.18	63.12	7.79

The values are given as mean and standard deviation SD within the groups

minant functions separating the subjects were calculated via Wilks' Lambda and the importance of the parameters for discriminant functions was computed via pooled-within-groups correlations between discriminating variables and canonical discriminant functions (Bortz 1999).

Results

Distribution of genotypes

Among our cohort of sport students from mixed sporting disciplines, we found a strong preference of II and DD types (Table 2). This result differed significantly ($P < 0.01$) from Hardy–Weinberg distribution (1:2:1) and from experimentally found distributions (Taylor et al. 1999; Woods et al. 2002), but it had no influence on an analysis of a correlation between individual muscle properties and genotype. For female subjects a preference of II and DD was even stronger, but I and D alleles were of same frequency.

Muscle properties

Individual muscle properties were determined for each subject from experimental leg-press data (Fig. 2). In general, we found no significant differences in a single muscle property between the three genotypes (Tables 3, 4). However, there were significant ($P < 0.05$) differences in mean values and standard deviations of certain muscle properties between one genotype and two others. For female subjects, mean value of isometric force f_{iso} differs between II and ID,

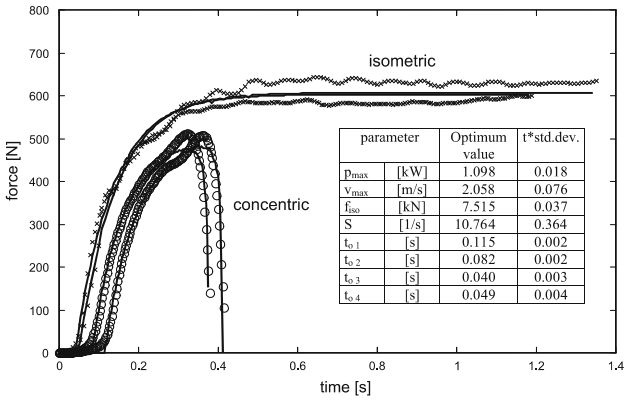

Fig. 2 A non-linear fit between the measured data (*circles and crosses*) and the model properties (*solid lines*) of subject #3 as an example. The numerical result is shown in the *table inserted*. The value $t \times$ SD represented the product of a t test and the standard deviation for the parameter and it should be below 5% of the optimum value

and ID and DD, but not between II and DD. For male subjects, there was only a significant difference between ID and DD. A difference in mean value and standard deviation of T was significant between II and ID and II and DD for male and female subjects. Maximum power output p_{max} differed only for women between ID and the other types, but not for men.

In summary, the results indicated that genotypes did not differ in a single muscle property but in a combination of properties.

Discriminant analysis

A step-wise discriminant analysis was performed to determine which combination of muscle properties separates subjects into three genotypes. As basic properties, we used isometric force f_{iso}, maximum contraction velocity v_{max}, maximum power output p_{max} and a parameter of activation S. For normalization we used length l_0 and cross section A_c of the thigh. All parameters mentioned below are combinations of these basic properties.

Male subjects

We found a significant connection between local muscle properties and ACE genotype ($P = 0.016$, Wilks' Lambda = 0.159). A discriminant analysis of male subjects correctly classified 89.7% regarding their genotype. ACE genotypes were best separated by 11 variables: maximum contraction velocity v_{max}, quantity T as defined in formula (5), normalized Hill's parameter c_n, normalized maximum power output p_{maxn}, parameter of activation S, and terms ($Sf_{iso}\ v_{max}\ p_{max}$), ($Sv_{max}$), ($abc$), ($v_{max}/b_n$), ($f_{iso}\ v_{max}\ p_{max}$) and ($c/b$). All s subjects with genotype II were separated from subjects of genotype ID and DD (Table 5). A separation of ACE genotypes could be illustrated in a discriminant space (Fig. 3). Evaluating canonical discriminant functions for each subject resulted in coordinates in the discriminant space. The position of the group centroid in the discriminant space was (–2.798, –0.033) for II, (0.938, –0.960) for ID and (0.851, 0.819) for DD.

The importance of each variable for the two discriminant functions was calculated via pooled-within-groups correlations between discriminating variables and canonical discriminant functions. Function 1 was mainly influenced by the values T, v_{max} and c_n and function 2 by c/b, abc, v_{max}. So function 1 was mainly described by the muscle properties T, v_{max} and c_n, and separates subjects with II genotypes from the two other groups (Fig. 3). Function 2 was mainly determined by c/b, abc, v_{max}, and separated ID and DD. However, separation by function 2 was not significant ($P = 0.38$).

Female subjects

For female subjects we again found a significant connection between local muscle properties and ACE genotype (Wilks' Lambda = 0.299, $P = 0.026$). A discriminant analysis leads to a correct classification of 87.9% of the subjects based on the following nine variables:

$$(p_{max}/f_{iso}), (p_{max}/v_{max}), c_n, f_{ison}, v_{max\,n}, p_{max\,n}, v_{opt}, f_{opt}, (v_{opt}/v_{max}).$$

All subjects of genotype DD were separated from subjects of genotype ID and II (Table 6).

The position of subjects in the discriminant space showed (Fig. 4) that II and DD types were separated by function 1, and function 2 separated ID from the other groups, but not significantly ($P = 0.31$).

Table 5 Results of the discriminant analysis (11 variables) of male subjects, e.g. every measured II-subject was predicted as II by the discriminant analysis, whereas two measured ID-subjects were predicted as DD

Measured	Predicted		
	II	ID	DD
II	7	0	0
ID	0	8	2
DD	0	1	11

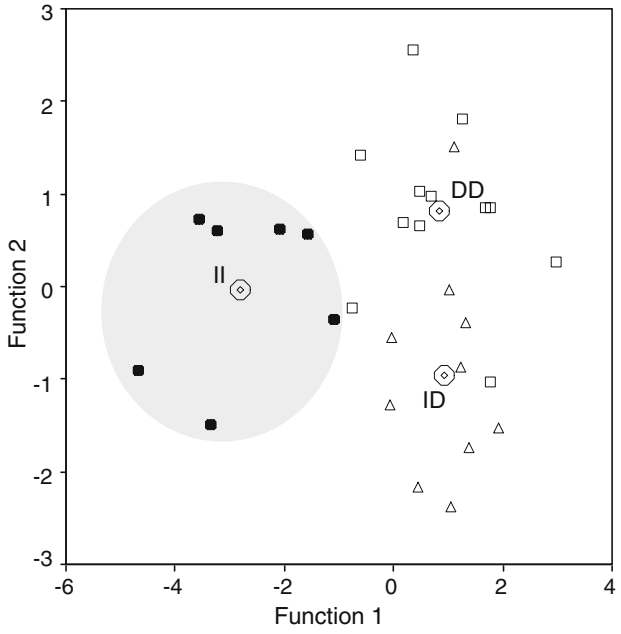

Fig. 3 Canonical discriminant function showing the position of 29 male subjects and the group centroids (*open circles*) in the discriminant space. Probands with genotype II were completely separated from subjects of genotypes ID and DD; *filled circles* genotype II, *rectangles* genotype DD, *triangles* genotype ID

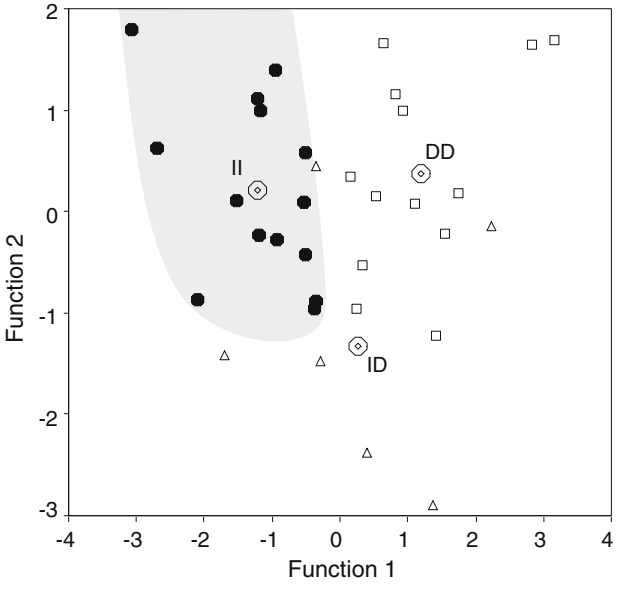

Fig. 4 Canonical discriminant function showing the position of 33 female subjects and the group centroids (*open circles*) in the discriminant space. Probands with genotype II were completely separated from the subjects of genotype ID and DD; *filled circles* genotype II, *rectangles* genotype DD, *triangles* genotype ID

Discussion

The purpose of this study was to separate the subjects of our cohort into three different ACE genotypes based on their individual local biomechanical muscle properties. Previous studies have shown an association of the ACE I/D gene polymorphism with physical performance. The I allele has been reported to be linked to enhanced endurance performance (Gayagay et al. 1998; Myerson et al. 1999; Alvarez et al. 2000) and response to physical training (Montgomery et al. 1998), whereas the D allele is present with an excess frequency among sprinting disciplines (Myerson et al. 1999) suggesting a possible effect of the D allele on muscle strength and power.

The results of the present study supported these findings. In this study we showed a significant connection between the ACE I/D gene polymorphism and individual biomechanical muscle properties ($P < 0.03$). With a combination of certain individual muscle parameters, we were able to assign 89% of the subjects to their individual ACE genotypes (II, ID, DD).

Table 6 Results of the discriminant analysis (nine variables) of female subjects, e.g. every measured II-subject was predicted as II by the discriminant analysis, whereas one measured DD-subjects were predicted as ID

Measured	Predicted		
	II	ID	DD
II	14	0	0
ID	2	3	1
DD	0	1	12

It was not a single individual muscle parameter but a certain combination of biomechanical muscle properties, which allowed a separation of our cohort into appropriate genotypes. Interestingly, it was impossible to find the same combination set of muscle properties for both women and men. For men, the most important variables were the maximum contraction velocity v_{max}, normalized Hill's parameter c_n and T described by $b_n S/v_{max}$. For females the normalized Hill's parameter c_n, normalized isometric force f_{ison} and optimum contraction velocity v_{opt} were important parameters. Female subjects of II genotype had higher c_n values, but they produced less isometric force and had a lower optimum velocity. Male II subjects as well had higher c_n values and higher values of T and lower maximum contraction velocities.

From this we may assume that subjects of II genotype showed lower maximum and optimum velocity. Previous experiments determining force–velocity relations of muscle fibres have shown that the maximum contraction velocity was affected by muscle length and fibre-type (Herzog 1994), and that slow-twitch fibres were linked to lower maximum and optimum contraction velocities and lower maximum isometric forces

(Barclay 1996). Hence, the lower maximum and optimum velocity values for male and female II subjects found in this study indicate that they may have higher ratios of cross-sectional areas of slow twitch versus fast twitch fibres. Such improvements may stem from increases in oxygen delivery, including cardiac output and muscle capillary density, conversion of type 2 fibres to slow-contracting type 1 fibres of high oxidative capacity, increased mitochondrial numbers and density, and raised myoglobin content of muscle. The association of increased body-fat stores with improved physical performance might suggest an effect of ACE genotype on energy balance, and on the nature and efficiency of use of oxidative fuel for metabolism (Montgomery et al. 1999; Sudi et al. 2001). All these physiological effects may influence local properties of muscles.

It is obvious that the subjects of the present population are members of different sporting groups, and they were not in Hardy–Weinberg Distribution, with a very low number of ACE ID genotypes. We calculated the significance of the discriminant functions separating the subjects via Wilks' Lambda. The results were significant, indicating that the sample size and their distribution according to the genotypes within the groups were enough. To reduce the influence of typical trainings on the statistical analysis, the subjects were not selected according to special sports, e.g. sprinters and endurance athletes.

We have to consider the validity of the methods. To our knowledge, no method exists to determine individual muscle properties of humans with non-invasive measurements. In the present study individual muscle properties were measured based on a non-linear regression method, which was restricted to maximum voluntary contractions. This method had been validated on single muscle experiments with rats, cuis and frogs (Siebert et al. 2003; Wagner and Blickhan 2003) and was used several times in human experiments (Sust et al. 1997a, b; Wagner and Blickhan 2003). The method to determine the ACE genotype is well established as well.

It should be clear that a genetic factor can be only *one* parameter beside metabolic, physiological, psychological and environmental factors influencing physical performance and muscle properties, and that the genetic component consists of a polygenic action of several genomic loci.

We conclude from our results that ACE-dependent local muscle effects discussed before are reflected in a correlation between biomechanical muscle properties and ACE genotype. Or, in other words, it is the individual set of biomechanical muscle properties influenced by ACE genotype that contributes to athletic performance.

Acknowledgment We would like to thank Prof. Dr R. Blickhan for the opportunity to perform the measurements at the Institute of Sport Science, Friedrich Schiller University of Jena.

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