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Association between the *ACE* I/D polymorphism and physical activity in Polish women

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Abstract

Angiotensin converting enzyme gene (*ACE*) is the most frequently investigated genetic marker in the context of genetic conditioning of athletic predispositions. However, the knowledge of ACE's potential modifying effect on changes in selected body traits achieved through a training programme is still limited. Therefore, we have decided to check whether selected body mass, body composition variables, oxygen uptake parameters as well as strength/speed parameters observed in physically active participants will be modulated by the *ACE* I/D polymorphism. The genotype distribution was examined in a group of 201 young healthy women measured for chosen traits before and after the completion of a 12-week moderate-intensive aerobic training programme. Our results revealed the significant genotype × training interactions for VEmax and power of countermovement jump, whereas training improvements were demonstrated for almost all parameters. In addition, main effects of the *ACE* I/D genotype on TGL, HDL, glucose and 10 m run were observed. A significant increase in VEmax was demonstrated for II and DD genotypes, but not for ID heterozygotes. The greatest gain in power of countermovement jump was observed in II homozygotes, although DD and ID were associated with a significant increase as well. Our study indicated that the polymorphism was associated with changes in VEmax and power of countermovement jump in response to a 12-week aerobic training programme in Caucasian women. However, more experimental studies are needed to establish the ACE gene × physical activity interactions.

KEYWORDS: *ACE* I/D polymorphism, gene × physical activity interaction, training programme.

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Introduction

Decades of physiological research in sport have resulted in gaining relatively considerable knowledge of the functional response of the human body to physical activities. Regular exercise brings about a variety of metabolic and morphological changes including mitochondrial biogenesis, muscle fiber-type transformation, substrate metabolism, and improvements in the circulatory system [1]. Although the physiological reactions in the human body

following regular exercise are quite well described, the genetic background of the reactions still remains mostly unknown. An understanding of the genetic determinants will allow us to clarify the criteria of physical activities for individuals [2]. In the future, this knowledge should help to identify individuals who are expected to respond well or poorly to exercise, thus making training programs much more efficient (possibility of accurate prediction of the training results including weight loss and improve health) and safer (early prevention of possible overload, injuries, cardiomyopathies, sudden death etc.) [3, 4]. It might be even used to predict the best sport-specific genetic background [5].

The most frequently investigated genetic markers in the context of athletic predisposition is the angiotensin converting enzyme gene (*ACE*) I/D polymorphism. Variants in this genes have been reported to be associated with elite athletic performance and with normal, quantitative physical performance traits in the general, even if this association has been often conflicting [6]. The product of *ACE* is one of the key components of the renin angiotensin system (RAS) which catalyses production of angiotensin II (ANG II) from angiotensin I (ANG I), resultantly increasing blood pressure. Furthermore, the enzyme is the essential part of the kallikrein-kinin system (KKS) where it degrades kinins into inactive fragments, thus reducing blood pressure. Moreover, the *ACE* is expressed in skeletal muscles, where it influences its function and biomechanical properties [7, 8, 9]. The human *ACE* gene is located on chromosome 17 in position 17q23.3 with a restriction fragment length polymorphism consisting of the presence (insertion, I) or absence (deletion, D) of a 287 base pair Alu repeat sequence in intron 16 [10]. In this case, three *ACE* genotypes include DD, II homozygotes, and ID heterozygotes [11]. Studies concerning links between the *ACE* genotype and athlete status have shown that the I allele is associated with lower ACE activity in both serum and tissue and improved performance in endurance sports [12]. Meanwhile, the D allele is associated with greater circulating and the ACE activity and causes an enhanced performance at sports requiring sprinting or short bursts of power [13, 14].

Considering the above-mentioned facts, we studied the possible impact of *ACE* I/D variant on the aerobic and anaerobic performance as well as body and mass composition measurements in physically active participants. Therefore, we examined the allele and genotype distribution in young Polish women measured for selected traits before and after the completion of a 12-week training programme to see whether there was an interaction between genotype and training.

Materials and Methods

Ethics Statement

All the procedures followed in the study were approved by the Ethics Committee of the Regional Medical Chamber in Szczecin (Approval number 09/KB/IV/2011) and were conducted ethically according to the principles of the World Medical Association Declaration of Helsinki and ethical standards in sport and exercise science research. Furthermore, the experimental procedures were conducted in accordance with the set of guiding principles for reporting the results of genetic association studies defined by the Strengthening the Reporting of Genetic Association studies statement (STREGA). All participants filled in a consent form and a written information sheet concerning the study, providing all pertinent information (purpose, procedures, risks, and benefits of participation).

Participants

201 Polish Caucasian women aged 19-24 years (21 ± 1) years met the inclusion criteria and were included in the study. None of these individuals had engaged in regular physical activity in the previous 6 months. They had no history of any metabolic or cardiovascular diseases. Participants were nonsmokers and refrained from taking any medications or supplements known to affect metabolism. Prior to the start of the training phase, participants were asked to maintain a balanced diet of approximately 2000 kilocalories a day.

Physical exercise training protocol

The training stage was preceded by a week-long familiarization stage, during which the examined women exercised 3 times a week for 30 minutes, at an intensity of about 50% of their maximum heart rate (HRmax). After the week-long familiarization stage, the proper training started. Each training unit consisted of a warm-up routine (10 minutes), the main aerobic routine (43 minutes), and a cool-down phase (stretching and breathing exercise for 7 minutes). The main aerobic routine was a combination of two alternating styles – low and high impact. The low impact style comprised of movements with at least one foot on the floor at all times, whereas the high impact style included running, hopping, and jumping with a variety of flight phases. Music of variable rhythm intensity (tempo) was incorporated into both styles. A 12-week program of low-high impact aerobics was divided as follows: (i) 3 weeks (9 training units), 60 minutes each, at about 50–60% of HRmax, 135–140 BPM; (ii) 3 weeks (9 training units), 60 minutes each, at 60–70% of HRmax,

140–152 BPM; (iii) 3 weeks (9 training units), 60 minutes with the intensity of 65%−75% of HRmax, 145–158 BPM; and (iv) 3 weeks (9 training units), 60 minutes with an intensity of 65%−80% of HRmax, 145–160 BPM. All 36 training units were administered and supervised by the same instructor.

Body Composition Measurements

All participants were measured for selected body mass and body composition variables before and after the completion of a 12-week training period. Body mass and body composition were assessed with the bioimpedance method (body's inherent resistance to an electrical current) using a "Tanita TBF 300M" electronic scale (Horton Health Initiatives, USA). The device was plugged in and calibrated to account for the weight of clothing (0.2 kg). Afterwards, data regarding a participant's age, body height, and sex were entered. Then, the subjects stood on the scale with their bare feet on the marked places. The device analyzes body composition based on the differences of the ability to conduct electrical current by body tissues (different resistance) due to different water content. Body mass and body composition measurements taken with the use of the "Tanita" electronic scale are as follows: total body mass (kg), fat free mass (FFM, kg), fat mass (kg), fat mass percentage (FM, %), body mass index (BMI, kg/m^2), tissue impedance (Ohm), total body water (TBW, kg), and basal metabolic rate (BMR, kJ or kcal).

Aerobic capacity test (VO_2max)

Participants performed a continuous graded exercise test on an electronically braked cycle ergometer (Oxycon Pro, Erich JAEGER GmbH, Hoechberg, Germany) to determine their VO_2max, HRmax, VEmax, Pmax before and after the completion of a 12-week training period. The test began with 5 minutes of continuous pedaling, with a frequency of 60 revolutions per minute (RPM) and a relative load of 1.2 W·kg^{-1}. After this phase, the workload was systematically increased by 15 watts every minute until exhaustion. The effort was interrupted when pedalling frequency declined by 10%, that is, when the pedalling frequency fell below 54 RPM. The highest value of the oxygen uptake, heart rate, minute ventilation and power output maintained for 15 seconds was considered to be the VO_2max, HRmax, VEmax, Pmax. The anaerobic threshold values were obtained using the V-slope method.

The Wingate anaerobic test

A 30-s Wingate test on a cycle ergometer (Monark Ergomedic 894 E, Monark, Sweden) was used to assess the peak power and total work before and after the completion of a 12-week training period. A relative load corresponding to 7.5% of the subject's body mass was applied [15]. Before performing the test, the participants completed a 10-min warm-up, including pedaling at a frequency of 60 rotations per minute (RPM), with a relative load of 1.2 W·kg^{-1} and three rapid accelerations between the 7th and 10th minute [16, 17]. After the warm-up, the subjects performed five minutes of stretching and relaxing exercises and then started the test.

Biochemical and Hematological Analyses

Fasting blood samples were obtained in the morning from the elbow vein. Blood samples from each participant were collected in two tubes. For biochemical analyses, a 4.9 mL S-Monovette tube with ethylenediaminetetraacetic acid (K 3 EDTA; 1.6 mg EDTA/mL blood) and separating gel (SARSTEDT AG & Co., Nümbrecht, Germany) were used. For complete blood count, a 2.6 mL S-Monovette tube with K 3 EDTA (1.6 mg EDTA/mL blood) (SARSTEDT AG & Co., Nümbrecht, Germany) was used. Blood samples for biochemical analyses were centrifuged 300 × g for 15 minutes at room temperature in order to receive blood plasma. Biochemical and hematological analyses were performed before the start of the aerobic fitness training programme and repeated at the 12th week of this training programme (after the 36th training unit). The analyses were performed immediately after the blood collection. Complete blood count, including white blood cells (WBC), red blood cells (RBC), haemoglobin (HGB), haematocrit (HTC), mean corpuscular volume (MCV), mean corpuscular haemoglobin (MCH), mean corpuscular haemoglobin concentration (MCHC), and total platelet level (PLT) were obtained using Sysmex K-4500 Haematology Analyzer (TOA SYSMEX, Kobe, Japan). All biochemical analyses were conducted using the Random Access Automatic Biochemical Analyzer for Clinical Chemistry and Turbidimetry A15 (BIO-SYSTEMS S.A., Barcelona, Spain). Blood plasma was used to determine lipid profile: triglycerides (Tg), cholesterol (Chol), high-density lipoprotein (HDL) and low-density lipoprotein (LDL) concentrations. Plasma Tg and Chol concentrations were determined using diagnostic colorimetric enzymatic method according to the manufacturer's protocol (BioMaxima S.A., Lublin, Poland). Manufacturer's declared intra-assay coefficients of variation (CV) of the method were < 2.5% and < 1.5% for the Tg and Chol determinations, respectively. HDL plasma concentration was determined using human

anti-ß-lipoprotein antibody and colorimetric enzymatic method according to the manufacturer's protocol (BioMaxima S.A., Lublin, Poland). The manufacturer's declared intra-assay CV of the method was < 1.5%. Plasma concentrations of LDL were determined using a direct method according to the manufacturer's protocol (PZ Cormay S.A., Lomianki, Poland). The manufacturer's declared intra-assay CV of the method was 4.97%. All analysis procedures were verified with the use of multiparameteric control serum (BIOLABO S.A.S, Maizy, France), as well as control serum of normal level (BioNormL) and high level (BioPathL) lipid profiles (BioMaxima S.A., Lublin, Poland).

Genetic Analyses

Genomic DNA was extracted from the buccal cells using a GenElute Mammalian Genomic DNA Miniprep Kit (Sigma, Germany), according to the manufacturer's protocol. PCR amplification of the polymorphic region of the *ACE* gene containing either the insertion (I) or deletion (D) fragment was performed. One pair of primers (forward: CTG GAG ACC ACT CCC ATC CTT TCT and reverse: GAT GTG GCC ATC ACA TTC GTC AGA) was used to determine the *ACE* genotype, yielding amplification products of approximately 490 bp (for the I allele) and 190 bp (for the D allele), as described by Rigat et al. [12]. PCR mixture and thermal--time profile were coequal as earlier described [18]. The amplified DNA fragments were visualized by using 1.5% agarose gels stained with ethidium bromide.

Statistical Analysis

Allele frequencies were determined by gene counting. A chi-square test was used to test the Hardy-Weinberg equilibrium. Normality was evaluated by Kolmogorov-Smirnov test as well as the visual inspection of the histograms. As there were between-subject (ACE genotype) and within-subject factors (training status), the 2 × 2 mixed-design ANOVA with repeated measures was used to test the influence of the *ACE* I/D polymorphism on training response. The post hoc Fisher's least significant difference (LSD) test was used. The level of statistical significance was set at $p < 0.05$.

Results

The *ACE* I/D genotype frequencies conformed to Hardy-Weinberg equilibrium (Chi-square 1.96, p = 0.162). The training adaptation of body mass, body composition variables, oxygen uptake parameters as well as strength/speed parameters with respect to I/D *ACE* polymorphism was evaluated with two-way factorial ANOVA with repeated measures. We found the genotype × training

Table 1. *ACE* genotypes and body mass and body composition variables before and after training (two-way mixed ANOVA)

Parameter	II (n = 42)		ID (n = 65)		DD (n = 40)		Genotype	Training	Genotype × training
	Before training	After training	Before training	After training	Before training	After training			
Body mass (kg)	61.3±7.9	60.3±7.5	59.7±7.2	59.2±7.2	62.0±7.4	61.2±7.7	p=0.334	p<0.0001*	0.274
BMI ($kg \times m^{-2}$)	21.8±2.6	21.4±2.5	21.5±2.0	31.4±2.1	22.0±2.8	21.8±2.7	p=0.638	p<0.0001*	0.203
BMR (kJ)	6087±333	6020±298	6018±312	5991±313	6111±307	6082±309	p=0.320	p<0.0001*	0.194
FM (%)	23.9±5.6	22.4±5.0	23.9±5.2	22.6±5.7	24.8±5.6	23.1±6.4	p=0.762	p<0.0001*	0.739
FM (kg)	14.9±5.4	14.0±5.1	14.6±4.8	13.7±5.0	15.7±5.1	14.5±5.5	p=0.621	p<0.0001*	0.708
FFM (kg)	46.2±2.8	46.6±3.1	45.0±3.0	45.5±3.0	46.3±3.3	46.8±3.3	p=0.057	p<0.0001*	0.906
TBW (kg)	33.8±2.1	34.1±2.3	33.0±2.8	33.4±2.3	33.9±2.5	34.3±2.5	p=0.088	0.005*	0.963
Chol (mg/dl)	166.8±24.4	166.6±24.6	171.0±25.0	169.3±27.3	171.4±25.7	172.5±30.2	p=0.605	0.902	0.799
TGL (mg/dl)	75.0±22.4	76.8±22.8	86.1±32.0	88.9±44.2	73.1±25.8	79.6±24.0	p=0.041*	0.178	0.803
HDL (mg/dl)	63.2±14.9	62.2±11.3	62.4±12.0	57.9±12.7	69.4±11.6	64.5±12.5	p=0.013*	0.0003*	0.191
LDL (mg/dl)	88.4±20.6	89.4±22.1	91.0±20.6	93.4±22.8	87.2±21.6	91.9±25.7	p=0.658	0.079	0.648
Glucose (mg/dl)	78.7±9.0	73.7±10.1	75.0±8.9	73.4±9.1	79.5±12.0	79.0±11.7	0.013*	0.009*	0.130

Mean ± standard deviation; p – values for main effects (genotype and training) and genotype × training interactions; BMI – body mass index; BMR – basal metabolic rate; FM – fat mass percentage; FFM – fat free mass; TBW – total body water; Chol – cholesterol; TGL – triglycerides; HDL – high-density lipoprotein; LDL – low-density lipoprotein

Table 2. *ACE* genotypes and maximal oxygen uptake test parameters before and after training (two-way mixed ANOVA)

Parameter	II (n = 42)		ID (n = 65)		DD (n = 40)		Genotype	Training	Genotype × training
	Before training	After training	Before training	After training	Before training	After training			
VO_2max (ml/kg/min)	34.18±5.1	38.30±6.0	33.37±5.2	36.68±6.3	33.34±4.6	36.03±5.1	0.311	p<0.0001*	0.412
VO_2/AT (ml/kg/min)	24.73±4.1	28.28±4.7	25.12±4.4	27.39±5.4	23.89±4.4	26.97±4.0	0.392	p<0.0001*	0.457
VO_2AT/ /VO_2 max (%)	0.73±0.10	0.74±0.08	0.75±0.08	0.75±0.07	0.72±0.09	0.75±0.08	0.393	p=0.108	0.130
HRmax (beats/min)	184.57±8.4	186.88±7.9	187.78±10.5	186.91±8.9	186.8±7.9	185.68±8.0	0.553	p=0.881	0.088
HR/AT (beats/min)	162.29±11.8	165.10±9.8	165.40±13.6	167.34±11.0	162.43±13.5	164.03±10.3	0.254	0.021	0.869
HR_{AT}/HRmax (%)	0.88±0.05	0.88±0.04	0.88±0.05	0.90±0.04	0.87±0.06	0.88±0.05	0.352	0.010	0.612
VEmax (l/min)	71.43±18.0	83.36±19.6	70.27±18.1	73.92±18.1	71.31±15.5	78.05±15.5	0.250	p<0.0001*	0.017*
VE/AT (l/min)	38.52±8.2	46.42±11.0	37.93±8.9	42.22±11.6	38.30±9.2	43.22±7.7	0.341	p<0.0001*	0.178
VE_{AT}/VEmax (%)	0.56±0.1	0.57±0.1	0.55±0.1	0.58±0.1	0.55±0.1	0.57±0.1	0.841	0.045	0.907

Values of the parameters of both groups before and after training are means (± SD); *p – value 0.05; VO_2max – maximum oxygen uptake; VO_2/AT – oxygen uptake at anaerobic threshold; VO_2AT/VO_2max – percentage of VO_2max at anaerobic threshold; HRmax – maximum heart rate; HR/AT – heart rate at anaerobic threshold; HR_{AT}/HRmax – percentage of HRmax at anaerobic threshold; VEmax – maximum minute ventilation; VE/AT – minute ventilation at anaerobic threshold; VE_{AT}/VEmax – percentage of VEmax at anaerobic threshold

Table 3. *ACE* genotypes and Wingate test parameters before and after training (two-way mixed ANOVA)

Parameter	II (n = 42)		ID (n = 65)		DD (n = 40)		Genotype	Training	Genotype × training
	Before training	After training	Before training	After training	Before training	After training			
Pmax (W/kg)	8.04±0.6	8.49±0.8	7.74±1.0	8.14±0.9	7.80±0.7	8.11±0.9	0.097	p<0.0001*	0.559
Tr (s)	6.41±2.1	5.60±2.2	6.85±2.1	5.77±2.0	6.38±2.1	5.30±2.1	0.418	p<0.0001*	0.754
Tm (s)	3.58±1.1	4.07±1.74	3.74±1.5	4.44±1.7	3.63±1.09	4.11±1.8	0.522	p<0.0001*	0.735

Values of the parameters of both groups before and after training are means (± SD); *p – value 0.05; Pmax – maximal power; Tr – time to reaching Pmax; Tm – time of maintaining Pmax

Table 4. *ACE* genotypes and speed parameters before and after training (two-way mixed ANOVA)

Parameter	II (n = 42)		ID (n = 65)		DD (n = 40)		Genotype	Training	Genotype × training
	Before training	After training	Before training	After training	Before training	After training			
Run 5 m	1.28±0.07	1.25±0.07	1.29±0.07	1.25±0.08	1.30±0.07	1.27±0.06	0.340	p<0.0001*	0.684
Run 10 m	2.21±0.11	2.14±0.10	2.21±0.14	2.15±0.12	2.25±0.09	2.21±0.10	0.031*	p<0.0001*	0.297
Run 30 m	5.26±0.28	5.22±0.34	5.29±0.32	5.25±0.34	5.40±0.23	5.31±0.23	0.243	p<0.0001*	0.642

Table 5. *ACE* genotypes and jump parameters before and after training (two-way mixed ANOVA)

Parameter	II (n = 42)		ID (n = 65)		DD (n = 40)		Genotype	Training	Genotype × training
	Before training	After training	Before training	After training	Before training	After training			
Jump with hands on hips (cm)	23.66±4.3	25.21±3.8	23.80±3.9	24.94±3.7	23.53±3.6	24.70±3.7	0.908	p<0.0001*	0.783
Power of jump – hands on hips (W)	32.84±5.2	35.88±4.4	33.80±4.3	35.57±4.4	32.20±4.9	35.22±3.1	0.398	p<0.0001*	0.353
Counter movement jump (cm)	27.99±5.0	30.82±4.6	27.31±5.9	29.75±4.0	27.28±4.9	29.77±4.6	0.567	p<0.0001*	0.880
Power of counter movement jump (W)	37.94±4.2	41.27±4.8	38.22±4.2	39.87±4.4	36.92±3.5	39.92±3.8	0.374	p<0.0001*	0.034*

interactions for VEmax ($F(2, 144) = 4.17$, $p = 0.017$) (Table 2) and power of countermovement jump ($F2, 144) = 3.47$, $p = 0.034$) (Table 5), whereas training improvements were demonstrated for almost all parameters, except blood lipids (Chol, TGL, LDL), VO_2AT/VO_2max, HRmax (Table 1-5). In addition, main effect of *ACE* I/D genotype on TGL, HDL, glucose and 10 m run were observed (Table 1, 4). A significant increase in VEmax was demonstrated for II (71.43 ± 18.0 vs 83.36 ± 19.6, $p = 0.0002$) and DD (71.31 ± 15.5 vs 78.05 ± 15.5, $p = 0.039$) genotypes, but not for ID heterozygotes (70.27 ± 18.1 vs 73.92 ± 18, $p = 0.323$) (Table 2). The greatest gain in power of countermovement jump was observed in II homozygotes (37.94 ± 4.2 vs 41.27 ± 4.8, $p = 0.0002$) although DD and ID were associated with a significant increase as well (36.92 ± 3.5 vs 39.92 ± 3.8 and 38.22 ± 4.2 vs 39.87 ± 4.4, respectively) (Table 5).

Discussion

The main objective of the proposed research was to determine the impact of a polymorphism of a selected gene on the characteristics and extent of the body's adaptive response to training. To detect a correlation between the *ACE* I/D genetic polymorphism and selected body composition measurements in participants undergoing training program we examined the genotype distribution of the *ACE* I/D allele in a group of 201 young Polish women measured for selected body mass, body composition variables, oxygen uptake parameters as well as strength/speed parameters before and after the completion of a 12-week training period. An important finding of this study was the significant genotype × training interactions for VEmax and power of countermovement jump, whereas training improvements were demonstrated for almost all parameters, except blood lipids (Chol, TGL, LDL), VO_2AT/VO_2max, and HRmax. In addition, main effects of *ACE* I/D genotype on TGL, HDL, glucose and 10 m run were observed. A significant increase in VEmax was demonstrated for II and DD genotypes, but not for ID heterozygotes. The greatest gain in power of countermovement jump was observed in II homozygotes, although DD and ID were associated with a significant increase as well.

The *ACE* gene is most often studied in the context of genetic determinants of a predisposition to sports, as the product of this gene (angiotensin converting enzyme) is considered to be a key element of the renin-angiotensin system (RAS) responsible for regulating blood pressure, one of the main factors determining the efficiency of the body [19]. Montgomery et al. [20] observed the increased effectiveness of implemented training (expressed in longer duration of exercise) in the British Army recruits with *ACE* II genotype, compared to recruits with DD genotype. The possibility of continuing exercise (arm bending with extra load – 15 kg medicine ball) were up to eleven times higher in men with genotype II compared to those with the DD genotype [20]. In another study, also on army recruits, Williams et al. [21] showed that the presence of *ACE* I allele may effectively enhance the efficiency of skeletal muscle after an 11-week aerobic training programme. Further research on a potential link between *ACE* gene II genotype and increased response parameters after training focused on patients suffering from cardiovascular and respiratory diseases. Defoor et al. [22] showed that the aerobic fitness of the body in response to training carried out in patients with coronary heart disease increased to a much greater extent in subjects with genotype II than in individuals having at least one D allele [22]. A similar conclusion was reached by Gosker et al. [23] in a study of people suffering from chronic lung disease. One of the few works of this type, based on research with athletes,

was Cam et al. (2007). In a study of 55 Turkish female athletes, after aerobic training, women with gene type II had significantly better results during the 30-minute run and displayed more favorable changes in physiological parameters than athletes under the same training regime but with the DD genotype [24]. The necessity of further research is not only connected with the hitherto very limited scope (only a few papers published) but also with ambiguous results. For example, Rankinen et al. [25] showed that much better gains in VO_2max in response to training were observed in individuals with DD homozygotes, not in those with II homozygotes, as indicated by the aforementioned papers [25].

One of the first papers to analyze the correlation between the insertion-deletion polymorphisms of the *ACE* gene and changes in the power/strength parameters of muscle contraction was Folland et al. [26]. After a 9-week long training schedule they observed higher increases in muscle strength in individuals with the D allele. Further studies included, for example, Giaccaglia et al. [27] who examined responses to bodybuilding training among the elderly (18 months long). In both groups, among men and women, greater increases in knee flexor muscle strength, were obtained by individuals with DD genotype compared with subjects with genotype II [27]. Cam et al. [24] (2007) examined the relationship between the I/D polymorphism of the *ACE* gene and speed training. The study covered a group of women among which a more favorable response (shown in parameters desirable in sprint) was observed among participants with the DD genotype [24] (Cam et al. 2007). Pescatello et al. [28] examined the effect of the I/D polymorphism of the *ACE* gene on dynamic strength/power gains in the upper limb in non-sporting and sporting men and women. The highest training-induced increments were observed in trained individuals possessing at least one D allele [28].

The present study was not without limitations. The obtained results of the genetic associations studies need to be interpreted with caution as they can be influenced by many factors. The failure to detect gene x physical activity interaction effects in our study may reflect the influence of population-specific characteristics such as high overall physical activity levels and relatively low weight in the studied population, a small sample size, or the effect of age. In addition, athletic performance is a polygenic trait; over 79 polymorphisms have been associated with elite athlete status. The genetic marker analysed independently is likely to make a limited contribution to an "elite phenotype": it seems more likely that such status depends on the simultaneous presence of multiple such variants [3].

In summary, we described the significant genotype × training interactions for VEmax and power of countermovement jump, whereas training improvements were demonstrated for almost all parameters. In addition, main effects of *ACE* I/D genotype on TGL, HDL, glucose and 10 m run were observed. However, more experimental studies are needed to establish the *ACE* gene × physical activity interactions.

What this study adds?

This study indicate that the ACE I/D polymorphism is associated with changes in VEmax and power of countermovement jump in response to a 12-week aerobic training programme in Caucasian women.

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