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ACTN3 ASSOCIATION ON MAXIMAL MUSCLE POWER, AFTER 6 WEEKS OF POWER TRAINING

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(Original scientific paper)

Vladimir Vuksanovikj[1], Aleksandar Aceski[1], Robert Hristovski[1], Zoran Popovski[2]
[1]Faculty of physical education sport and health, University Ss. Cyril and Methodius, Skopje, Macedonia
[2]Faculty of Agricultural Sciences and Food, University Ss. Cyril and Methodius, Skopje, Macedonia

Abstract

The research tried to discover/find out [1] whether the success of transformation of max muscle power will be just because of the training sessions? Or perhaps [2] the genetic potential of examinees will be important to make bigger and faster transformation of max muscular power. The experimental program was applied within (N) 21 examinees, age of 18-20 years, non-athlete population, for a period of 6 weeks. The programme included exercises for transformation of the maximal strength component of the flexors and extensors muscles on the elbow of the examinees' non-dominant arm (load- 90-95% of one repetition maximum). Three tests for one repetition maximum (flexion+ extension) were performed (program start, 3th and 6th week). Mutation of ACTN3 genotype allele, of the examinees, were analysed, according to establish three types of examinees: (type I) slow-oxidative - XX type/group, (type IIA) fast oxidative/glycolytic RX type/group and (type IIB) fast glycolytic -RR type/group. The three groups of examinees (XX, RR, XR) do not differ in terms of the achievements of one repetition maximum (flexors/extensors) after 3th and after 6th week. The genetic predisposition of the muscle tissue for strength capacities is not always decisive for the transformation of the maximal strength abilities.

Key words:*1RM; ACTN3; flexion; extension; training;*

Introduction

Sport is continuously rising the levels of physical performance limits. This can be proved by the official records or physical demand that athletes are expressing. The methodological structure of periodisation in sports and training sessions has been highly improved. Most of this improvement is due to the available technology and its implementation in sport training processes.

The logical framework in sport modelling includes selection as a factor of success. In addition to testing the manifested abilities of human performance, sport science attempted to include the genetic factor for pre-selection in sport. This idea should perhaps answer which of the athletes can be genetically predominant for sport success? Or perhaps, which of the athletes' performance abilities can be developed, having in mind the athletes' genetic potential.

The genetic impact has been related to sport physical performances in heredity association studies (Druzhevskaya et al., 2008; Yang et al., 2003; Norman et al., 2014). This genetic predisposition has been shown as stable within the human life span (Matthew et al., 2006), which is supporting the idea that there is some pattern which can precisely describe human abilities. The crucial association of genetic influence on physical sport performance has been found within the ACE gene (Angiotensin-converting enzyme), IGF-1 (Insulin-like growth factor 1), mitochondrial DNA, alpha actinin 3 genotype–(ACTN3), and AMP deaminase 1-AMPD3 (Davids and Backer, 2007). Somehow, ACTN3 is pointed as a nearby responsible factor for genetic association on physical performances (Druzhevskaya et al., 2008; Eynon et al., 2009; Clarkson, et al., 2005). α-actinin mutation (domination of XX allele) has been shown that has a direct (negative) influence on muscle efficiency to contract fast (Niemi and Majamaa, 2005; Papadimitriou et al., 2008; Roth et al., 2008). This mean that presence of RR allele is associated with fast twitched muscle fibres, also responsible for manifestation of strength occupied movements (Clarkson et al., 2005; Walsh et al., 2008). This type of picture of association is confirming the theory of linear genetics domination (Bouchard and Hoffman, 2011) which iseven proven by meta-analysis (Fang, 2013). Regarding the sport selection methods, the fact that, the XX allele is found within 16–19% of the general populations of European and

Asian origin, and is much less frequent in populations of African origin (Vincent et al., 2012), can sometimes be supportive, too. Same authors recommend that in the process of transformation of strength abilities, there is also a genetic impact, in which "not only" the ACTN3 gene participates. Buks (Buks et al., 1998) and Kees (Kees et al., 2012) remark that this "not only" impact is because of variance in environmental conditions. Molenaar claim for a third source of phenotypic variability besides the genetic and environmental (Molenaar et al., 1993; Molenaar and Raijmakers, 1999). The dynamics of development stands as a strong candidate for theis third source. It is based on the developmental enhancement of the initial random differences of the tissue organization which in time bring about significant differences of it. These differences, then, produce differences in the functional output.

Regarding those studies, Rankinen et al. (2005) point that diversity between physically active and non-active individuals is not directly conditioned by differences in human genotype. This is opening the theory that the genetic potential is also present within non-physically active individuals, but has never emerged on surface. The idea of this research which differs it from the previous researches is to discover [1] whether the success of transformation of max muscle power will be just because of the training sessions? Or perhaps [2] the genetic potential of candidate will be important to make bigger and faster transformation of max muscular power. Polymorphism of XX, RR or XR allele, within the ACTN3 genotype, was chosen as a best represent for the association of heredity on max muscle power, regarding the studies explored above in the text.

Materials and Methods

Participants

Total of 21 participants (average age of 19 [± 0.5] years), within 3 groups ((XX) N=5, (RR) N=8, and (RX) N=8), were included in this study. They were non-athlete population and the study was conducted on participant's non-dominant arm.

Design and Procedures

The ACTN3 genotype analysis was performed through a blood sample, taken from each of the examinees included in the research. This enabled us to perform the human type of examinees according to the dominance of some of the 3 types of muscle fibres, and to divide the examinees in 3 groups: (type I) slow-oxidative – XX type, (type IIA) fast oxidative/glycolytic RX type and (type IIB) fast glycolytic -RR type. In accordance with this classification of muscle fibres, the hypothesis was set that, most probably, the examinees with dominant muscle fibres of the type IIb (fast – RR allele type) shall be more successful in the transformation of the maximal strength component as compared to those with dominant muscle fibres of type I (slow-oxidative XX allele type). No position was established on the examinees with dominant mixed type of muscle fibres (RX allele type) since the expectation was that the speed and intensity of maximal strength transformation shall be analysed following the completion of the experimental programme.

In order to check the impact of genetics on the maximal strength transformation, an experimental training programme was designed. The programme included exercises for transformation of the maximal strength component of the flexors and extensors muscles on the elbow of the examinees' non-dominant arm. For exercises of the elbow flexor muscles (*m. biceps brachii*, *m. brahialis, m brachioradialis*), exercises with barbells were performed on a Scott bench, in seating position. For the extensor muscles (mm. triceps brachi), elbow exercises were performed with triceps pulley pushdown during which the objective was to level the elbow (from flexion towards extension), in a position when the back side of the upper arm is supported on the Scott bench board, in an upright position. The experimental programme was performed by the examinees in a period of six weeks and they have worked with maximal external load, three times a week (Ramsay at al., 1990; Marx at al., 1998). The external load dosage in the experimental programme was the same for all examinees and it referred to both included muscle groups, and the same was defined in the test for one maximal repetition amounting 90% of 1RM of the achieved results, individually for both muscle groups, for all examinees. The change of muscle strength of the examinees was individually monitored, during each training. In order to intervene so as to keep the load level of 90% of 1RM throughout the programme (Zatsiorsky and Kraemer, 2006), weight was chosen (for each examinee, in each group, at each training) which limited the number of repetitions in each series from one to three repetitions. The number of series for both muscle groups was limited to 3 series per training. Breaks between series were limited to 3-5 minutes. During the same training, the examinees of the three groups have first performed

the flexor exercises, and after the 10 minutes' break, they would commence the performance of elbow extensors exercises.

For the purposes of assessing the muscle strength, tests were realised for one maximal repetition of the muscle flexors (1RMBI) and extensors (1RMTRI). Tests were performed at the beginning of the experimental programme, after 3 weeks, and following the completion at 6 weeks of the planned programme.The results of these tests were used to assess the performance differences between groups (XX, RR, and XR) and to detect the potential genetic impact of the examinees on the test results.

Statistical Analysis

In order to test the planned program, Friedman Anova (and Post Hoc Wilcoxon matched pairs test) was performed on each of the group (XX, RR, XR) between 3 test points, as well as Kruskal-Wallis Anova for between group differences comparison. The Cohen'd effect size with 90 % CL were evaluated as trivial (0–0.19), small (0.20–0.49), medium (0.50–0.79) and large (0.80 and greater) (Cohen, (1992)).

Results and Discussion

Descriptive statistic is presented at Table 1.

Table 1. Descriptive statistics: 1RMBI-one repetition maximum test for elbow flexor; 1RMTRI-one repetition maximum test for elbow extensor;

test	Group	Initial			control			final		
	(genetic domination)	mean±Sd	Min	Max	mean±Sd	Min	Max	mean±Sd	Min	Max
1RMBI	XX (slow twich)	13.0±1.2	12.0	15.0	16.6±2.68	13.0	20.0	19.1±2.79	15.0	22.5
	RR (fast twich)	13.1±1.15	12.0	15.0	17.4±4.8	12.5	27.5	18.8±3.4	15.0	25.0
	RX (mixed)	13.5±1.3	12.0	15.0	16.3±2.3	12.0	20.0	20.0±2.67	15.0	44.5
1RMTRI	XX (slow twich)	28.0±2.09	25.0	30.0	33.1±5.75	25.0	40.0	37.8±4.6	32.0	44.0
	RR (fast twich)	26.0±4.4	20.0	32.0	32.7±7.19	22.5	42.5	37.4±6.95	30.0	47.5
	RX (mixed)	27.1±3.08	21.5	30.0	32.5±4.01	27.5	40.0	38.2±4.11	34.0	44.5

FridmanAnova (Table 2) shows significant differences in each group (XX, RR and XR), between 3 test time points (initial, control and final)

Table 2. Friedman Anova-differences in groups at initial, control, and final tests (1RMBI-one repetition maximum test for elbow flexor; 1RMTRI-one repetition maximum test for elbow extensor)

group	тест	Chi Sqr.	N	df	p-level
XX	1RMBI	10.00	5	2	**0.007**
	1RMTRI	9.58	5	2	**0.008**
RR	1RMBI	13.00	8	2	**0.002**
	1RMTRI	16.00	8	2	**0.000**
RX	1RMBI	15.00	8	2	**0.001**
	1RMTRI	15.55	8	2	**0.000**

According thePost hoc testresults (Table 3) it can be concluded that there are significant maximal strength effects as regards the flexors (1RMBI) in all 3 groups (XX, RR and XR). The examinees of the XX group have succeeded in improving the mean maximal strength of flexors by 27.2% (±18.3) (p=0.043, d=1.38±0.82) in the first 3 weeks, 15.3% (±2.7) (p=0.043, d=0.81±0.13) in the second 3 weeks, (total in 6 weeks) 46.6% (±19.7), (p=0.043, d=2.19±0.77), as was expected by the experimental programme. The same XX group, for the extensor muscles, has shown (Table 4) significant changes in the second 3 weeks 15.0% (±7.2), (p=0.043, d=0.84±0.38) and total after the 6 weeks of exercises of 34.5% (±12.7), (p=0.043, d=1.77 ± 0.57), but not in the first 3 weeks. The muscle extensor group is an antigravity group, which could essentially more strongly resist the external load; therefore, it is possible for the same quality also to be reflected during the transformation process, thus requiring more time (perhaps even more intensive training) for producing similar changes as in the flexor muscles; perhaps that is the reason why there are no significant changes in the first 3 weeks (even though there is improvement of 18.2%).

Table 3. Post Hoc test for in the groups differences, change in mean % and effect size at initial, control, and final tests, for flexor muscles -1RMBI-test (initial/control - first 3 weeks; control /final-second 3 week (3th to 6th week); initial/final- total 6 weeks of experimental program)

Group	Test: 1RMBI	Wilcoxon p-level	Change in mean %	Chances for value: smaller/similar/greater	Uncertainty in the true differences	Cohen's d ± 90% CL
XX	initial/control	**0.043**	27.2 ±18.3	98/1/1	very likely +ive	1.38±0.82
	control /final	**0.043**	15.3 ±2.7	100/0/0	most likely +ive	0.81±0.13
	initial/final	**0.043**	46.6 ±19.7	100/0/0	most likely +ive	2.19±0.77
RR	initial/control	**0.012**	29.5 ±18.6	99/1/0	very likely +ive	1.24 ± 0.68
	control /final	0.161	9.6 ± 9.8	86/13/1	likely +ive	0.44 ± 0.43
	initial/final	**0.012**	42 ± 10.5	100/0/0	most likely +ive	1.68 ± 0.35
RX	initial/control	**0.018**	19.7 ± 10.7	99/1/0	very likely +ive	1.23 ± 0.61
	control /final	**0.018**	23.1 ± 11.3	100/0/0	most likely +ive	1.43 ± 0.63
	initial/final	**0.012**	47.4 ± 14.1	100/0/0	most likely +ive	2.66 ± 0.65

The group (RR) with dominant fast muscle fibres has also shown statistically significant improvement of values for the maximal strength of flexors of 29.5% (±18.6), (p=0.012, d=1.24 ± 0.68) in the first 3 weeks, as well as total after 6 weeks of exercises of 42% (± 10.5), (p=0.012, d=1.68 ± 0.35), but not in the second 3 weeks of exercises (Table 3.), although it was expected that precisely this group with dominant fast twitched fibres in the muscles shall be the group that will demonstrate the greatest benefit from the transformation programme. Unlike the flexors, significant changes were observed (Table 4) for the extensor muscles of this (RR) group in all 3 relations after 3 weeks = 24.5% (± 10.5), (d=0.99 ± 0.38), from week three to six = 15.4% (±7.3), (d=0.65 ± 0.29), and total in six weeks = 43.6% (±10.7), (d=1.64 ± 0.34) at level of p=0.012. Group XR, with demonstrated mixed genetic predispositions (dominance of fast + slow muscle fibres), has demonstrated similar changes as the other two groups (dominance of slow muscle fibres) in flexor muscles 19.7% (±10.7), (p=0.018, d=1.23 ± 0.61) after 3rd week, 23.1% (±11.3), (p=0.018, d=1.43 ± 0.63) from week three to six, and 47.4% (±14.1), (p=0.012, d=2.66 ± 0.65) total after 6 weeks of exercises (Table 3.). The extensor muscles have also had significant reaction to the experimental procedure in this group (RX) with increase in the maximal strength values of 19.8% (±10.3), (p=0.018, d=1.39 ± 0.66), 17.7% (±4.2), (p=0.012, d=1.25 ± 0.27) and 40.9% (±15.0), (p=0.012, d=2.64 ± 0.82), Table 4.

The analysis has shown that the experimental procedure, for a relatively short period, managed to enable the positive transformation of the maximal values of the flexor muscles, as well as of the extensors, within the 3 groups of examinees, with high success percentage. This means that the incentive for this transformation was adequately chosen, and was properly/continuously increased. Therefore, the examinees have successfully responded to the transformation of the maximal strength capacities.

Table 4. Post Hoc test for in the groups differences, change in mean % and effect size at initial, control, and final tests, for flexor muscles -1RMTRI-test (initial/control - first 3 weeks; control /final-second 3 week (3th to 6th week); initial/final- total 6 weeks of experimental program

Group	Test:1RMTRI	Wilcoxon p-level	Change in mean %	Chances for value: smaller/similar/greater	Uncertainty in the true differences	Cohen's d ± 90% CL
XX	initial/control	0.068	17.0 ±15.5	95/3/2	likely +ive	0.94 ± 0.79
	control /final	**0.043**	15.0 ± 7.2	99/1/0	very likely +ive	0.84 ± 0.38
	initial/final	**0.043**	34.5 ± 12.7	100/0/0	most likely +ive	1.77 ± 0.57
RR	initial/control	**0.012**	24.5 ± 10.5	100/0/0	most likely +ive	0.99 ± 0.38
	control /final	**0.012**	15.4 ± 7.3	99/1/0	very likely +ive	0.65 ± 0.29
	initial/final	**0.012**	43.6 ± 10.7	100/0/0	most likely +ive	1.64 ± 0.34
RX	initial/control	**0.018**	19.8 ± 10.3	99/0/0	very likely +ive	1.39 ± 0.66
	control /final	**0.012**	17.7 ± 4.2	100/0/0	most likely +ive	1.25 ± 0.27
	initial/final	**0.012**	40.9 ± 15.0	100/0/0	most likely +ive	2.64 ± 0.82

In order to assess the potential differences in the achievements of each of the groups as a result of the genetic potential possessed by the examinees, Kruskal-Wallis Anova was performed (Table 5).
Although the expectations were that there will be inter-group differences in terms of the muscle transformation quality, the analysis of the arithmetic means through Kruskal-Wallis Anova has

demonstrated that there are no differences between the three groups (XX, RR, and XR), after 3 weeks of exercises (initial test), after six weeks of exercises (final), as well as from week three to week six (control). This shows that the three groups of examinees, regardless of the different dominance of the XX, RR, and XR alleles, do not differ in terms of the achievements in both measuring points (Chart 1).

Table 5. Kruskal-wallis Anova-between groups differences (XX, RR, and RH), tested at initial, control, and final tests. (Initial-before start of the experimental program; control-after 3th week of program; final-after the 6th week of program; 1RMBI-one repetition maximum test for elbow flexor; 1RMTRI-one repetition maximum test for elbow extensor)

period	test	K-W test	N	df	p-level
Initial	1RMBI	0.79	21	2	0.674
	1RMTRI	0.90	21	2	0.639
control	1RMBI	0.17	21	2	0.919
	1RMTRI	0.22	21	2	0.895
final	1RMBI	0.73	21	2	0.694
	1RMTRI	0.09	21	2	0.954

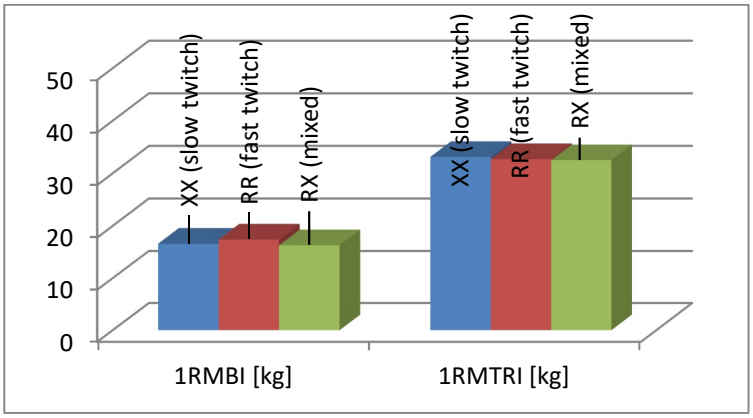

Chart 1: differences between XX, RR, RX group in three-time sequences (initial, control, final), for two performed test (1RMBI and 1RMTRI)

Conclusion

According to our findings, and the research so far in the field of genetics and exercises, we were unable to find a study that touches upon the genetic factor impact (more specifically, ACTN3) on the success from the realised experimental programme for maximal strength transformation of the non-dominant arm, in the muscles flexors and extensors of the elbow.

The subject of research of this study was whether [2] the success of transformation of the strength abilities in both involved muscle groups shall be conditioned upon the genetic factor of the involved examinees, which is presented in Table No 5. In the three groups of examinees (which were homogenised according to the ACTN3 domination of the XX, RR, or XR alleles), throughout the experimental procedure, there was statistically identical occurrence of transformation of the maximal strength in both flexors and extensors. Therefore, it can be concluded that, in this research, the decisive performance factor in the maximal strength transformation of the examinees was the training process through a strictly directed individual programme [1]. The research has shown that the impact of the genetic factor carried by the examinees cannot be incorporated in the transformation success of such type of ability in these two muscle groups.

The available articles (Niemi and Majamaa, 2005; Moran, 2007) have indicated the possibility that the group of examinees (group: “fast”) with determined RR factor (and potentially RX, group: mixed) in the ACTN3 analysis, shall demonstrate relatively improved achievements as regards the maximal strength throughout the experimental procedure, as compared to the group of examinees (group: “slow”) which have XX factor of ACTN3 in their genetic code (and potentially those with RX). Nevertheless, this research has shown that the logic for genetic predisposition of the muscle tissue for strength capacities is not always decisive for the transformation of the maximal strength abilities through such an experimental procedure.

Perhaps, in the future, one should analyse "whether and which" motor capacities in humans are most certainly affected by R577X, followed by the "strength" of this mutation as a predictor, as well as to more precisely "define" how α-actinin-3 impacts the skeletal muscle function (MacArthur, 2007; Bouchard and Hoffman, 2011).

The results of this research are closest to the interpretations of Davids and Backer, (2007) that, most probably, there is no "magic gene" for a specific motor ability. Perhaps that is why "the long step from polypeptide sequences to motor behaviour is a step that covers much incompletely understood theories" (Johnston and Edwards, 2002). Even the most common "noise" in the chemical-electrical fluctuations (Kauffman, 2003) should be a serious topic for study of the genetic networks (Davids and Baker, 2007).

The seriousness of the approach to be chosen in the subsequent such analysis can be perceived in the example of linearity between heredity and height in humans (occurring as a generally stable dimension). GWAS (genome-wide association study) approach has shown that the height of the individual can be assessed up to 80% and that this genetic predisposition is supported by about 40 genetic regions, which actually explain in total about 5% of the dependence of the human height from their parents (Bouchard andHoffman, 2011). What about the unexplained part?

Most probably, while creating the concept for the future analysis, one should take into consideration the overall system behaviour and its self-organisation, which represents wider than the linear view of issues (Brutsaert and Parra, 2006; Davids et al., 2003; Garcia, et al., 2015). Recognising the previous experiences of the authors who have worked on this issue, one should take into consideration the complexity of genetics as a field, the environment in which the individual exists, as well as the self-organising processes which have an impact on humans.

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