

Blind Title Page**A Comparison of Training Modality and Total Genotype Scores to Enhance Sport-Specific Biomotor Abilities in Under 19 Male Soccer Players**

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ABSTRACT

Soccer-specific training (SST) and small-sided games (SSGs) have been shown to develop physical proficiency in soccer. Research on genetics and epigenetics in the prescription of training is limited. The aims of this study were to compare the impact of three different SST/SSG methods and investigate if a total genotype score (TGS) influences training response. Subjects ($n = 30$ male soccer players, mean \pm *SD*; age 17.2 ± 0.9 years, stature = 172.6 ± 6.2 cm; body mass = 71.7 ± 10.1 kg) were stratified into a ‘power’ (PG) or ‘endurance’ (EG) gene profile group, where a 15 Single Nucleotide Polymorphism (SNP) panel was used to produce algorithmically weighted TGS. Training 1 (T1 – SSGs only), Training 2 (T2 – SSGs / SST) and Training 3 (T3 – SST only) were completed (in that respective order), lasting 8 weeks each, interspersed by 4-week washouts. Acceleration (10 m sprint) was improved by T2 only (1.84 ± 0.09 s v 1.73 ± 0.05 s; Effect Size (ES) = 1.59, $p < 0.001$). Speed (30 m sprint) was improved by T2 (4.46 ± 0.22 s v 4.30 ± 0.19 s; ES = 0.81, $p < 0.001$) and T3 (4.48 ± 0.22 s v 4.35 ± 0.21 s; ES = 0.58, $p < 0.001$). Agility (T-test) was improved by T1 (10.14 ± 0.40 s v 9.84 ± 0.42 s; ES = 0.73, $p < 0.05$) and T3 (9.93 ± 0.38 s v $9.66 \pm .45$ s; ES = 0.66, $p < 0.001$). Endurance (Yo-Yo Level 1) was improved by T1 (1682.22 ± 497.23 m v 2028.89 ± 604.74 m; ES = 0.63, $p < 0.05$), T2 (1904.35 ± 526.77 m v 2299.13 ± 606.97 m; ES = 0.69, $p < 0.001$) and T3 (1851.76 ± 490.46 m v 2024.35 ± 588.13 m; ES = 0.35, $p < 0.05$). Power (Countermovement Jump) was improved by T3 only (36.01 ± 5.73 cm v 37.14 ± 5.62 cm; ES = 0.20, $p < 0.05$). There were no differences in T1, T2 and T3 combined when comparing PG and EG. The PG reported significantly ($\chi^2_{20} = 4.42, p = 0.035, ES = 0.48$) better training responses to T3 for power than the EG. These results demonstrate the efficacy of SSGs and SSTs in developing biomotor abilities. Although these results refute talent identification through the use of a TGS,

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there may be use in aligning training method to TGS to develop power-based qualities in soccer.

Key Words Training intervention, genetics, sports performance, Academy, genetics, DNA.

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A Comparison of Training Modality and Total Genotype Scores to Enhance Sport-Specific Biomotor Abilities in Under 19 Male Soccer Players

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INTRODUCTION

Reaching professional levels of performance within soccer is underpinned by extensive deliberate practice, strong social support, effective coaching and can also be impacted by an athlete's place or month of birth (31). The extent to which each of these characteristics contributes to professional soccer status is unknown; however, an aspect to be considered is epigenetic and genetic variation, with many traits having a highly heritable component (2). Epigenetic variation (alternation in gene expression as a result of non-genetic modifications of DNA) has been shown to influence performance responses as a result of environmental exposure (26). The genetic make-up of the performer is suggested to account for approximately 50% of overall physical performance, although estimations for each performance component is thought to vary between 14-81% (29).

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Single nucleotide polymorphisms (SNPs) represent common variations in the DNA sequence between individuals, potentially altering translation of the corresponding protein (1). For example, a SNP in *ACTN3* termed R577X (rs1815739) has been shown to alter the production of α -actinin-3, a protein found in type-II muscle fibres (31). Individuals with the “R” version of this genotype have been found to be able to increase muscle force production (15, 20), recruit greater volumes of type-II muscle fibres (28) and increase muscle mass response from training (15, 20). The “RR” genotype of this SNP (associated with elite status in power based sports) and has been found to have a significantly higher distribution in a FIFA world ranked soccer club than controls and elite runners (25). Using this methodology, single candidate gene association studies have linked a range of SNPs to elite athlete status (2). Ahmetov and Fetodovskaya (1) reviewed research on 120 SNPs which had been proposed to be associated with elite athletic status and found conflicting results in 81 SNPs, whereby additional studies found either no effect, or the opposite effect, of these SNPs on elite athletic status. An example of this is in the *ACTN3* gene, which was found to have 12 studies ($n=1484$) with positive results associated to power athlete status and 5 studies ($n=498$) with negative results associated to power athlete status (1). As such, the role of individual SNPs within elite athlete status remains unclear (15) and its use for talent identification is currently deemed not appropriate, as well as unethical (30).

Williams and Folland (29) coined the term ‘Total Genotype Score’ (TGS), which was generated by the quantification of the combined influence of 23 SNPs associated with elite performance. Egorova et al. (9) demonstrated a significantly higher mean \pm SD TGS for 246 professional Russian soccer players (52 ± 17.6) when compared to 872 Russian controls (41.3 ± 15.5 ; $p < 0.001$) using a TGS derived from only four SNPs. Although a TGS could be a promising method of combining SNPs, no difference was found between elite, sub-elite and

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non-elite soccer professionals, suggesting that using this TGS may not be sensitive enough in predicting elite soccer status within professional soccer (9).

The impact of genetics outside of achieving elite status has received limited attention and it may be beneficial for practitioners to gain an understanding of how certain SNPs impact training responses (14). Bouchard, Rankinen and Timmons (5) adopted a 20-week exercise programme for 473 sedentary adults, demonstrated that 39 SNPs were associated with enhanced gains in aerobic capacity and estimated that this explained 47% of the heritability of aerobic capacity. Zarebska et al. (33) found that responses of explosive power variables from 201 sedentary women to twelve weeks of aerobic dance training were significantly associated with the *AGT* M235T SNP. Using an algorithmic weighted TGS devised from 15 SNPs, Jones et al. (14) stratified 67 sub-elite athletes into a 'power' or 'endurance' group. The 'power' group conducted high-intensity resistance training and the 'endurance' group conducted low-intensity resistance training respectively, resulting in significantly greater adaptive responses in both counter-movement jump (CMJ) and 3-minute cycle test. Although these three studies provide some information around potential training responses based on a genetic profile, no studies have looked at the responses of physical conditioning in soccer in relation to certain SNPs or TGS, despite the heritability behind physical development and its relative importance to success in soccer (2).

Due the importance of physiological development in attaining professional soccer status (22), studies have looked at physical development, with a recent shift towards a sport-specific paradigm of soccer-specific training (SST) and small-sided games (SSGs) (18). Using SST/SSGs, studies have shown a higher meter squared (m^2) per player (i.e. playing area size squared ($\text{width} \times \text{length}$) \div number of players), increased player numbers, longer duration,

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lower intensity and higher work:rest ratios to be more effective in inducing greater aerobic gains (6, 11, 13, 16). In contrast, studies have found a smaller m^2 per player, decreased player numbers, shorter duration, higher intensity and lower work:rest ratios to be more effective in inducing greater gains in acceleration, speed (12), agility (7) and power (22). The interaction between these methods of training and SNPs and TGSs has not been considered, thus the first aim of this study was to compare the effect of three different soccer-specific training (SST/SSG) methods on acceleration, speed, agility, power and aerobic capacity (biomotor abilities). The second aim of this study was to investigate if an algorithmically weighted TGS has an impact on training response to SST/SSGs. It was hypothesized that (a) T1, T2 and T3 will significantly develop acceleration, speed, agility, power and endurance in 8 weeks, (b) there will be no significant difference between those classified in the power and endurance groups with respect to acceleration, speed, agility, power and endurance at baseline or post-training methods combined and (c) there will be a significant interaction between group (power vs endurance) and the training program performed (T1, T2 and T3) with respect to the development of acceleration, speed, agility, power and endurance.

METHODS

Experimental approach to the problem

The study was a mixed model design with two factors. The first factor (within subjects) was the training modality implemented (*i.e.* SSG, SSG/SST and SST), the second factor (between subjects) was the gene profile group (*i.e.* power (PG) or endurance (EG)). The investigation was conducted during the 2015-2016 competitive season on an outdoor third generation artificial turf pitch (17) and the training protocol can be found in Figure 1.

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Insert Figure 1 about here

Training methodology and periodisation can be found in Table 1. Pre- and post-training intervention, the subjects performed 10 and 30 m sprint tests, Agility T-Test, a Counter Movement Jump (CMJ) and the Yo-Yo Intermittent Recovery Level 1 Test (YoYo1). The subjects were familiarised with both the testing parameters and then the training methods prior to the experimentation.

Insert Table 1 about here

Each intervention session began with a 15-minute warm-up following the FIFA 11+ Injury Prevention Program (which, briefly, involves core stabilisation, proprioceptive training, eccentric training of thigh muscles, plyometrics and dynamic stabilization), followed by the physical conditioning session, finishing with low-intensity tactical training. During active recovery periods, players could drink fluids *ab libitum* and were encouraged to engage in low-intensity technical practice. Subjects were grouped during SSGs based on the minutes of competitive play, performance on the YoYo1, subjective coach rating and playing position and appropriately disseminated across training groups (6). During all training interventions coaches and resting players offered high levels of encouragement to ensure high motivation levels (8), balls were distributed around the outside of practice areas and returned to play within 3 seconds.

Subjects

Thirty young male subjects (mean \pm *SD*; age 17.2 ± 0.9 years, stature = 172.6 ± 6.2 cm; body mass = 71.7 ± 10.1 kg; soccer playing experience = 11 ± 1.8 years) volunteered for this study and were all part of an under-19 sub-elite soccer programme competing nationally in the

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premier division within the English Football College Association. Subjects trained 4-5 times a week (~ 90-100 minutes per session) which included competitive match play taking place mid-week and/or at the weekend, and technical and tactical skill development training sessions (equating to ~ 80% of the training time), with physical conditioning performed twice a week. All subjects were advised to maintain a normal diet. Subjects were required to complete 12 of 16 sessions (75%) within each block to be a part of the relevant training intervention. The study was approved by the University of Portsmouth Science Faculty Ethics Committee. Each participant gave written informed consent after procedures were fully explained. Informed parental or guardian consent for all subjects under the age of 18 was ascertained.

Procedures

The subjects were instructed not to do any physical activity for at least 48 hours before the testing session and not to drink caffeine-containing beverages on the day of the tests. Height and body mass were measured using a calibrated Tanita stadiometer (model Leicester, Seca Ltd, Birmingham, United Kingdom) and a Tanita scale (model BC-418MA, Tanita Corporation, Tokyo, Japan), respectively.

Physical Performance Tests

All performance tests took place at the same time of the day, starting at 10 AM. CMJ was measured using an Optojump system (Altes Model, Microgate, Bolzano, Italy) following the protocol used by Markovic, Dizdar, Jukic and Cardinale (21). The 10 and 30 m sprint times were measured by six-infrared light gates (TC System, Browner Timing Systems, Utah, USA) placed 0, 10 and 30 meters apart (32). The Agility T-Test was measured by four infrared light gates (TC System, Browner Timing Systems, Utah, USA) set up at beginning/end point of the test (24). The YoYo1 (17) utilised the YoYo1 program, which consists of repeated 2 X 20 m runs back and forth between a finishing line with an intermittent 10 s active resting period

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involving 2 X 5 m jogging at an increasing speed controlled by the YoYo1 Program (Ruval Entrprises, Guelph, Canada).

DNA Analysis & Grouping

All subjects had a genomic DNA test completed at the beginning of the season within their current college soccer programme. Genomic DNA was extracted from buccal swabs (Whatman's Sterile Omni Swab) using the Genra Puregene Tissue Kit (cat#158422, QIAGEN) according to the manufacturer's guidelines. The sample from the swabs were rendered acellular within 7 days of sampling for subsequent storage and future testing (*i.e.* the DNA was extracted within 24 hours of the sample being taken), in accordance with the Human Tissue Act. Using DNAFit's Peak Performance AlgorithmTM (PPA), Table 2 shows the percentage threshold that represents each individual's power/endurance threshold. The DNAFit PPA assigns a 'gene score' to each allele of 0, 1 or 2 associated with power or endurance performance. This produces an algorithmically weighted TGS culminating in a power/endurance percentage. This is similar to the approach adopted by Egorova et al. (9) and Jones et al. (14) who both adopted a weighted allele score to produce an overall TGS for comparison across populations and training groups respectively.

Insert Table 2 about here

The list of SNPs, Genes and Effect Alleles can be found in Table 3.

Insert Table 3 about here.

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Statistical analysis

Data were statistically analysed using IBM SPSS 24.0 for Windows (SPSS Inc., Chicago, IL). Normal distribution of data was checked using the Kolmogorov-Smirnov and Shapiro-Wilk tests. Pre-post comparisons were conducted using paired samples *t*-test. A mixed model ANOVA utilising delta scores was assessed (10). Post-hoc analysis used paired samples *t*-test and Kurskall-Wallis test to assess differences between groups and interaction between group and training. A Bonferroni correction was applied to post-hoc analysis and effect size (Cohen's *d*) was calculated to determine the meaningful difference between power and endurance groups. Effect size (ES) values of 0 to .19, .20 to .49, .50 to .79 and .80 and above were considered to represent trivial, small, moderate and large changes, respectively (10). The criterion for significance was set at $\alpha = 0.05$.

RESULTS*Responses to training modality irrespective of genotype group*

Pre-post intervention mean scores were found to be significantly different for 30 m sprint ($F_{(2, 16)} = 6.147, p = .010$) and Agility T-Test ($F_{(2, 20)} = 3.88, p = .038$) times and non-significant for 10 m sprint times ($F_{(2, 14)} = 1.75, p = .210$), CMJ ($F_{(2, 18)} = .817, p = .458$) and YoYo1 ($F_{(2, 20)} = 1.303, p = .294$) in response to all training types combined. Pre-post intervention mean scores for 10 and 30 m sprint times, Agility T-Test, CMJ and YoYo1 in response to T1, T2 and T3 can be found in Table 4.

Insert Table 4 about here.

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Efficacy of training modality irrespective of genotype group

T2 demonstrated an improvement in 10 m sprint time compared to T1 ($t_{(11)} = -2.05$; $p = .065$; $ES = .84$) and T2 ($t_{(19)} = 5.121$; $p < .001$; $ES = .70$). There was a significant improvement in 30 m sprint performance in response to T2 ($t_{(12)} = -2.910$; $p = .013$; $ES = 1.03$) and T3 ($t_{(9)} = -2.859$; $p = .019$; $ES = 1.32$) compared to T1 but no meaningful difference between T2 and T3. There was a significant improvement in Agility T-Test performance in response to T3 ($t_{(18)} = 3.163$; $p = .005$; $ES = 1.16$) compared to T2. There was a significant ($t_{(15)} = 2.240$; $p = .041$; $ES = .95$) improvement in YoYo1 scores in response to T2 compared to T3. T3 showed a moderate improvement in CMJ scores compared to T1 ($Z_{(15)} = -.107$; $p = .285$; $ES = .62$) and T2 ($Z_{(15)} = -1.718$; $p = .086$; $ES = 0.65$).

Interaction between training and genotype

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No significant differences were found between training types combined and genotype for 10 m ($F(2, 14) = .125, p = .883$), or 30 m ($F(2, 16) = .189, p = .830$) sprint times, Agility T-Test ($F(2, 20) = .199, p = .821$) times, CMJ ($F(2, 18) = .569, p = .576$) or YoYo1 ($F(2, 20) = .299, p = .745$) scores. No significant differences were found between the PG and the EG at baseline for 10 m ($t_{(29)} = -.257; p = .799; ES = .02$) or 30 m ($t_{(33)} = .283; p = .799; ES = .02$) sprint times, Agility T-Test times ($t_{(30)} = -1.227; p = .230; ES = .01$) or CMJ scores ($t_{(27)} = .141; p = .889; ES = .03$) for each of the training scenarios. At baseline, the PG scored significantly higher than the EG on the YoYo1 ($t_{(25)} = 2.131; p = .043; ES = .05$) for the training types combined. No significant differences were found between training and genotype post-training for 10 m ($t_{(29)} = -.032; p = 0.975; ES = .02$) or 30 m ($t_{(30)} = .243; p = .810; ES = .02$) sprint times, Agility T-Test times ($t_{(30)} = -1.614; p = .117; ES = .01$), CMJ scores ($t_{(27)} = -0.541; p = .593; ES = .01$) or YoYo1 ($t_{(25)} = .661; p = .515; ES = .01$) scores. No significant differences were found between the PG and the EG in response to T1, T2 or T3 for 10 and 30 m sprint times, Agility T-Test times or the YoYo1 scores.

Mean percentage change in CMJ for the PG and the EG in response to training modality

Figure 2 shows mean percentage change in CMJ scores from pre- to post-training for T1, T2 and T3. The PG recorded significantly greater improvements than the EG in response to T3 ($X^2(20) = 4.42, p = .035, ES = .48$). The EG showed small ($t_{(16)} = -.565; p = .580; ES = .27$) and moderate ($t_{(23)} = -1.435; p = .165; ES = .57$) improvements in CMJ when compared to the PG in response to T1 and T2 respectively.

Insert Figure 2 about here.

Mean percentage change in YoYo1 for the PG and the EG in response to training modality

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Figure 3 demonstrates the mean percentage change in YoYo1 scores between the PG and the EG groups in response to T1, T2 and T3. The PG showed a trivial ($t_{(15)} = .269$; $p = .792$; $ES = .13$) improvement in YoYo1 scores in response to T3 when compared to EG. The EG showed a trivial ($t_{(16)} = -.395$; $p = .698$; $ES = .19$) and moderate ($t_{(21)} = -.361$; $p = 1.88$; $ES = .57$) improvement in YoYo1 scores in response to T1 and T2 respectively when compared to the PG.

Insert Figure 3 about here.

DISCUSSION

The results of this study demonstrate the SST/SSGs approach can significantly improve acceleration, speed, agility, power and aerobic capacity. Moreover, the results of this study indicate that these physical responses to SST/SSGs were largely be achieved irrespective of a TGS profile. Despite this, utilising T3 for soccer players with a power TGS profile can significantly improve mean CMJ scores when compared to an endurance TGS. To the best of the authors' knowledge, this is the first study to examine the efficacy of utilising genetic profiling methods to target a range of athletic qualities in the form of SST/SSGs in soccer.

In contrast to previous research (4, 11, 27), our study found that T1 in isolation was not effective in developing acceleration, speed or power. In concurrence with previous research (7), however, our study found that T1 significantly improved agility. Although agility has been highly correlated with strength per kilogram of body weight, the vast changes of direction in SSGs may have been sufficient in stimulating neural adaptation (27). Despite this, we did not measure strength or deceleration and it may have been that our method of measurement for

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acceleration and power may not have been sufficient in tracking improvements in these qualities. The T1 methodology in our study reflected elements of Chaouachi, Chtara, Hammami and Castagna's (7) study (3v3, 2min, 1:1), however these authors also utilised 1v1 and 2v2 conditions (30s, 1:4 and 1 min, 1:2, respectively) more closely reflecting T2 methodology in our study.

SSGs have been found to frequently be an effective method of developing aerobic capacity, thought to be driven by acute stress to heart rate and blood lactate resulting in long-term physiological adaptations (4, 11, 27) the present study also supports this theory. Larger relative SSGs (i.e. 100 m² per player and above) with smaller player numbers (2v2 to 4v4) were responsible for stimulating the highest levels of HR, blood lactate and RPE (13) although there were no differences found in the magnitude of improvement in aerobic capacity between T1 and T2. T2 may have improved aerobic capacity due to increases in expression of the monocarboxylate transporter 1 (slowing intra-cellular H⁺ accumulation, thus delaying fatigue), possibly as T2 reflected a higher intensity training mechanism (22). Despite conflicting research (12, 22), this study found T2 significantly increased both acceleration and speed. Moreover, although this study found no improvement in power in response to T2, power improved significantly in response to T3, and also improved moderately in response to T3 when compared to both T1 and T2, possibly through increased creatine kinase activity (22). Another possible mechanism for greater acceleration / speed and power improvements to T2 and T3 respectively may have been epigenetic skeletal muscle memory (26). In a study conducted by Seaborne et al. (26) with eight previously untrained male adults, DNA hypomethylation (enhanced gene expression) was observed after a 7-week hypertrophy training period, in-line with increases in muscle mass. Interestingly, this epigenetic response was maintained after a 7-week unloading (detraining) period, despite muscle mass returning to

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baseline. This epigenetic response then increased in frequency after a period of re-loading (following the same training program as in the loading phase), in-line with greater increases in muscle mass and average load lifted when compared to the loading phase (26).

This study also found, in agreement with previous research (7), that T3 induced significant improvements in agility as well as a significantly better response to T3 than T2, most likely due the starting, breaking and turning actions being frequently present in the T3 training methodology. Theoretically, these actions may have allowed for great motor skill learning through changes to locus of action selection in brain circuit connections, increases in synaptic weight from neurons in the brain connected to the muscles and potential increases in plasticity of neurons at the local muscle level (23). These changes may improve the speed, accuracy and consistency of the body to accelerate, decelerate and change body position quickly, improving agility (23).

T3 also improved aerobic capacity significantly (possible due to general training and fixture volume) (18) although T2 significantly improved aerobic endurance by 8.47% more than T3, supporting previous research (22). Despite this, acceleration saw a significant decrease with an improvement in, speed in response to T3. This may have been due to the lack of resistance or plyometric training or due to neuromechanical changes potentially caused by the adoption of a 'long-to-short' training method adopted (i.e. 30 m sprints on week 1 v 15 m sprints on week 8) (22).

Utilizing a TGS was not predictive of training response for any physical performance parameters with no differences found between PG or EG in response to all training types combined. Moreover, there was a significantly higher aerobic capacity score at baseline for the PG when compared to the EG, both of which refute the use of genetic for broad talent

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identification. It is likely that the 15 SNPs used may not contain enough specific genetic markers that have been linked to the phenotypes in question. Although genes such as *ACTN3* have been associated with composition of muscle fibres (2), there is a lack of evidence supporting an individual SNP on performance (1). It is likely that a broad range of genes underpin training response and are likely to be both specific to the phenotype in question and polygenic in nature, as well as being influenced by epigenetic factors (2). To date, no studies have compared the impact of a gene profile on phenotype responses to football-specific training.

Despite the lack of evidence associating individual SNPs with performance (1), athletes in our study with a higher 'power' bias in their gene profile, responded significantly better to the sprint training methodology (T3) in terms of power. For athletes with a higher 'endurance' bias in their gene profile, the training methodologies with lower intensity and longer duration (T1 and T2) evoked small and moderate improvements, respectively, in power responses and trivial and moderate improvements, respectively, in aerobic capacity. These results may support the hypotheses made by Jones et al. (14), Bouchard, Rankinen and Timmons (5) and Zabreska et al. (33) that using gene profiles to select training methodology could be effective by maximizing the physiological development pathway activated in performance gains, although significant improvements would be needed in terms of the sensitivity of such profiles.

Our findings suggest the uses of TGS' in the optimization of training prescription is still some way off, with greater sensitivities required in the categorization of power- and endurance-based athletes. Our study is not, however, without its limitations. With any training study of this length attrition can be an issue, therefore for future studies a larger sample size will be recruited. Additionally, the small number of genes analyzed (20) may have had a part

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to play in the limited number of significant findings linking the TGS to training response, which could be vastly increased with recent progress in the field of genetics (2). It may well be that the results observed were impacted by the effect of overall training and match hours (19) reducing the isolation of the genetic variants, although the authors felt this was an appropriate trade-off in the interest of protecting the ecological nature of the study.

In conclusion, this study indicates that adopting an SST/SSGs approach to physical conditioning can be an appropriate method in developing acceleration, speed, agility, power and aerobic capacity, dependent on manipulations of intensity, duration, work:rest, sets, reps and total work. This study demonstrated that utilizing 15 SNPs in an algorithmically weighted TGS was not effective in optimizing training for acceleration, speed, agility or aerobic capacity, but may have modest utility in optimizing training for power. Moreover, no differences found at baseline or in response to training combined indicates the lack of predictive power when using gene profiles to assess soccer players. Future research should look to increase sample size, enhance the robustness of phenotype measures, and increase the number of genes on TGS panels to establish whether or not genetics is predictive of individual soccer players' training responses.

PRACTICAL APPLICATIONS

Our study suggests that using SST/SSGs may be effective in developing a range of biomotor abilities. It has long been known that physical capacities and trainability are variable between soccer players. Our study also found that genetic profiling using 15 SNPs was not capable of predicting training response for acceleration, speed, agility or aerobic capacity, but might have utility when planning for power development. Finally, our study found no difference baseline or post training combined when comparing gene profile groups, refuting the use of genetic testing for athlete assessment or talent identification.

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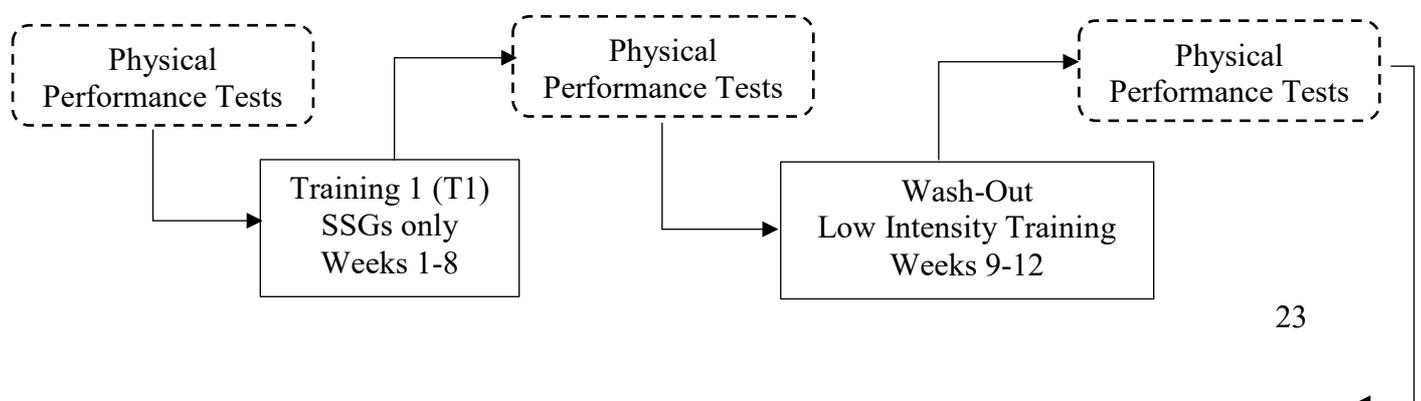
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Figure Captions



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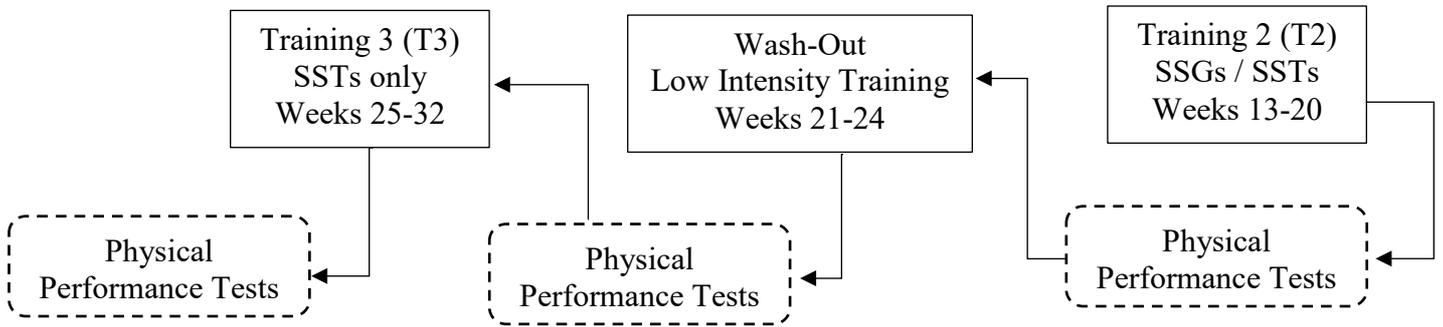
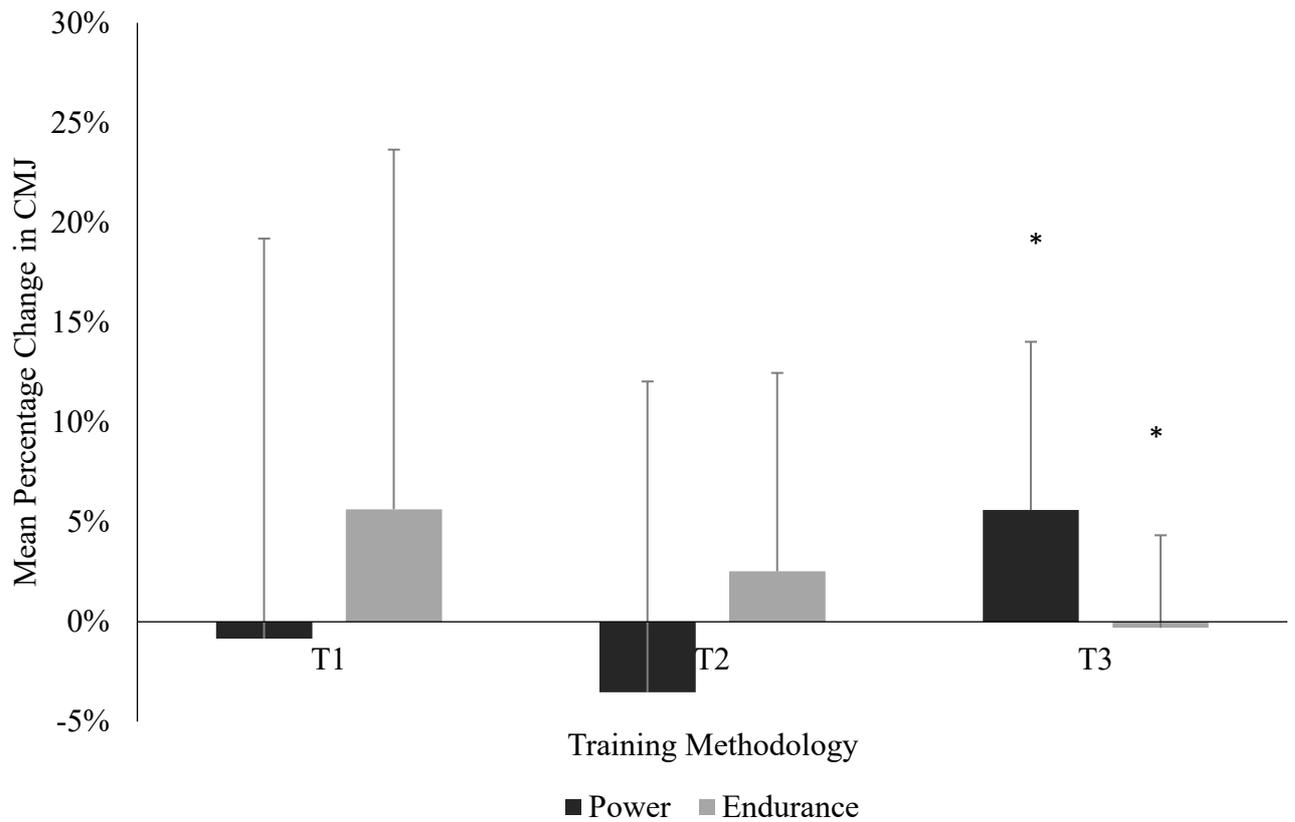


Figure 1. Training intervention flow diagram.



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Figure 2. Percentage change (mean \pm *SD*) in CMJ pre-post training for the PG and EG in response to T1, T2 and T3.

* $p < 0.05$

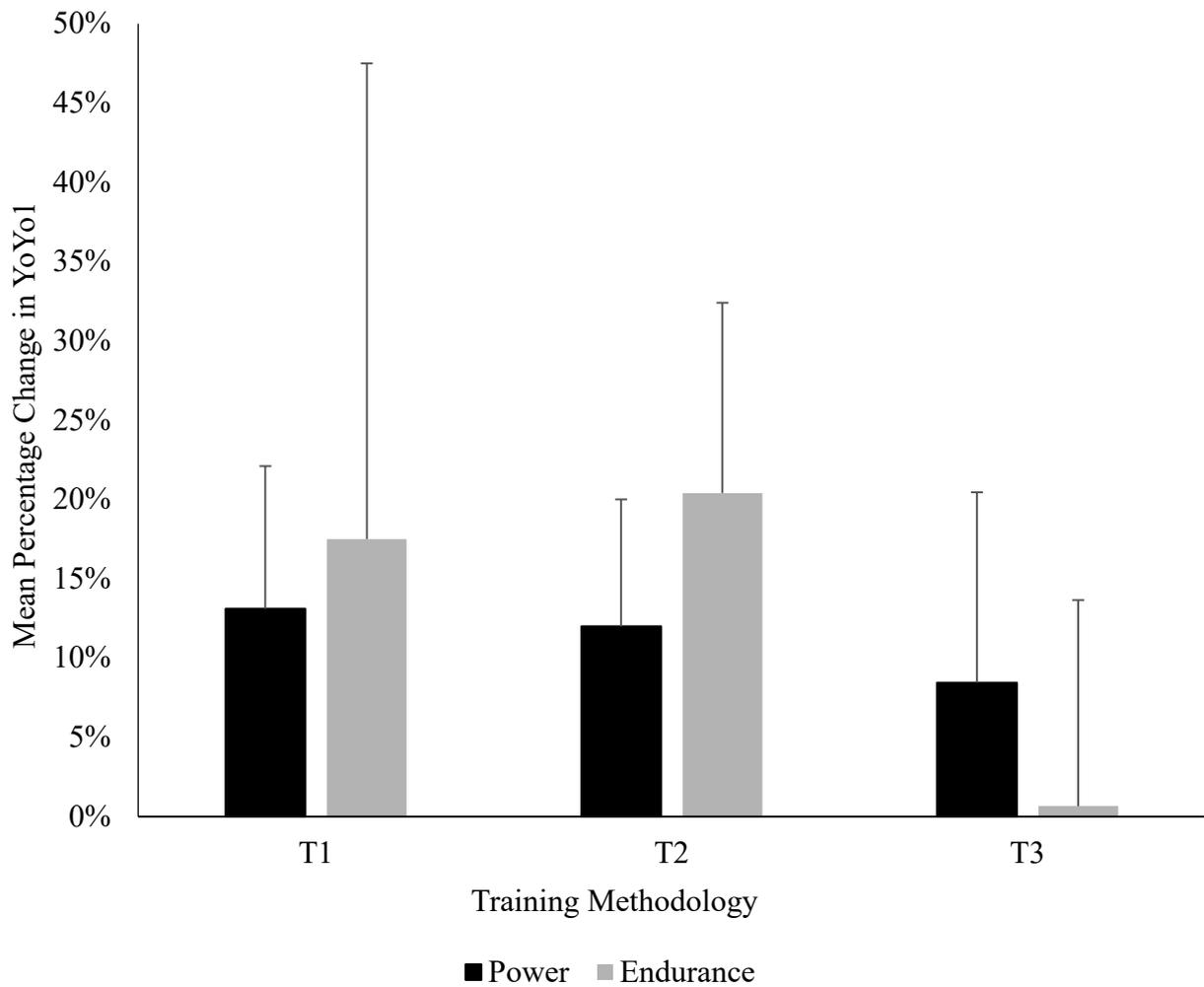


Figure 3. Percentage change (mean \pm *SD*) in YoYo1 pre-post training for the PG and the EG in response to T1, T2 and T3.

* $p < 0.05$

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Table 1. Training 1, 2 and 3 progressive overload, total work, training methodology, area per player and rest.

Week No.	T1 Progressive Overload	T1 Total Work	T2 Progressive Overload	T2 Total Work	T3 Progressive Overload	T3 Total Work
Week 1	4 × 3-min	12 min	4 × 40 s	160 s	12 × 30 m	360 m
Week 2	4 × 3.5-min	14 min	5 × 35 s	170 s	12 × 30 m	360 m
Week 3	4 × 4-min	16 min	6 × 35 s	205 s	14 × 25 m	336 m
Week 4	4 × 4.5-min	18 min	8 × 30 s	240 s	14 × 25 m	336 m
Week 5	4 × 5-min	20 min	8 × 30 s	240 s	15 × 20 m	300 m
Week 6	4 × 5.5-min	22 min	10 × 25 s	250 s	15 × 20 m	300 m
Week 7	4 × 6-min	24 min	10 × 25 s	250 s	18 × 15 m	270 m
Week 8	4 × 6.5-min	26 min	14 × 20 s	280 s	18 × 15 m	270 m
Training Methodology	SSGs		SSGs & SST		SST	
Area Per Player	90 m ²		60 m ²		N/A	
SSG Format	3-a-side – 6-a-side		1-a-side – 2-a-side		N/A	
Rest	1:1-0.46		1:2-1:4		1:6-1:8	

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Table 2. Polygenic DNA profile grouping strategy based on power / endurance total genotype score (TGS).

DNA Profile Group	Percentage Threshold (%)	N
Power Group (PG)	51.0-78.3%	15
Endurance Group (EG)	31.9-50.0%	15

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Table 3. List of Genetic Variants Analysed by DNAFit Peak Performance Algorithm™

Gene	Full Name	Proposed Function	Polymorphism	Endurance / Power	Reference
ACE	Angiotensin I converting enzyme	Circulatory homeostasis regulation	Alu I/D (rs4646994)	Endurance: I Power: D	32
ACTN3	α -actinin-3	Stabilization of type II muscle fibre contractile properties	Arg577Ter (rs1815739 C/T)	Endurance: T Power: C	32
ADRB2	β -2 adrenoreceptor	Regulation of central nervous, cardiac, pulmonary, vascular and endocrine systems	Gly16Arg (rs1042713 G/A) Gln27Glu (rs1042714 C/G)	Endurance: A Endurance: C	45
AGT	Angiotensinogen	Determination of blood pressure	Met235Thr (rs699 T/C)	Power: C	16
BDKRB2	Bradykinin receptor B2	Endothelium-dependent vasodilation	Rs1799722 C/T	Endurance: T	40

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COL5A1	Collage, type v, α 1	Supports collagen assembly	Rs12722 C/T	Endurance: T	6,36
CRP	C-reactive protein, pentraxin-related	Elimination of damaged cells within the blood	Rs1205 A/G	Endurance: A	25
GABPB1 (NRF2)	GA binding protein transcription factor, β subunit 1	Supports activation of cytochrome oxidase expression and mitochondrial function	Rs7181866 A/G	Endurance: G	13
IL6	Interleukin-6	Differentiation, proliferation and survival of muscle cells	-174 (rs18007959)	C/G Power: G	37
PPARA	Peroxisome proliferator-activated receptor α	Regulates lipid metabolism, myocardial hypertrophy, glucose homeostasis and mitochondrial biogenesis	rs4253778 G/C	Endurance: G Power: C	29

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PPARAGC1A	Peroxisome proliferator-activated receptor γ coactivator 1 α	Regulation of formation of muscle fibres, fatty acid oxidation, glucose utilization and mitochondrial biogenesis	Gly482Ser (rs8192678 G/A)	Endurance: G	30
TRHR	Thyrotropin-releasing hormone receptor	Supports development of skeletal function through release of thyroxine	rs16892496 A/C	Power: C	28
VDR	Vitamin D receptor	Effects bone and skeletal muscle biology	Bsm1 A/G (rs1544410)	Power: A	44
VEGFA	Vascular endothelial growth factor A	Supports cell growth	Rs2010963 G/C	Endurance: C	2

Adapted from Jones et al. (2015).