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The Predictive Ability of Total Genotype Score and Serum Metabolite Markers in Power-Based Sports Performance Following Different Strength Training Intensities — A Pilot Study

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ABSTRACT

Muscular power is one of the factors that contribute to an athlete's performance. This study aimed to explore the predictive ability of total genotype score (TGS) and serum metabolite markers in power-based sports performance following different strength training (ST) intensities. We recruited 15 novice male field hockey players (age = 16.27 ± .12 years old, body mass index = 22.57 ± 2.21 kg/m^2) and allocated them to; high-intensity strength training (HIST, n=5), moderate intensity strength (MIST, n=5), and control group (C, n=5). Both training groups completed an eight-week ST intervention. Pre- and post-training muscular power (vertical jump) was measured. The participants were genotyped for; *ACE* (rs1799752), *ACTN3* (rs1815739), *ADRB3* (rs4994), *AGT* (rs699), *BDKRB2* (rs1799722), *PPARA* (rs4253778), *PPARGC1A* (rs8192678), *TRHR* (rs7832552), and *VEGF* (rs1870377). TGS was calculated to annotate for strength-power (STP) and endurance (END) qualities. Subsequently, serum metabolomics analysis was conducted using Liquid chromatography-mass spectrometry

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Quadrupole-Time-of-Flight (LC-MS QTOF) to profile differentially expressed metabolite changes induced by training. Multiple regression analysis was conducted to explore the ability of TGS and differentially expressed metabolite markers to predict muscular power changes following the intervention. Multiple Regression revealed that only TGS STP might be a significant predictor of muscular power changes following MIST (adjusted R^2=.906, p<.05). Additionally, ST also resulted in significant muscular power improvement (p<.05) and perturbation of the sphingolipid metabolism pathway (p<.05). Therefore, selected gene variants may influence muscular power. Therefore, STP TGS might be able to predict muscular power changes following MIST.

Keywords: Genetics, metabolomics, single nucleotide polymorphism, strength training, training response

INTRODUCTION

Field hockey is a field-based team sport characterised as a high-intensity intermittent event that places greater demand on athletes' physiological and physical capacities (Lemos et al., 2017; Ibrahim Hassan, 2018). This sport requires the dynamic action of accelerating, decelerating, jumping, and changing direction (Pimenta et al., 2012). Relevant studies demonstrated that it is essential for athletes to have a significant level of muscular strength, power, and cardiorespiratory fitness to perform optimally (Lemos et al., 2017; Bishop et al., 2015; Lemmink & Visscher, 2006; Konarski et al., 2012).

Generally, strength training (ST) is widely used to enhance performance across various sports events (Kikuchi et al., 2019). ST is known to improve muscle energy status, resulting in the ability to maintain greater force output for a longer time (Laursen, 2010). A good design ST program involves the manipulation of training variables (e.g., intensity, volume, frequency, rest interval, exercise selection, and order) (Kikuchi et al., 2019; Mangine et al., 2015). Studies have demonstrated that both high-intensity (HI) and moderate-intensity (MI) ST improved several performance components in intermittent sport (Mangine et al., 2015; Assuncao et al., 2016; Astorino et al., 2004; Christou et al., 2006; Lesinski et al., 2016).

It has been shown that there were associations between gene variants and athletic performance (e.g., muscular strength, muscular power, endurance, and neuromuscular coordination) (Varillas-Delgado et al., 2022; Ahmetov et al., 2016). Gene variants that involve metabolism regulation, muscle fibre type, muscle contraction and circulatory homeostasis in at least two independent studies were selected (Jones et al., 2016; Ahmetov et al., 2016; Egorova et al., 2014). Athletic performance is highly polygenic as it involves complex multiple genes interactions that affect overall outcomes (de la Iglesia et al., 2020). The total genotype score (TGS) was used to quantify the combined influence of multiple gene interactions (Massida et al., 2019). Genes and environment (e.g., training, experience, and diet) are likely to play an important role, but further investigation is required to explore

the contribution of genetic factors toward performance (Kelly et al., 2020; Wishart, 2019; Sarzynski et al., 2016).

ST is an external challenge that leads to differentially expressed metabolites (Kelly et al., 2020). Different ST intensity imposes variable demands on the physiological and physical capacities, shown via changes in the metabolic pathways (Laursen, 2010). Therefore, exploring metabolite profile changes is useful for illustrating an individual's current state and responses to stimuli (Kelly et al., 2020; Wishart, 2019). Metabolic changes have been observed in response to acute ST, but less is known about the ST response following a longer intervention period (Morville et al., 2020). To improve the understanding of metabolite alteration following ST, the employment of the metabolomics approach is deemed essential. Therefore, this study aimed to explore the ability of TGS to predict changes in muscular power and the associated metabolic pathways following different ST intensities being prescribed.

METHOD

Participants and Study Design

A true experimental design was used in the present study. Purposive sampling was employed to recruit fifteen (N=15) male field hockey players who represented their district, aged between 16 to 17 years old. The study was conducted during the off-season and commenced in October 2018. The participants were randomly assigned to; high-intensity strength training (HIST, n=5), moderate-intensity strength training (MIST, n=5), and control group (C, n=5). Study inclusion criteria were that participants; were healthy and active field hockey players, who had participated in competitive tournaments, free from any injuries for the past six months, had not performed strength training at least six months prior to participation, and had not taken any supplementation prior to participation. Participants were informed about the study procedure and potential risks associated with the study, and informed consent was obtained before participation. In addition, participants completed the PAR-Q^+ questionnaire as a pre-screening and Electrocardiogram (ECG) test (Tanisawa et al., 2020). The study was approved by Universiti Teknologi MARA Research Ethics Committee [ref no.:600-IRMI(5/1/6)]. A flow chart of the study design is presented in Figure 1.

Muscular Power Measurement

Pre- and post-test muscular power was measured by vertical jump (VJ) (Yingling et al., 2018). Participants were to perform the jump with a pause at >90° squat before jumping with the dominant hand reaching for the swivel vane on a vertec apparatus (Yingling et al., 2018). Three jump attempts were carried out with one minute of rest in between

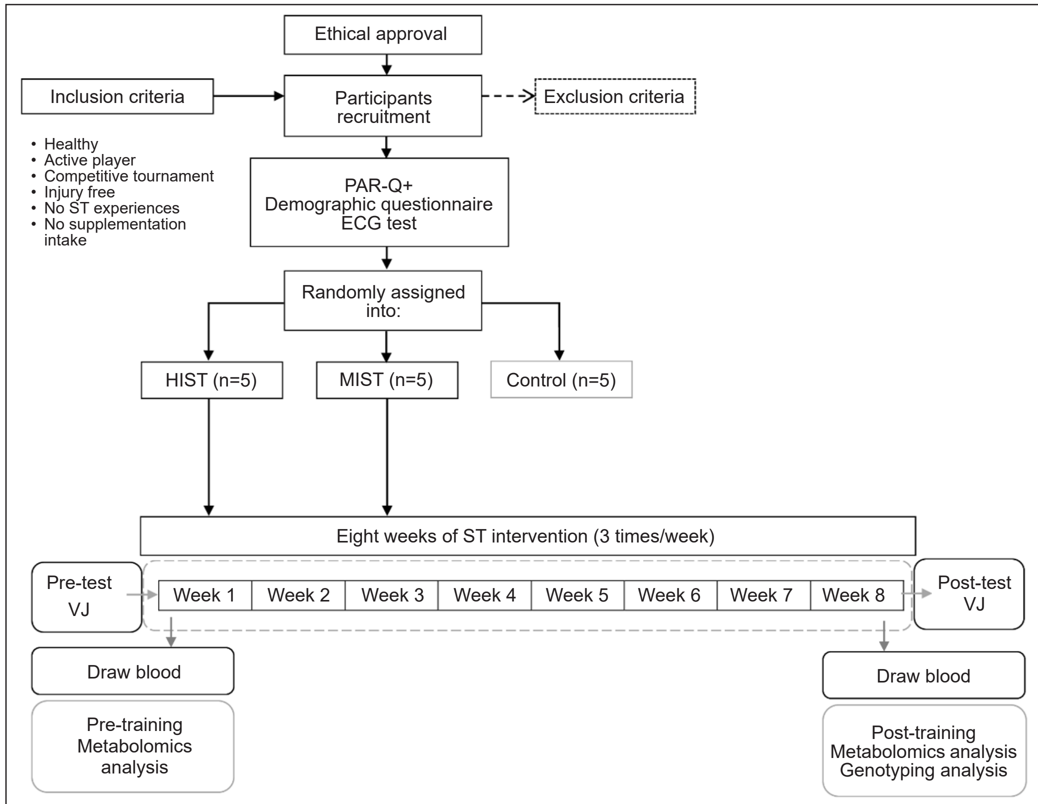

Figure 1. Study design flow chart

jumps. The best jump was equated to the equation (Sayers et al., 1999). The procedure was explained and demonstrated to the participants. Proper stretching, warm-up, and cooling-down sessions were conducted prior to the test. In addition, a familiarisation session was provided before the actual measurement.

Strength Training Intervention

The training was held three times per week for eight weeks. Training volume was pre-determined and equated (Klemp et al., 2016). Familiarisation was designed to ensure correct lifting techniques. The upper body (biceps curl, triceps extension, shoulder press) and lower body (leg press, hamstring curl, calf raise) exercises were prescribed to the participants. The HIST performed three sets of one to six repetitions at 80 to 90% 1-RM, while the MIST performed three sets of eight to 12 repetitions at 60 to 75% 1-RM. The warm-up was conducted using the peck deck machine (upper body) and leg press machine (lower body) at 50% of 1RM for 10 repetitions. Participants in the training groups were advised to refrain from additional ST sessions and maintain their habitual physical activity and dietary intake. In contrast, the control group maintained their usual activities without engaging in ST sessions.

Genotyping

Deoxyribonucleic Acid (DNA) Extraction. DNA was extracted using an in-house modified conventional DNA extraction method. Five (5 ml) of 1 × lysis buffer (0.64 M sucrose, 0.02 M Tris hydrochloric acid, 2% Triton X-100, autoclaved Mili-Q water) were added to 5 ml of blood sample and inverted (10 times). Later, another 5 ml of 0.5 × lysis buffer was added to the blood sample and inverted (10 times), left on ice (10 minutes), and later centrifuged (Eppendorf, model 5810R, Hamburg, Germany) at 2,700 × g, 20°C for 15 minutes. Next, the pellet was rinsed with 25 ml of Tris Ethylenediaminetetraacetic acid (EDTA) (pH 8.0) buffer followed by centrifugation at 2,700 × g, 20°C for 15 minutes. The supernatant was discarded while the pellet was kept. The steps above were repeated three times until a clear supernatant and pellet were formed. The pellet was re-suspended with 2 ml saline EDTA (pH 8.0) and incubated overnight in a water bath at 37°C with 100 µl of 20% Sodium Dodecyl Sulfate (SDS) and 10 µl proteinase-K solution (20 mg/ml). This step was taken to lyse the cell and nuclear membrane while maintaining DNA's integrity. Then, 100 µl of 2.0 M Potassium Chloride (KCl) was added, followed by 4 ml of cold 100% ethanol. The step was taken to precipitate the DNA. DNA became visible as a floating strand in the solution. Then, 700 µl of cold 70% ethanol was used to rinse the DNA by allowing excess KCl to dissolve. The DNA was left to dry. The DNA was reconstituted with Tris EDTA buffer and stored at -20°C. The DNA's concentration and purity were measured using NanoDrop 2000 Spectrophotometer (Thermo Scientific, Wilmington, USA).

Allele-Specific Polymerase Chain Reaction (ASPCR). In-house genotyping procedures were conducted using ASPCR assays. The primers were designed to target specific single nucleotide polymorphisms (SNPs). ASPCR was conducted using a thermal cycler (Takara Bio, CA, USA) with the final mixture volume of 20 µl, containing the participant's DNA (100 ng/µl), 0.5 U/µl of *taq* DNA polymerase (NEB, MA, USA), various concentrations of primers (0.1–2.0 µM), 0.16 mM deoxyribonucleotide triphosphate (dNTPs) and autoclaved Mili-Q water. A touchdown thermal cycle condition was composed of; pre-denaturation at 95°C (2 minutes), denaturation at 95°C (30 seconds), and extension at 68°C (30 seconds). In the initial 10 cycles of annealing, the temperature was gradually decreased (-1°C) from 65°C to 55°C (30 seconds). In the latter half (30 cycles), the temperature was maintained at 55°C (30 seconds). Finally, post-extension was set at 68°C (5 minutes). PCR products were electrophoresed at 200 V for 60 minutes and examined on 3.5% agarose gel stained with Ethidium Bromide (EtBr). The gel was visualised under ultraviolet light to detect the specific band sizes that indicate the alleles.

Total Genotype Score (TGS) Calculation. The TGS was calculated using an algorithm that incorporated all genotype scoring (GS) in a simple additive model, TGS = 100 / (9 ÷

2) × $GS_1 + GS_2 + \ldots + GS_9$ (Ruiz et al., 2009; Ruiz et al., 2010). Next, the homozygous, intermediate 'heterozygous' and 'less optimal' homozygous were assigned with the scoring of 2, 1, and 0, respectively. Finally, the total score was calculated and associated with strength-power (STP) and endurance (END) qualities (Table 1).

Table 1
Genotype scoring

Variables	Genotype scoring (GS)	Qualities
Genes		
ACE	DD=2, ID=1, II=0	STP
	DD=0, ID=1, II=2	END
ACTN3	CC=2, CT=1, TT=0	STP
	CC=0, CT=1, TT=2	END
PPARA	CC=2, CG=1, GG=0	STP
	GG=2, CG=1, CC=0	END
ADRB3	AA=2, AG=1, GG=0	END
BDKRB2	TT=2, TC=1, CC=0	END
VEGF	CC=2, CG=1, GG=0	END
PPARGC1A	GG=2, AG=1, AA=0	END
AGT	TT=2, TC=1, CC=0	STP
TRHR	CC=2, AC=1, AA=0	STP

Abbreviation: END=endurance, STP=strength-power

Metabolomics

Serum Sample Collection and Preparation. Ten (10) ml of blood samples were drawn pre-and post-training. It was collected in a serum separator tube. Serum samples were prepared by centrifugation (ALC, model PK 121R, Turin, Italy) at 4,600 × g, 4°C, 15 minutes, and stored at -80°C until analysis. Prior to the analysis, 150 µl of double distilled water (ddH_2O) was added to 150 µl of aliquoted serum. In order to precipitate the protein, 450 µl of cold Acetonitrile (Merck, Darmstadt, Germany) was added, followed by 30 seconds of vortex mixing and centrifuged (ALC, model PK 121R, Turin, Italy) at 10,600 × g, 4°C, 10 minutes. Then, 650 µl of supernatant was transferred to a new microcentrifuge tube. This step was repeated two times and dried in the vacuum concentrator (Eppendorf, model 5301, Hamburg, Germany). Quality control (QC) was prepared from pooled serum samples. Each QC sample was analysed independently with each batch of samples. The performance of QC was evaluated by the determination of relative standard deviation (RSD).

LC-MS Conditions and Analysis. The samples were analysed using LC/MS (Agilent Technologies, model 6250, CA, USA). The column used was the ZORBAX Eclipse Plus C18, 100 mm × 2.10 mm × 1.80 µm (Agilent Technologies, CA, USA) with electrospray ionisation (ESI) in positive mode. The drying gas temperature was set at 300°C with a flow rate of 3.0 L/min. The nebuliser pressure was set at 15 psi, and the fragmentor voltage was set at 175 V. The temperature was maintained at 40°C and the total run time was 48 minutes. The system was operated at a 0.25 ml/min flow rate with mobile phase A (ddH_20 with 0.1% Formic acid) and B (Acetonitrile with 0.1% Formic acid) over a gradient of 0 to 36 minutes with an increasing percentage of B from 5 to 95%. The dried sample was reconstituted with 30 µl of mobile phases (15 µl from mobile phase A and 15 µl from mobile phase B) followed by 30 seconds of vortex mixing and centrifuged at 16,400 × g, 4°C, 10 minutes. Four randomly selected samples, one QC and one blank, were analysed per

batch. Twenty (20) µl from each sample was transferred into inserts and injected into the LC-MS for further analysis. The analysis was performed in four replicates for each sample.

Untargeted Metabolomics Data Processing. The raw data (.d) from the LC-MS QTOF analysis were collected using the Agilent MassHunter Data Acquisition software version B.05.00 (Agilent Technologies, CA, USA) and processed in the Agilent MassHunter Qualitative Analysis software version B.05.00 (Agilent Technologies, CA, USA). Data was converted from (.d) to (.cef) using DA Reprocessor software (Agilent Technologies, CA, USA) and transferred into Agilent MassHunter Profiler Professional (MPP) software version B.12.01 (Agilent Technologies, CA, USA). MPP software was used to conduct differential analyses of the entities. Data were subjected to normalisation, filtration, and recursion analysis. MetaboAnalyst 5.0 (https://dev.metaboanalyst.ca) was used to conduct a multivariate analysis. The principal component analysis method (PCA) was used to visualise the clustering of metabolites following the intervention. Later, pathway analysis was conducted to identify the significant pathway involved following the intervention. Then, receiver operating characteristic (ROC) curve analysis was conducted on the identified significant metabolites. The metabolites with an area under the curve (AUC) value of >.65 and the Variable Influence on Projection (VIP) index (>1.00) were used to screen significant metabolites.

Statistical Analysis

Statistical analysis was performed using SPSS for Windows version 26.0. Statistical significance was set at ($p<.05$). Data are presented in mean ± standard deviation (M±S.D) unless otherwise stated. Muscular power data were normally distributed (Shapiro-Wilk test, $p>.05$). Differences in TGS were analysed using the independent sample t-test, whilst muscular power changes following intervention were analysed using paired sample t-test. Multiple regression was conducted to assess the ability of TGS and serum metabolite markers to predict muscular power changes following ST intervention.

RESULTS

Demographic and TGS

The participants were homogenous at baseline. There were no significant differences between training groups: age (HIST = 16.00 ± .00, MIST = 16.40 ± .55, C = 16.40 ± .55 years old), height (HIST = 1.67 ± .04, MIST = 1.60 ± .06, C = 1.70 ± .08 m), body weight (HIST = 67.70 ± .04, MIST = 57.98 ± 8.65, C = 61.24 ± 5.21 kg) and body mass index (HIST = 24.11 ± 1.96, MIST = 22.48 ± 2.72, C = 21.12 ± .48 kg/m^2). All participants completed the training program with no missing sessions. No injuries were reported in the study. There were no statistically significant muscular power differences between training groups. Most

participants presented with heterozygous genotypes except for *PPARA* GG = 100% and *ADRB3* AA = 100%, which were homozygous genotypes. There was a significant difference (p<.05) in the TGS STP across training groups, whereby MIST (M=22.24) is higher as compared to HIST (M=17.79) and C (M=13.34). There was no significant difference in TGS END across training groups. The TGS is presented in Table 2.

Table 2
TGS between training groups

Variables	HIST (M±S.D)	MIST (M±S.D)	C (M±S.D)	*p*-value
TGS				
STP	17.79 ± 4.65	22.24 ± 3.93	13.34 ± 4.97	.03*
END	50.04 ± 6.81	48.93 ± 4.65	54.49 ± 10.69	.51

*significant different (p<.05), Abbreviation: END=endurance, STP=strength-power

Muscular Power Changes Between Training Groups

There was a significant muscular power improvement (p<.05) in both training groups, HIST (M_{diff}=.84 kW) and MIST (M_{diff}=.41 kW), pre-and post-training. The muscular power changes are presented in Table 3.

Table 3
Muscular power changes between training groups

Variables	n	Pre-test (M±S.D)	Post-test (M±S.D)	M_{diff}	*p*-value
LB muscular power (kW)					
HIST	5	4.30 ± .61	5.14 ± .55	.84	.00*
MIST	5	3.65 ± 1.06	4.06 ± 1.09	.41	.00*
C	5	3.71 ± .46	3.56 ± .47	.15	.00*

*significant different (p<.05), Abbreviation: M_{diff}=mean difference

Serum Metabolite Analysis Between Training Groups

Data Acquisition and Processing of Metabolite Profile. Total ion chromatograms (TIC) for the analysis of the serum samples representing the HI, MI and control groups are shown in Figures 2, 3 and 4, respectively. Analytical reproducibility between batches was within the acceptable range of RSD (<20%), as shown in Table 4.

Serum Metabolomics Profile and Biomarker Identification. LC-MS analysis revealed that a total of 941 metabolites were detected, and about 224 differed significantly between training groups and post-tests. The PCA score plot between training groups demonstrated a clear separation between the training and control groups (Figure 5). The pathway analysis revealed about 35 pathways involved in differentiating the effect of strength training intensity. However, only the Sphingolipid metabolism pathway was significantly

Table 4
Relative standard deviation (RSD) values of selected mass in the pooled sample between batches

Compound	Mass different (ppm)			Retention time (mins)		
	Mean	S.D	RSD (%)	Mean	S.D	RSD (%)
2	199.15	28.53	14.32	.96	.01	.88
3	140.36	22.56	16.07	.98	.03	2.85
4	142.28	26.56	18.66	1.02	.04	3.90
9	161.64	23.20	14.35	1.57	.26	16.55
36	524.46	89.06	16.98	22.72	2.14	9.43
37	504.87	79.02	15.65	23.12	1.94	8.39
40	513.95	86.28	16.78	24.21	2.10	8.68

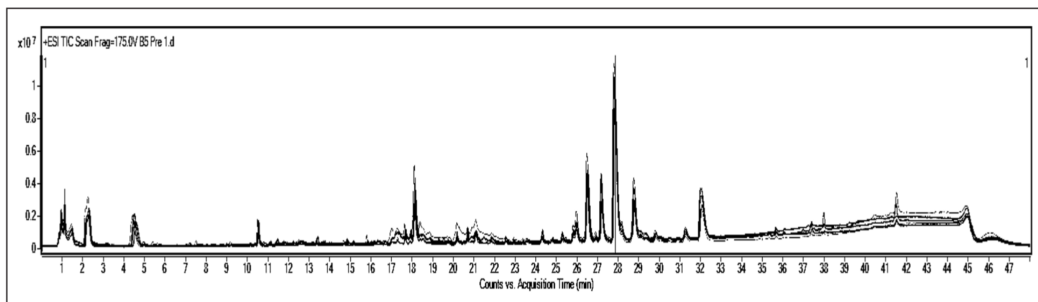

Figure 2. TIC in the HI ST group

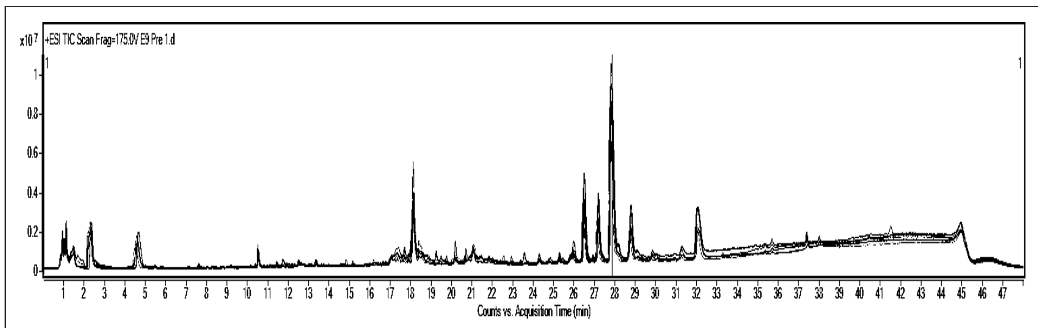

Figure 3. TIC in MI ST group

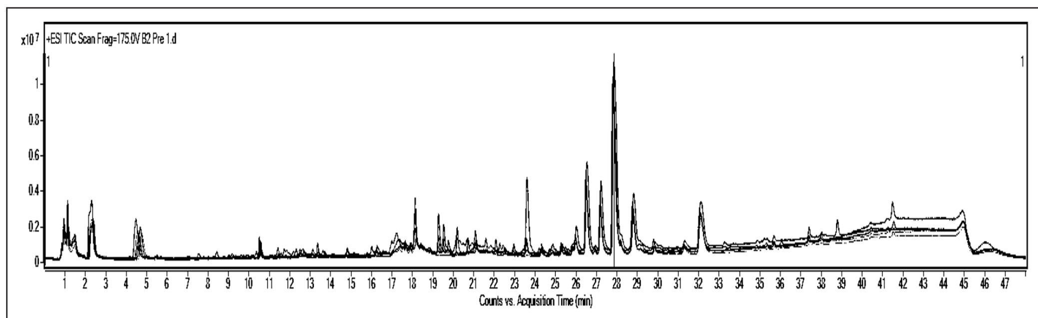

Figure 4. TIC in the control group

different (p<.05) across training groups. Presently, only three metabolites (AUC>.65, VIP≥1.0, p<.05) (Figures 6, 7, and 8) belong to the Sphingolipid pathway, which is the most perturbated pathway (Table 5). There is a pattern of differences in Sphingolipid metabolite between strength-trained participants and their healthy non-trained counterparts.

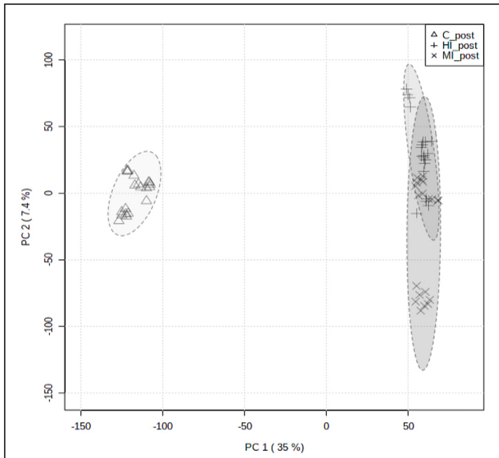

Figure 5. PCA score plot across training groups

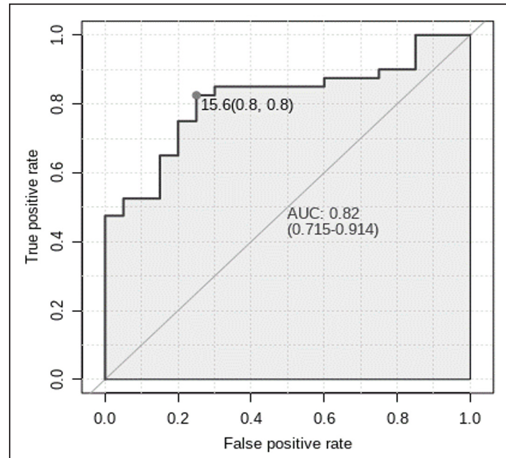

Figure 6. ROC curve of 3-O-Sulfogalactosylceramide

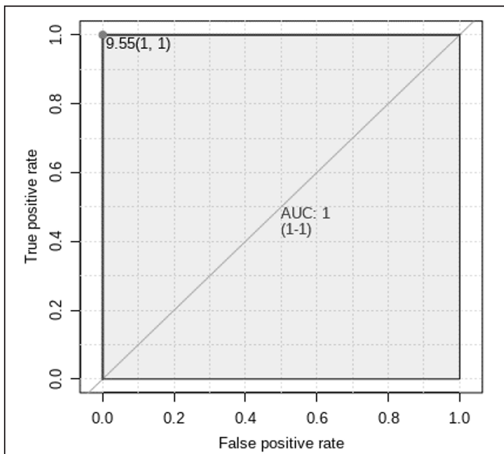

Figure 7. ROC curve of Sphingosine-1-phosphate

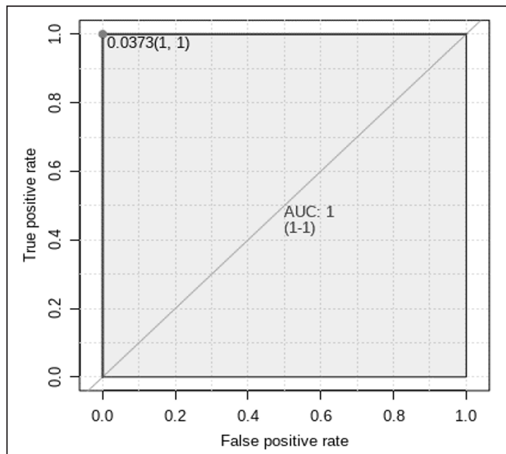

Figure 8. ROC curve of Sphinganine

Table 5
Potential serum metabolite markers between training groups

Variables	AUC	VIP	FC	p-value
Metabolites				
3-O-Sulfogalactosylceramide	.82	1.08	1.17	<.05*
Sphingosine-1-phosphate	1.00	1.90	-2.43	<.05*
Sphinganine	1.00	1.23	-9.28	<.05*

*significant different (p<.05)

Regression Analysis Between Predictors and Muscular Power Changes Between Training Groups

There is a significant regression (adjusted R^2 =.91) of TGS STP with muscular power changes following MI ST, F(2,2)=20.30, *p*<.05. The predicted muscular power is equal to .42–.01 (TGS STP) + .00 (TGS END). Other predictors did not significantly predict muscular power changes. The regression coefficient is presented in Table 6.

Table 6
The regression coefficient of predictors on muscular power changes between training groups

Variables	*B*	*S.E*	*t*	*p*-value	95% CI
LB muscular power (kW)					
HI (n=5)					
TGS					
Constant	1.78	.39	4.51	.05	.08, 3.48
STP	-.00	.01	-.35	.76	-.04, .04
END	-.02	.01	-2.77	.11	-.05, .01
Biomarkers					
Constant	.85	.11	7.50	.08	-.59, 2.29
3-O-Sulfogalactosylceramide	.03	.07	.46	.72	-.85, .91
Sphingosine-1-phosphate	-.39	.93	-.42	.75	-12.23, 11.45
Sphinganine	.01	.01	.58	.67	-.17, .19
MI (n=5)					
TGS					
Constant	.42	.08	5.58	.03	.10, .74
STP	-.01	.00	-6.34	.02*	-.02, -.00
END	.01	.00	3.22	.09	-.00, .01
Biomarkers					
Constant	.45	.06	7.09	.09	-.35, 1.25
3-O-Sulfogalactosylceramide	-.00	.01	-.15	.91	-.08, .08
Sphingosine-1-phosphate	.12	.12	1.01	.50	-1.38, 1.62
Sphinganine	.01	.01	.71	.61	-.11, .12
C (n=5)					
TGS					
Constant	-.25	.58	-.43	.71	-2.77, 2.26
STP	.01	.02	.59	.62	-.06, .08
END	.00	.01	-.05	.97	-.02, .03
Biomarkers					
Constant	-.23	.02	-10.77	.01	-.33, -.14
3-O-Sulfogalactosylceramide	.01	.00	3.07	.09	-.00, .02
Sphinganine	.01	.00	3.62	.07	-.00, .03

*significant different (*p*<.05), Abbreviation: END=endurance, STP=strength-power

DISCUSSION

The study aimed to determine the predictive ability of TGS and serum metabolite marker power-based sports performance following different strength training (ST) intensities. Most of the participants harboured endurance-related genotypes, and Amato et al. (2018) demonstrated similar findings whereby their professional Italian soccer athletes were more

endurance-oriented (TGS=56.44%) as compared to strength (TGS=43.52%). However, the Russian professional soccer players presented with higher strength TGS (M=52.00) as compared to healthy control (M=41.30) (Egorova et al., 2014). The present study demonstrated that only TGS STP might significantly predict muscular power changes following MIST. Murtagh et al. (2020) also demonstrated similar findings among elite soccer players who were more strength-power oriented than control. Although the endurance component dominates energy delivery during the match, the strength-power component plays a greater role in determining success during events in intermittent sports (e.g., winning ball possession, scoring, conceding goals) (Egorova et al., 2014; Lemmink & Visscher, 2006).

Suraci et al. (2021) demonstrated that the mean percentage change for power performance in power TGS was higher compared to the endurance TGS following eight weeks of soccer-specific training compared to the small-sided game and a combination of both among adolescent soccer players. Pickering et al. (2019) demonstrated a greater endurance improvement in participants who presented with greater TGS endurance following aerobic training. The inter-individual variation in response to training must be considered, as some participants might experience greater improvement than others with the same intensity being prescribed (Pickering et al., 2019).

In addition, participants also demonstrated significant improvement in muscular power following HIST and MIST interventions. McKinlay et al. (2018) also demonstrated a similar finding whereby eight weeks of strength and plyometric training among novice adolescent soccer players significantly improved muscle strength and power performance. Earlier, Juarez et al. (2009) also demonstrated similar findings as their male habitually active collegiate athletes presented with power performance improvement (12.3%) following eight weeks of a conventional strength training program. Novice athletes typically possessed a significant reserve of potential improvement regardless of the training they were prescribed (Juarez et al., 2009). Wetmore et al. (2020) also agreed that untrained participants required lesser stimulus than previously trained participants.

Th three serum metabolite markers which were significant in this study could not predict muscular power changes following ST intervention. However, there was a significant alteration of sphingolipid metabolites following different ST interventions. Participants in HIST showed an increment in ceramides metabolites post-training. The sphingolipids are a family of lipid molecules that circulate in the serum and accumulate around the skeletal muscle (Bergman et al., 2015). Skeletal muscle is well-known for sphingolipid metabolism (Nikolova-Karakashian & Reid, 2011). Recently, Nikolova-Karakashian and Reid (2011) explained the ‘sphingolipid rheostat’ mechanism whereby sphingosine-1-phosphate slows fatigue, preserving force over time, ceramides, on the other hand, promote fatigue. The findings of Shepherd et al. (2014) and Sarin et al. (2019)

were contradictory to the present study as they demonstrated that ST led to a significant increment in intramuscular triglyceride (IMTG) storage, muscle strength, and body fat percentage reduction. Shepherd et al. (2014) assumed that greater IMTG storage might be created, leading to low availability of circulating ceramides following the ST.

It is well known that muscular strength and power are partly responsible for rapid movements such as sprinting and accelerating (Kobal et al., 2017). This study emphasises that effectively prescribing HIST and MIST developed muscular power, especially in novice field hockey players. Moreover, identifying an individual's TGS might be useful in predicting response to training. An individual with higher strength-power or endurance TGS could be given appropriate training to maximise training adaptation. The combination of a favourable genetic profile with appropriate training prescription is advantageous to novice field hockey players.

Although the current study yielded some useful findings, there is a limitation that should be taken into consideration. The sample size in this study is relatively small for a gene-metabolite study. Further larger studies should be conducted to obtain a sufficient sample size for a more accurate result. Despite the limitations, the results remain valid and applicable since the training intervention was standardised, and participants in each group were homogenous in terms of age, sex, training experience, training volume, and baseline muscular power.

CONCLUSION

This study improves the understanding of the predictive ability of TGS and serum metabolite markers in determining muscular power changes following ST among novice athletes. It is concluded that STP TGS may influence muscular power changes following MIST in novice field hockey players. Selected gene variants may potentially influence muscular power following strength training. Overall, the result of the current study could potentially represent the tentative steps toward understanding the application of genomic and metabolomic in a sport that improves overall sports performance.

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