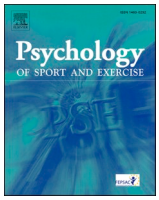
Psychology of Sport & Exercise 61 (2022) 102209

Contents lists available at ScienceDirect

Psychology of Sport & Exercise

journal homepage: www.elsevier.com/locate/psychsport



Genetic associations with personality and mental toughness profiles of English academy football players: An exploratory study

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ARTICLE INFO

Keywords:
Athlete development
Genomics
Polygenic profile
Psychology
Soccer

ABSTRACT

Psychological characteristics influence the performance of youth football players and are significant predictors of development and success at adulthood. Although genetic factors may explain a considerable portion of inter-individual differences in psychological traits, psychogenetic research in football is scarce. As such, the purpose of this study was to examine the association of ten single nucleotide polymorphisms (SNPs) with personality and mental toughness profiles of academy football players. Seventy-three male under-12 to under-18 football players from a Category 3 English academy were genotyped for ten SNPs. Personality and mental toughness were assessed using a 50-item IPIP Big Five personality traits questionnaire and the Mental Toughness Index, respectively. Simple linear regression was used to analyse individual SNP associations with personality dimensions and mental toughness, whereas both unweighted and weighted total genotype scores (TGSs; TWGSs) were computed to measure the combined influence of all SNPs. There was a significant association between *DRD3* (rs167771) and agreeableness ($p = .043$), where A/A homozygotes scored higher than G allele carriers. TGSs and/or TWGSs were significantly correlated with mental toughness and each personality dimension except openness, explaining between 3 and 17% of the variance. The results of this study suggest psychological characteristics of youth football players are partly determined by genetic factors.

1. Introduction

Psychological characteristics are now an integral component of multidimensional athlete development models in football (soccer) (Vaeyens et al., 2008; Williams et al., 2020). Early research in football indicated higher performing youth football players possessed superior psychological capacities that facilitated greater development than their lower performing counterparts (Williams & Reilly, 2000). Psychological attributes have been researched frequently over the last two decades and have not only been regularly associated with current performance levels in youth football players, but are also significant predictors of development and success in adulthood (Murr et al., 2018). As a result, psychological aspects have been integrated into many countries' multidisciplinary long-term youth development strategies (e.g., the Elite Player Performance Plan in England; Premier League, 2013). However, at present, research on the psychological aspects of performance in football is scarce compared to other multidimensional components, such as physiological and technical (Sarmento et al., 2018).

Psychological research in football has identified a wide range of important psychological characteristics, which have been associated with objective performance metrics as well as through subjective coach, player, and scout perceptions (Williams et al., 2020). The predominant method of assessing psychological characteristics has been through self-report validated questionnaires/scales (Musculus & Lobinger, 2018). Several recent systematic reviews on football-specific research have synthesised the findings of these studies from both psychological (e.g., Gledhill et al., 2017; Murr et al., 2018; Ivarsson et al., 2020) and multidisciplinary (e.g., Bergkamp et al., 2019; Sarmento et al., 2018; Williams et al., 2020) perspectives. Overall, and similar to other multidimensional predictors, studies have generally reported small to moderate effect sizes for psychological variables on current and future performance in football. Some of the more strongly supported

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https://doi.org/10.1016/j.psychsport.2022.102209
Received 1 February 2022; Received in revised form 25 March 2022; Accepted 22 April 2022
Available online 28 April 2022

psychological variables include: (a) achievement goal orientation, (b) achievement motives, (c) commitment, (d) concentration, (e) coping strategies, (f) effort, (g) motivation, (h) resilience, (i) self-determination, (j) self-regulation, and (k) task orientation (Murr et al., 2018; Ivarsson et al., 2020).

In psychological research, there are umbrella terms which encompass many of the psychological variables outlined, such as personality and mental toughness (Lin et al., 2017). Personality is most commonly assessed in contemporary sport research using the Big Five/Five Factor model, which contends that there are five main personality dimensions (i.e., extraversion, agreeableness, openness, conscientiousness, and neuroticism) that embody several more specific psychological facets (McCrae & John, 1992). More specifically, extraversion assesses the quantity and intensity of interpersonal interactions, agreeableness assesses individuals' concern for cooperation and social harmony, openness assesses individuals' tendency to seek out new experiences, conscientiousness assesses organisation and goal-directed behaviour, and neuroticism assesses the degree to which individuals are prone to emotional instability (Allen et al., 2013). Mental toughness on the other hand has been defined as a collection of psychological resources that facilitate an individual's capacity to produce reliable objective and subjective performance in the presence of varying situational demands (Gucciardi et al., 2015). Unsurprisingly, several studies have reported significant associations between personality traits, mental toughness, and performance in sport (Allen et al., 2013; Liew et al., 2019). In general, more successful athletes tend to score lower on neuroticism and higher on mental toughness (Benítez-Sillero et al., 2021; Piepiora, 2021; Steca et al., 2018).

To assess the underpinning mechanisms of psychological variables, behavioural scientists have investigated the extent to which genetic and environmental factors influence inter-individual differences (Chabris et al., 2015). These studies identified a sizable genetic component in both personality and mental toughness (Allen et al., 2013; Lin et al., 2017; Power & Pluess, 2015). Specifically, the heritability estimate for overall personality and mental toughness is approximately 50%, with the sub-components of each generally ranging from 35% to 65% (Horsburgh et al., 2009). There is considerable evidence supporting the influence of genetics on psychological variables. For example, Turkheimer (2000) noted in a seminal article that the "Three Laws of Behaviour Genetics" are: (a) all human behavioural traits are heritable, (b) the effect of being raised in the same family is smaller than the effect of genes, and (c) a substantial portion of the variation in complex human behavioural traits is not accounted for by the effects of genes or families. These suggestions are further supported by a meta-analysis encompassing 50 years of twin research reporting an average heritability estimate across all behavioural and physical traits of 49% (Polderman et al., 2015).

Grounded on the amalgamation of a vast amount of empirical evidence from molecular genetic research, a "Fourth Law of Behaviour Genetics" was proposed by Chabris et al. (2015). Namely, a typical human behavioural trait is associated with very many genetic variants, each of which accounts for a very small percentage of the behavioural variability. As such, analogous to physical phenotypes, the genetic architecture of psychological phenotypes is highly polygenic (i.e., many different genetic variants influence each trait) and pleiotropic (i.e., each genetic variant influences many different traits). Although, in comparison to physical phenotypes, psychological phenotypes may be *more* polygenic based on the number and mean effect sizes of the associated genetic variants identified in contemporary research (Chabris et al., 2015). Most of these common genetic variants (i.e., polymorphisms) are within genes in the serotonergic and dopaminergic systems (Ausmees et al., 2021; Balestri et al., 2014). However, whilst there is considerable research on these and other variants, the validity of their associations with psychological traits requires further research (KarlssonLinnér et al., 2019; Sanchez-Roige et al., 2018; Strawbridge et al., 2018).

Within a football-specific context, there is very limited psychogenetic research. For instance, a recent systematic review of genetic association research in football only identified three studies involving football players (see McAuley, Hughes, et al., 2021a). These studies reported significant genetic associations with overall athlete status (Filonzi et al., 2015) and specific psychological phenotypes such as anxiety, depression, impulse control, and neuroticism (Cochrane et al., 2018; Petito et al., 2016). However, the cohorts under investigation in these studies consisted of a combination of footballers and athletes from other sports, which limits the implications of their findings to a football-specific context. To the authors' knowledge, there is yet to be a psychogenetic study on footballers in isolation. Accordingly, the aim of this exploratory study was to examine the associations amongst ten psychogenetic single nucleotide polymorphisms (SNPs) with personality and mental toughness profiles of academy football players. Identifying a panel of SNPs associated with relevant psychological phenotypes may enhance athlete development processes in football by enabling more individualised psychological intervention programmes, which may help manage athlete welfare and improve long-term performance. Based on prior work in this area, our hypotheses were: (a) in isolation the individual SNPs will not have large enough effects to produce significant associations, and (b) the collective influence of all SNPs will be associated with some personality dimensions and/or mental toughness.

2. Methods

2.1. Participants

In total, 73 male under-12 to under-18 (aged 14.31 ± 2.16 years) football players from a Category 3 English academy participated in this study. Prior to the study commencing, informed assent from all players, consent from parents/guardians, and gatekeeper consent from each academy was collected. All experimental procedures were conducted in accordance with the guidelines in the Declaration of Helsinki and ethical approval was granted by the corresponding author's institutional Ethics Committee. This study was conducted in accordance with the recommendations for reporting the results of genetic association studies defined by the STrengthening the REporting of Genetic Association studies (STREGA) Statement (Little et al., 2009).

2.2. Personality

Personality was assessed using a 50-item Big Five personality traits questionnaire from the International Personality Item Pool (IPIP) (Goldberg et al., 2006). Each personality trait was measured by the sum of each participant's answers to ten statements using a five-point Likert scale ranging from one (disagree) to five (agree). For example, 'I am the life of the party' (extraversion), 'I am interested in people' (agreeableness), 'I am full of ideas' (openness), 'I am always prepared' (conscientiousness), and 'I get stressed out easily' (neuroticism). Higher scores represent higher levels of each personality trait. Previous studies that applied this tool have reported good internal consistency (Cronbach's alpha) across all trait measurements (i.e., extraversion = 0.87, agreeableness = 0.81, openness = 0.78, conscientiousness = 0.77, and neuroticism = 0.88) (Power & Pluess, 2015).

2.3. Mental toughness

Mental toughness was assessed using the Mental Toughness Index (MTI) (Gucciardi et al., 2015). The MTI is an eight-item unidimensional measure which instructs participants to indicate how they typically think, feel, and behave as an athlete using a seven-point Likert scale ranging from one (False, 100% of the time) to seven (True, 100% of the time) (Cooper et al., 2021; Jones & Parker, 2019). The sum of the eight items yields a mental toughness score that is then used for analysis, with higher scores representing higher levels of mental toughness. Previous

studies examining the internal consistency and reliability of the MTI have demonstrated both high Cronbach's alpha (0.90) and composite reliability (0.90) levels (Jones & Parker, 2018).

2.4. Genetic procedures

2.4.1. Genotyping

Saliva was collected from players via sterile, self-administered buccal swabs, following a minimum of 30 min since food or drink ingestion. Saliva samples were safely stored at room temperature and within 36 h were sent to AKESOgen, Inc. (Peachtree Corners, GA, USA) for DNA extraction. Using Qiagen chemistry, DNA was extracted on an automated Kingfisher FLEX instrument (Thermo Fisher Scientific, Waltham, MA, US). The manufacturers recommended guidelines and procedures were followed throughout. To measure the extracted DNA's quality and quantity, PicoGreen and Nanodrop measurements were taken. Input to the custom testing array occurs at 200 ng in 20 μL. Biomek FXP was used to perform amplification, fragmentation, and resuspension. Hybridisation was performed in a Binder oven at 48° for 24 h and GeneTitan instrumentation (Thermo Fisher Scientific, Waltham, MA, US) was used to stain and scan the arrays, following the Affymetrix Axiom high throughput 2.0 protocol. Data analysis was then performed using a raw CEL file data input into the Affymetrix Axiom Analysis Suite (Affymetrix, Santa Clara, CA, US). Procedures are in accordance with Pickering et al. (2019).

2.4.2. Polymorphism selection

To identify potentially associated polymorphisms, empirical research, review articles, book chapters, and the GWAS catalog (https://www.ebi.ac.uk/gwas/) were examined. Priority was given to GWAS results that were replicated in independent cohorts, followed by candidate gene studies with large homogenous sample sizes which produced similar associations in more than one study. The polymorphisms that were finally included were dependent on the coverage of the microarray and quality control procedures. After an extensive search of the literature, the following ten polymorphisms were selected based on their proposed biological function and relevant associations in previous studies: 5-hydroxytryptamine receptor 2A (*HTR2A*; rs6311), Brain derived neurotrophic factor (*BDNF*; rs6265), Cholinergic receptor muscarinic 2 (*CHRM2*; rs1824024), Catechol-O-methyltransferase (*COMT*; rs4680), Catenin alpha 2 (*CTNNA2*; rs7600563), Dopamine receptor D2 (*DRD2*; rs1800497), Dopamine receptor D3 (*DRD3*; rs167771), Dopamine receptor D4 (*DRD4*; rs1800955), Gamma-aminobutyric acid type A receptor subunit alpha6 (*GABRA6*; rs3219151), and Oxytocin receptor (*OXTR*; rs2254295) (see Table 1). These gene names and symbols are in accordance with those officially approved by the Human Gene Nomenclature Committee (HGNC; https://www.genenames.org). Standard genomic quality control procedures and thresholds were applied when selecting polymorphisms: SNP call rate (>95), sample call rate (>95), fisher's linear discriminant (>3.6), and minor allele frequency (>0.05).

2.4.3. Total genotype score

Unweighted and weighted total genotype scores (TGS; TWGS) were calculated to assess the combined influence of the included SNPs on each personality dimension and mental toughness. Both TGSs and TWGSs have demonstrated sufficient discriminatory power in previous sport genomic research (Massidda et al., 2014; Varillas Delgado et al., 2020; Williams & Folland, 2008). To generate both the TGS and TWGS, each genotype of a respective SNP initially received a score between 0 and 2 based on the observed associations with a dependent variable. Genotypes of dominant (AA vs. Aa-aa) and recessive (AA-Aa vs. aa) models were assigned a score of two (i.e., associated genotype[s]) or zero (i.e., alternate genotype[s]), whereas genotypes of co-dominant models (AA vs. Aa vs. aa) were assigned three scores (i.e., homozygous-associated genotypes received a score of two, the heterozygote received a score of one, and the alternate homozygous genotype received a score of zero) (Guilherme & Lancha, 2020).

For the TGS, the genotype scores (GS) were then summed and transformed into a 0–100 scale by dividing the total score by the maximum possible score and multiplying by 100.

$$TGS = (\text{combined-GS} / \text{maximum-GS}) * 100$$

For the TWGS, a similar procedure to Varillas Delgado et al. (2020) was used. Each GS was multiplied by the standardised beta coefficients (β) of each SNP following multiple regression with each dependent variable to create weighted genotype scores (WGS). The WGSs were then summed and transformed into a 0–100 scale by dividing the total score by the maximum possible score and multiplying by 100.

$$TWGS = (\text{combined-WGS} / \text{maximum-WGS}) * 100$$

2.5. Data analysis

Each SNP was tested for adherence with Hardy-Weinberg equilibrium (HWE) using an exact test via SNPStats (Solé et al., 2006). Linkage disequilibrium (LD) was analysed using LDlink (Machiela & Chanock, 2015) and data from the 1000 Genomes Project European ancestry population (1000 Genomes Project Consortium, 2015). All other data were analysed using Jamovi version 1.8.1 and IBM SPSS version 25. Normality was assessed with the Shapiro-Wilk test and homoscedasticity was assessed using Levene's test. Akaike information criterion (AIC) was used to select which genetic model (i.e., co-dominant, dominant, recessive) best fit the data and would be subjected to hypothesis testing. However, if MAF ≤0.25 a dominant model was utilised to retain statistical power (Murtagh et al., 2020). Simple linear regression was performed to assess the association of genotype models with each personality dimension and mental toughness. Multiple regression was used to calculate the standardised beta coefficients (β) of each SNP for the TWGS models. Simple linear regression was then performed to assess the association of each TGS and TWGS with each personality dimension and mental toughness. Pearson's correlation coefficient (*r*) with thresholds

Table 1
Gene and single nucleotide polymorphism (SNP) information.

Gene	Symbol	Chr	SNP	Consequence	MAF
5-hydroxytryptamine receptor 2A	*HTR2A*	13q14.2	rs6311	Intron Variant C > T	T = 0.44
Brain derived neurotrophic factor	*BDNF*	11p14.1	rs6265	Missense Variant C > T (Val > Met)	T = 0.20
Cholinergic receptor muscarinic 2	*CHRM2*	7q33	rs1824024	Intron Variant C > A	C = 0.29
Catechol-O-methyltransferase	*COMT*	22q11.21	rs4680	Missense Variant G > A (Val > Met)	A = 0.50
Catenin alpha 2	*CTNNA2*	2p12	rs7600563	Intron Variant T > G	G = 0.34
Dopamine receptor D2	*DRD2*	11q23.2	rs1800497	Missense Variant G > A (Glu > Lys)	A = 0.19
Dopamine receptor D3	*DRD3*	3q13.31	rs167771	Intron Variant G > A	G = 0.20
Dopamine receptor D4	*DRD4*	11p15.5	rs1800955	2 KB Upstream Variant T > C	C = 0.41
Gamma-aminobutyric acid type A receptor subunit alpha6	*GABRA6*	5q34	rs3219151	3 Prime UTR Variant C > T	C = 0.42
Oxytocin receptor	*OXTR*	3p25.3	rs2254295	Intron Variant C > T	C = 0.11

Note. Chr = chromosome location; MAF = minor allele frequency (according to European population; 1000 Genomes Project Consortium, 2015).

values of ≤0.1 (trivial), >0.1–0.3 (small), >0.3–0.5 (moderate), >0.5–0.7 (large), >0.7–0.9 (very large), and >0.9–1.0 (almost perfect) were used to measure correlation (Hopkins et al., 2009). The coefficient of determination (R^2) was computed to determine the variance explained by each TGS and TWGS. Statistical significance was set at $p < .05$.

3. Results

Genotype and allele distributions of all SNPs were in HWE except for *DRD4* ($p = .040$), and all SNPs were in linkage equilibrium. Assumptions of normality and homoscedasticity were not violated. Descriptive statistics and genotype frequencies are displayed in Table 2.

3.1. Individual SNPs

There was a significant association between *DRD3* ($F_{(1, 71)} = 4.24$, $p = .043$) and agreeableness, where A/A homozygotes scored higher than G allele carriers ($B = 2.43$). No other associations were found (see Table 3).

3.2. TGS

Associations were also noted between the TGS and agreeableness ($F_{(1, 69)} = 6.31$, $p = .014$), extraversion ($F_{(1, 69)} = 7.16$, $p = .009$), mental toughness ($F_{(1, 68)} = 5.77$, $p = .019$), and neuroticism ($F_{(1, 69)} = 8.40$, $p = .005$). Moreover, small positive correlations were found with agreeableness ($r = 0.29$; $R^2 = 0.08$) and mental toughness ($r = 0.28$; $R^2 = 0.08$), whilst moderate positive correlations were found with extraversion ($r = 0.31$; $R^2 = 0.09$) and neuroticism ($r = 0.33$; $R^2 = 0.11$) (see Figure 1).

3.3. TWGS

There were significant associations between the TWGS and agreeableness ($F_{(1, 69)} = 10.57$, $p = .002$), conscientiousness ($F_{(1, 69)} = 4.30$, $p = .042$), extraversion ($F_{(1, 69)} = 9.50$, $p = .003$), mental toughness ($F_{(1, 68)} = 7.85$, $p = .007$), and neuroticism ($F_{(1, 69)} = 14.58$, $p < .001$). Moreover, a small positive correlation was found with conscientiousness ($r = 0.24$; $R^2 = 0.06$), whilst moderate positive correlations were found with agreeableness ($r = 0.35$; $R^2 = 0.12$), extraversion ($r = 0.36$; $R^2 = 0.13$), mental toughness ($r = 0.32$; $R^2 = 0.10$), and neuroticism ($r = 0.42$; $R^2 = 0.17$) (see Figure 2).

4. Discussion

This study examined associations amongst ten psychogenetic polymorphisms, both individually and collectively, with the personality and mental toughness profiles of academy football players. To our knowledge, this is the first assessment of the influence of genetic markers in isolation, and as part of a polygenic profile, on psychological traits within a homogenous football cohort. This study presents a novel association of *DRD3* (rs167771) with agreeableness and a preliminary polygenic model that explains a small proportion of the inter-individual variance in mental toughness and each personality dimension except openness. As such, these findings suggest several psychological characteristics of youth football players may be partly determined by genetic factors.

The A/A genotype of *DRD3* (rs167771) was associated with higher levels of agreeableness compared to the G allele. The *DRD3* gene encodes the DRD3 protein, which is the D3 subtype of the five dopamine receptors highly expressed in the limbic regions of the brain (e.g., hippocampus, nucleus accumbens, and ventral striatum) (Bouthenet et al., 1991; Gurevich & Joyce, 1999). The *DRD3* (rs167771) SNP is an intron (i.e., non-coding elements of a gene), which are important for efficient

Table 2
Descriptive statistics of personality dimensions and mental toughness.

Gene (SNP)	Genotype = *n* (%)	O	A	C	N	E	MT	MAF	HWE
Overall		25.47 ± 5.46	26.27 ± 4.98	27.67 ± 5.07	22.78 ± 6.85	23.67 ± 6.75	43.21 ± 5.56	N/A	N/A
HTR2A (rs6311)	C/C = 28 (38)	25.96 ± 4.99	25.18 ± 4.59	27.71 ± 4.04	23.79 ± 7.41	25.39 ± 6.41	43.46 ± 5.57	0.42	0.15
	C/T = 29 (40)	25.10 ± 5.17	27.59 ± 5.58	27.69 ± 5.86	22.03 ± 6.11	21.03 ± 7.21	42.63 ± 5.60		
	T/T = 16 (22)	25.25 ± 6.88	25.81 ± 4.17	27.56 ± 5.48	22.38 ± 7.31	25.44 ± 5.02	43.75 ± 5.76		
BDNF (rs6265)	C/C = 45 (62)	25.40 ± 5.55	26.91 ± 4.54	27.82 ± 5.58	23.47 ± 7.31	24.16 ± 6.36	43.53 ± 5.29	0.21	1
	C/T = 25 (34)	26.20 ± 5.34	25.68 ± 5.67	27.36 ± 4.35	21.16 ± 6.12	22.32 ± 7.39	42.25 ± 6.28		
	T/T = 3 (4)	20.33 ± 2.31	21.67 ± 3.06	28.00 ± 3.61	26.00 ± 2.00	27.67 ± 6.51	45.50 ± 3.32		
CHRM2 (rs1824024)	A/A = 23 (32)	26.22 ± 5.97	27.09 ± 5.83	27.87 ± 5.55	20.83 ± 8.01	24.04 ± 4.88	42.48 ± 6.80	0.40	0.22
	A/C = 41 (56)	24.90 ± 5.37	25.73 ± 4.48	27.44 ± 4.78	23.78 ± 5.84	23.54 ± 7.50	43.97 ± 4.32		
	C/C = 9 (12)	26.11 ± 4.73	26.67 ± 5.07	28.22 ± 5.63	23.22 ± 7.66	23.33 ± 7.91	41.78 ± 6.94		
COMT (rs4680)	A/A = 13 (18)	26.31 ± 5.02	24.92 ± 5.02	27.69 ± 4.61	19.54 ± 5.95	21.46 ± 5.61	44.85 ± 6.39	0.41	0.81
	G/A = 34 (47)	25.00 ± 5.05	26.85 ± 5.08	27.41 ± 5.31	23.71 ± 6.97	24.59 ± 6.58	42.38 ± 4.28		
	G/G = 26 (36)	25.65 ± 6.27	26.19 ± 4.88	28.00 ± 5.15	23.19 ± 6.86	23.58 ± 7.43	43.42 ± 6.48		
CTNNA2 (rs7600563)	G/G = 3 (4)	24.33 ± 9.50	24.67 ± 4.16	27.33 ± 8.50	26.00 ± 7.55	28.00 ± 9.64	44.33 ± 5.13	0.24	0.54
	T/G = 29 (40)	25.90 ± 6.20	25.72 ± 4.83	27.86 ± 4.44	22.69 ± 7.84	23.03 ± 6.69	43.89 ± 5.86		
	T/T = 40 (56)	25.20 ± 4.73	26.60 ± 5.11	27.30 ± 5.17	22.70 ± 6.19	23.88 ± 6.72	42.56 ± 5.49		
DRD2 (rs1800497)	A/A = 5 (7)	25.20 ± 5.40	24.00 ± 4.24	26.60 ± 2.30	25.20 ± 5.89	24.80 ± 5.89	40.60 ± 3.65	0.25	0.75
	G/A = 26 (36)	25.69 ± 5.55	26.54 ± 5.62	27.35 ± 5.61	22.38 ± 6.31	23.77 ± 7.34	42.91 ± 4.43		
	G/G = 42 (58)	25.36 ± 5.53	26.38 ± 4.68	28.00 ± 5.02	22.74 ± 7.34	23.48 ± 6.60	43.67 ± 6.24		
DRD3 (rs167771)	A/A = 46 (63)	25.41 ± 6.07	27.17 ± 5.30	28.15 ± 5.09	23.00 ± 7.37	24.07 ± 7.52	43.20 ± 5.98	0.21	0.72
	A/G = 23 (32)	25.39 ± 4.25	24.87 ± 3.96	27.26 ± 4.96	22.30 ± 6.31	23.65 ± 5.12	43.81 ± 4.98		
	G/G = 4 (5)	26.50 ± 5.26	24.00 ± 5.03	24.50 ± 5.51	23.00 ± 3.92	19.25 ± 4.86	40.25 ± 2.63		
DRD4 (rs1800955)	C/C = 18 (25)	25.78 ± 4.67	27.06 ± 5.77	26.94 ± 5.00	20.39 ± 7.92	22.06 ± 6.65	41.94 ± 7.30	0.43	0.04
	T/C = 27 (37)	25.56 ± 6.25	27.04 ± 4.90	28.19 ± 5.56	23.22 ± 6.41	25.11 ± 6.51	44.21 ± 4.74		
	T/T = 28 (38)	25.18 ± 5.28	25.04 ± 4.41	27.64 ± 4.74	23.89 ± 6.37	23.32 ± 6.99	43.13 ± 5.07		
GABRA6 (rs3219151)	C/C = 11 (15)	24.64 ± 3.91	26.73 ± 4.50	29.27 ± 5.57	21.82 ± 6.68	25.00 ± 5.46	42.83 ± 5.13	0.44	0.23
	T/C = 41 (58)	25.37 ± 5.75	26.49 ± 5.22	27.29 ± 4.52	23.07 ± 6.40	23.32 ± 6.32	43.53 ± 5.39		
	T/T = 19 (27)	26.00 ± 6.01	24.95 ± 4.62	27.68 ± 4.84	23.47 ± 7.95	23.37 ± 8.43	42.70 ± 6.42		
OXTR (rs2254295)	C/C = 3 (4)	21.67 ± 5.03	27.67 ± 5.51	29.00 ± 8.19	21.00 ± 4.36	21.00 ± 6.00	47.33 ± 5.13	0.16	0.36
	T/C = 17 (23)	26.12 ± 5.06	26.12 ± 4.92	27.65 ± 4.36	23.65 ± 5.99	23.71 ± 7.50	41.13 ± 4.63		
	T/T = 53 (73)	25.47 ± 5.60	26.25 ± 5.06	27.60 ± 5.20	22.60 ± 7.26	23.81 ± 6.63	43.62 ± 5.71		

Note. Data presented in mean ± standard deviation. O = openness; A = agreeableness; C = conscientiousness; N = neuroticism; E = extraversion; MT = mental toughness; MAF = minor allele frequency; HWE = Hardy-Weinberg equilibrium.

Table 3
Simple linear regression analysis.

Gene (SNP)	Model	*B*	*SE B*	β	*t*	*P*
Openness						
HTR2A (rs6311)	T/T-T/C vs. C/C	−0.81	1.32	−0.15	−0.61	.542
BDNF (rs6265)	T/T-T/C vs. C/C	0.17	1.32	0.03	0.13	.897
CHRM2 (rs1824024)	C/C–C/A vs. A/A	−1.10	1.38	−0.20	−0.80	.429
COMT (rs4680)	G/G-G/A vs. A/A	−1.02	1.68	−0.19	−0.61	.543
CTNNA2 (rs7600563)	T/T vs. T/G-G/G	−0.55	1.31	−0.10	−0.42	.676
DRD2 (rs1800497)	G/G vs. G/A-A/A	−0.26	1.30	−0.05	−0.20	.845
DRD3 (rs167771)	G/G-G/A vs. A/A	0.14	1.33	0.03	0.11	.915
DRD4 (rs1800955)	T/T vs. T/C–C/C	−0.47	1.32	−0.09	−0.35	.726
GABRA6 (rs3219151)	T/T vs. T/C–C/C	0.79	1.49	0.14	0.53	.598
OXTR (rs2254295)	T/T vs. T/C–C/C	0.02	1.44	0.00	0.02	.988
Agreeableness						
HTR2A (rs6311)	T/T-T/C vs. C/C	1.78	1.19	0.36	1.49	.139
BDNF (rs6265)	T/T-T/C vs. C/C	−1.66	1.19	−0.33	−1.39	.168
CHRM2 (rs1824024)	C/C–C/A vs. A/A	−1.19	1.26	−0.24	−0.95	.348
COMT (rs4680)	G/G-G/A vs. A/A	1.64	1.52	0.33	1.08	.284
CTNNA2 (rs7600563)	T/T vs. T/G-G/G	0.98	1.17	0.20	0.83	.408
DRD2 (rs1800497)	G/G vs. G/A-A/A	0.25	1.19	0.05	0.21	.833
DRD3 (rs167771)	G/G-G/A vs. A/A	−2.43	1.18	−0.49	−2.06	**.043***
DRD4 (rs1800955)	T/T vs. T/C–C/C	−2.01	1.18	−0.40	−1.70	.094
GABRA6 (rs3219151)	T/T vs. T/C–C/C	−1.59	1.32	−0.32	−1.20	.233
OXTR (rs2254295)	T/T vs. T/C–C/C	−0.10	1.32	−0.02	−0.08	.937
Conscientiousness						
HTR2A (rs6311)	T/T vs. T/C–C/C	−0.14	1.44	−0.03	−0.10	.923
BDNF (rs6265)	T/T-T/C vs. C/C	−0.39	1.23	−0.08	−0.32	.750
CHRM2 (rs1824024)	C/C vs. C/A-A/A	0.63	1.82	0.12	0.35	.730
COMT (rs4680)	G/G vs. G/A-A/A	0.51	1.25	0.10	0.41	.683
CTNNA2 (rs7600563)	T/T vs. T/G-G/G	−0.51	1.18	−0.10	−0.43	.666
DRD2 (rs1800497)	G/G vs. G/A-A/A	0.77	1.21	0.15	0.64	.523
DRD3 (rs167771)	G/G-G/A vs. A/A	−1.30	1.23	−0.26	−1.06	.293
DRD4 (rs1800955)	T/T-T/C vs. C/C	0.96	1.38	0.19	0.70	.488
GABRA6 (rs3219151)	T/T-T/C vs. C/C	−1.86	1.56	−0.39	−1.19	.237
OXTR (rs2254295)	T/T vs. T/C–C/C	−0.25	1.34	−0.05	−0.18	.855
Neuroticism						
HTR2A (rs6311)	T/T-T/C vs. C/C	−1.63	1.65	−0.24	−0.99	.326
BDNF (rs6265)	T/T-T/C vs. C/C	−1.79	1.65	−0.26	−1.09	.281
CHRM2 (rs1824024)	C/C–C/A vs. A/A	2.85	1.70	0.42	1.68	.098
COMT (rs4680)	G/G-G/A vs. A/A	3.94	2.06	0.58	1.92	.059
CTNNA2 (rs7600563)	T/T vs. T/G-G/G	−0.30	1.64	−0.04	−0.18	.856
DRD2 (rs1800497)	G/G vs. G/A-A/A	−0.10	1.63	−0.01	−0.06	.951
DRD3 (rs167771)	G/G-G/A vs. A/A	−0.59	1.67	−0.09	−0.35	.724
DRD4 (rs1800955)	T/T-T/C vs. C/C	3.17	1.83	0.46	1.73	.088
GABRA6 (rs3219151)	T/T-T/C vs. C/C	1.38	2.24	0.20	0.62	.540
OXTR (rs2254295)	T/T vs. T/C–C/C	−0.65	1.81	−0.09	−0.36	.722
Extraversion						
HTR2A (rs6311)	T/T-T/C vs. C/C	−2.79	1.60	−0.41	−1.74	.086
BDNF (rs6265)	T/T-T/C vs. C/C	−1.26	1.63	−0.19	−0.78	.441
CHRM2 (rs1824024)	C/C–C/A vs. A/A	−0.54	1.71	−0.08	−0.32	.752
COMT (rs4680)	G/G-G/A vs. A/A	2.69	2.05	0.40	1.31	.195
CTNNA2 (rs7600563)	T/T vs. T/G-G/G	0.37	1.62	0.06	0.23	.818
DRD2 (rs1800497)	G/G vs. G/A-A/A	−0.46	1.61	−0.07	−0.29	.776
DRD3 (rs167771)	G/G-G/A vs. A/A	−1.07	1.64	−0.16	−0.65	.519
DRD4 (rs1800955)	T/T-T/C vs. C/C	2.14	1.83	0.32	1.17	.245
GABRA6 (rs3219151)	T/T-T/C vs. C/C	−1.67	2.22	−0.25	−0.75	.456
OXTR (rs2254295)	T/T vs. T/C–C/C	0.51	1.78	0.08	0.29	.775
Mental Toughness						
HTR2A (rs6311)	T/T vs. T/C–C/C	0.70	1.59	0.13	0.44	.663
BDNF (rs6265)	C/C vs. C/T-T/T	0.82	1.36	0.15	0.60	.547
CHRM2 (rs1824024)	A/A vs. A/C–C/C	−1.08	1.41	−0.19	−0.77	.446
COMT (rs4680)	G/G-G/A vs. A/A	−2.00	1.70	−0.36	−1.18	.244
CTNNA2 (rs7600563)	G/G-G/T vs. T/T	1.37	1.35	0.25	1.02	.312
DRD2 (rs1800497)	A/A-A/G vs. G/G	−1.17	1.35	−0.21	−0.87	.388
DRD3 (rs167771)	A/A vs. A/G-G/G	−0.04	1.39	−0.01	−0.03	.975
DRD4 (rs1800955)	T/T-T/C vs. C/C	1.67	1.55	0.30	1.08	.284
GABRA6 (rs3219151)	T/T vs. T/C–C/C	−0.66	1.49	−0.12	−0.44	.659
OXTR (rs2254295)	C/C–C/T vs. T/T	−1.51	1.49	−0.27	−1.01	.315

Note. Bold values and * highlight statistical significance at $p < .05$. *B* = unstandardised beta; *SE B* = standard error; β = standardised beta.

splicing or other regulatory elements involved in transcription (Kim et al., 2008). In line with the present study, *DRD3* has previously been associated with emotional and cognitive functions (Bouthenet et al., 1991; Gurevich & Joyce, 1999). However, at the time of writing, there is no data available regarding the underpinning mechanism of the allelic association between *DRD3* (rs167771) and psychological traits, so it remains unclear whether they have a functional effect or are in LD with the actual effect alleles (Staal, 2015).

To the authors' knowledge, this is the first study to find a direct association between *DRD3* (rs167771) and agreeableness. This may be due to the unique participants under investigation in this study. However, this finding is comparable to previous relationships between *DRD3* (rs167771) and other relevant phenotypes. For instance, previous research has indicated that the G allele of *DRD3* (rs167771) may be associated with an increased risk of autism (de Krom et al., 2009; Toma et al., 2013). Autism is an applicable phenotype to use for an indirect comparison, as lower levels of agreeableness have also been associated

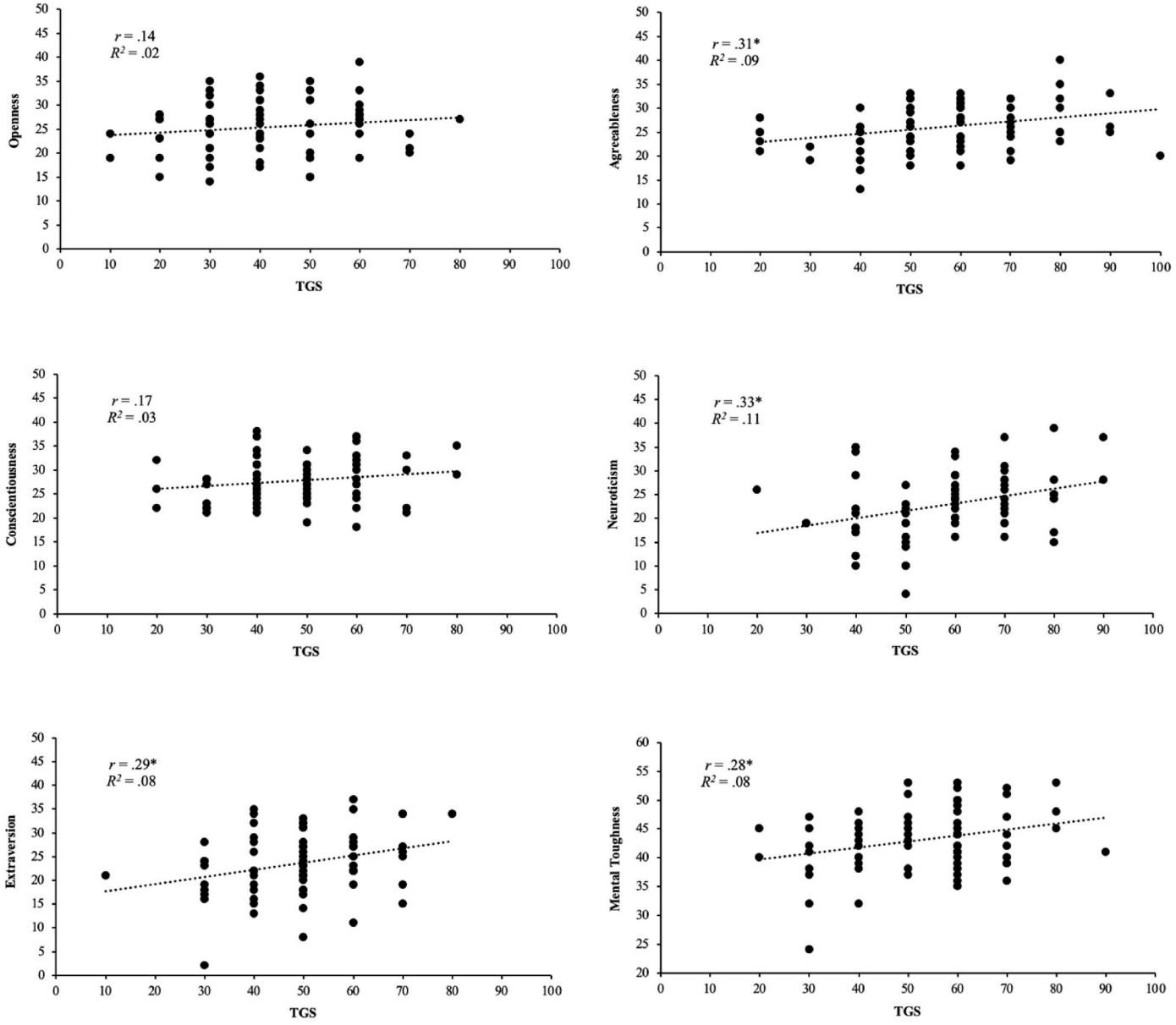

Fig. 1. Total genotype score (TGS) correlations. * Statistically significant at $p < .05$.

with an increased risk of autism (Austin, 2005). Although further research is required, these findings are consistent with the scientific literature associating *DRD3* (rs167771) with psychological traits.

There was no association between any other SNP in isolation and a personality dimension or mental toughness. Accurately measuring personality dimensions and mental toughness is notoriously difficult. Moreover, their overall phenotypic complexity means it is highly unlikely that inter-individual variance would be explained by individual SNPs. Indeed, identifying genetic associations between common variants and psychological traits may be even more challenging than with physical traits. For instance, whilst the SNPs associated with the largest effects on physical traits explain an estimated 0.3% of the inter-individual variance, the SNPs associated with the largest effects on psychological traits only explain an estimated 0.02% of the inter-individual variance (Chabris et al., 2015). This suggests a larger number of SNPs with smaller effect sizes may account for the variability observed in psychological traits. Moreover, it was shown that a psychological condition (i.e., schizophrenia) was influenced by ~5,000 more SNPs than four physical conditions (i.e., celiac disease, coronary artery disease, rheumatoid arthritis, and type 2 diabetes) (Ripke et al., 2013).

Candidate gene approaches are exemplified by numerous limitations in psychological research (see Munafò & Flint, 2011), but can also confer inferential advantages when sample cohorts are small and/or unique (e.g., athletic populations) (Jorgensen et al., 2009). However, evidence suggests a vast number of SNPs with small effect sizes may underpin psychological phenotypes. In light of this, polygenic profiles were used to assess the combined influence of the selected SNPs, as they have not only proved effective in psychogenetic research (Ausmees et al., 2021), but also previous research on phenotypes in football (e.g., athlete status, injury, physiological performance, and technical ability) (McAuley, Hughes, et al., 2021a). Moreover, applying a weight to each SNP based on its individual influence on a respective phenotype has been shown to increase the variance explained by a polygenic profile on physiological and technical phenotypes in football (Massidda et al., 2014). The results from this study correspond with and expand previous research, as the TGSs and TWGSs were associated with several psychological phenotypes, but the TWGS consistently explained more variance.

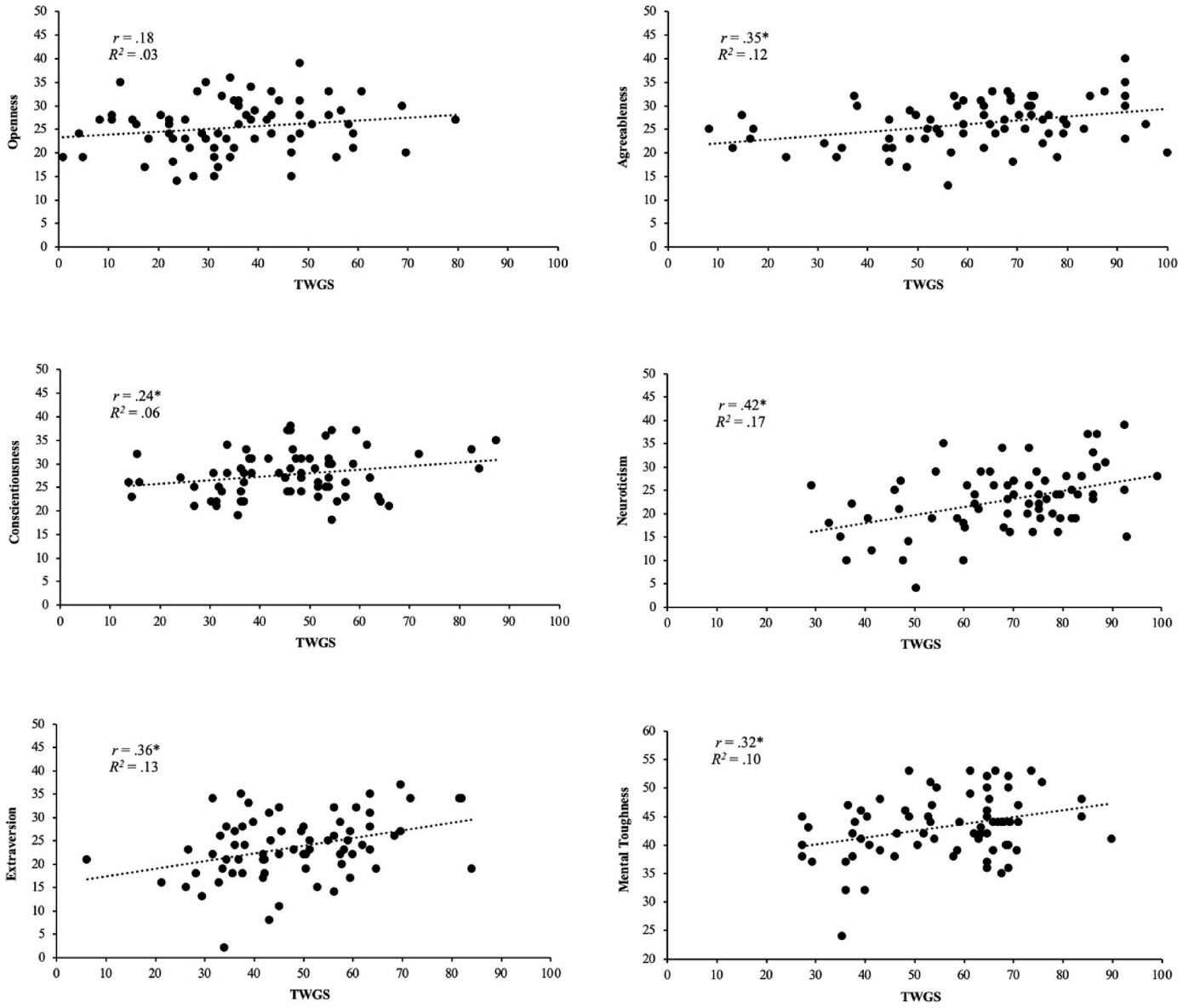

Fig. 2. Total weighted genotype score (TWGS) correlations. * Statistically significant at $p < .05$.

The polygenic profiles in this study were associated with mental toughness and all personality dimensions except openness. The combined variance explained by the SNPs in the polygenic models differed between each psychological phenotype, where the strongest association was observed with neuroticism (e.g., neuroticism = 11–17%, extraversion = 9–13%, agreeableness = 8–12%, mental toughness = 8–10%, conscientiousness = 3–6%, and openness = 2–3%). All personality dimensions and mental toughness have been previously associated with differentiating successful and less successful individual and team-sport athletes (Benítez-Sillero et al., 2021; Piepiora, 2021; Steca et al., 2018). That said, more consistent associations and larger effect sizes are generally reported with neuroticism, which is encompassed by facets such as anxiety, depression, hostility, impulsiveness, self-consciousness, and vulnerability (Piepiora & Piepiora, 2021). As such, the most noteworthy feature of this study is the identification of a panel of SNPs that may be associated with one of the key psychological variables in sport, and more specifically football.

These SNPs may prove useful in creating a genetic tool capable of assisting practitioners with implementing more individualised psychological intervention programmes in the future. That said, precisely how genetic information should be utilised by practitioners is still unclear and requires careful thought and consideration (McAuley, Hughes, et al., 2021b). Reducing the complex biological nature of psychological phenotypes to answers in a questionnaire is ill-advised. The intricacies of an individuals' moment to moment experiences can be difficult to articulate, perceptual, and difficult to measure, even if more robust measures are used. Therefore, the validation of these results in larger homogenous and independent football cohorts is required, as well as the identification of more relevant genetic variants, before implementing the current findings into practice. However, even if these results are validated in larger independent cohorts, we believe they should not be used for talent identification purposes due to the multifactorial nature of performance in football and the accompanying social, ethical, and legal issues associated with potential genetic discrimination (McAuley, Hughes, et al., 2022). Instead, validated results could be used to enhance athlete development processes by managing athlete welfare to facilitate improved long-term performance. Genetic information should not be seen as an isolated determinant by practitioners, but rather as an additional objective tool to enhance often subjective decisions (McAuley, Baker, et al., 2021).

This study has some limitations that should be acknowledged when interpreting its findings. First, the sample size was small, so the study may have been underpowered and unable to detect more significant associations, increasing type 2 error. However, highly skilled athletic populations are generally small by nature and are notoriously difficult to gain access to. Indeed, this study's sample ($N = 73$) is larger than the median sample size ($N = 60$) reported in a recent review of eighty genetic association studies in football (see McAuley, Hughes, et al., 2021a). Building this research base with studies using transparent methodologies is important so they can contribute to research synthesis approaches in the future to draw more valid and reliable conclusions (McAuley, Baker, & Kelly, 2022). Second, this study did not make adjustments for multiple comparisons, which may have increased type 1 errors. However, due to the exploratory nature of this study, in regards to the novel experimentation methods employed and unique cohort, reducing type 2 errors was considered a priority. This is recommended in exploratory research (see Althouse, 2016), as a main aim is to ensure an important discovery is not missed in the first instance, which can be validated in subsequent dedicated replication studies. Power analyses indicated that our sample size ($N = 73$) was sufficiently powered at .80 to detect significant associations ($p < .05$) with an effect size equivalent to $R^2 = 0.10$. This suggests the study was underpowered to detect individual SNP associations but had adequate power to detect some TGS/TWGS associations. Third, the weighting of SNPs and the direction of the allelic associations in the polygenic models were data driven due to the unique population and lack of prior literature using high-powered research designs. As such, inaccurate weightings and opposite scores could have been assigned to specific SNPs and alleles, which may decrease external validity. Lastly, participants were only genotyped for SNPs and epistatic interactions were not considered. There are many other types of genetic polymorphisms associated with psychological phenotypes (e.g., insertions-deletions and copy number variants) and the interactions between genetic variants may alter associations, thus changing the accuracy of polygenic models. Therefore, the addition of more SNPs and other polymorphisms, whilst also considering their interactions, may increase the polygenic models' accuracy.

5. Conclusion

This study has presented novel evidence regarding the association of inter-individual genetic variation with the mental toughness and personality profiles of youth football players. These findings suggest that in isolation the *DRD3* (rs167771) SNP G allele is associated with lower levels of agreeableness. In addition, the collective influence of all SNPs included in this study were shown to be associated with mental toughness and all personality dimensions except openness. As such, the findings from this exploratory study suggest that several psychological characteristics of academy footballers may be partly determined by genetic factors. Therefore, we suggest that future studies incorporate SNPs associated with psychological variables when conducting research into genetic associations with behavioural traits and/or athletic prowess. Successful independent replication in large homogenous cohorts may allow practitioners in the future to implement more individualised psychological intervention programmes during athlete development, which may help manage athlete welfare and facilitate improved long-term performance.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Declaration of competing interest

None.

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