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Association of the MuRF-1/TRIM63 polymorphism with muscle injuries in professional soccer players
Asociación del polimorfismo MuRF-1/TRIM63 con lesiones musculares en futbolistas profesionales

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Abstract. Muscle injuries are one of the biggest medical problems in professional soccer. Evidence has suggested that genetic polymorphism is a mediating factor in physiological and structural alterations that can lead to muscle injury. The TRIM63 gene polymorphism may affect the MuRF-1 protein, which is vital in the regulation of muscle mass, and is differentially regulated by exercise mode, muscle contraction, and training status. However, central aspects of the relationship between genetic variations, the environment, and muscle injuries still need to be explained. This study aimed to investigate whether MuRF-1/TRIM63 (A/G, rs2275950) was associated with the occurrence of muscle injury in professional soccer players. Forty-six Brazilian soccer players were evaluated. Genomic DNA was extracted using blood samples and semi-structured interviews on muscle injuries were applied after two seasons (2021-2022). Fisher's exact test was used to verify if MuRF-1/TRIM63 was associated with muscle injuries. The MuRF-1/TRIM63 genotypes ($\chi^2 = 2.19$; $p = 0.292$), the dominant model ($\chi^2 = 1.04$; $p = 0.299$), and the recessive model ($\chi^2 = 1.94$; $p = 0.208$) showed no association with muscle injuries in soccer players. Preliminary evidence suggests that this genetic polymorphism may not be a reliable biomarker of muscle injuries in Brazilian professional soccer players.
Keywords: Football, Genetic, Muscle injury, Performance, Single Nucleotide Polymorphism (SNP), Sport genomic.

Resumen. Las lesiones musculares son uno de los mayores problemas médicos en el fútbol profesional. Las pruebas han sugerido que el polimorfismo genético es un factor mediador en las alteraciones fisiológicas y estructurales que pueden provocar lesiones musculares. El polimorfismo del gen TRIM63 puede afectar a la proteína MuRF-1, que es vital en la regulación de la masa muscular, y está regulada de forma diferencial por el modo de ejercicio, la contracción muscular, y el estado de entrenamiento. Sin embargo, aún quedan por explicar aspectos centrales de la relación entre las variaciones genéticas, el entorno, y las lesiones musculares. El objetivo de este estudio era investigar si MuRF-1/TRIM63 (A/G, rs2275950) estaba asociado con la aparición de lesiones musculares en futbolistas profesionales. Se evaluaron cuarenta y seis jugadores de fútbol brasileños. Se extrajo DNA genómico a partir de muestras de sangre y se aplicaron entrevistas semiestructuradas sobre lesiones musculares después de dos temporadas (2021-2022). Se utilizó la prueba exacta de Fisher para verificar si MuRF-1/TRIM63 estaba asociado con las lesiones musculares. Los genotipos MuRF-1/TRIM63 ($\chi^2 = 2.19$; $p = 0.292$), el modelo dominante ($\chi^2 = 1.04$; $p = 0.299$), y el modelo recesivo ($\chi^2 = 1.94$; $p = 0.208$) no mostraron asociación con las lesiones musculares en futbolistas. Las pruebas preliminares sugieren que este polimorfismo genético puede no ser un biomarcador fiable de lesiones musculares en futbolistas profesionales brasileños.
Palabras clave: Fútbol, Genética, Lesión muscular, Rendimiento, Polimorfismo de Nucleótido Único (SNP), Genómica deportiva.

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Introduction

Muscle injuries are extremely frequent in soccer, in their clinical and epidemiological outcome are multifactorial (Mandorino et al., 2023; Sinovas et al., 2020). Its high prevalence is documented in the international literature and has been the target of studies in professional soccer (Maestro et al., 2022; Murtagh et al., 2023; Uchamocha et al., 2024). Evidence indicates that up to 95% of all muscle injuries occur in non-contact situations, and the most common type of injury was muscle/tendon with 4.6 injuries/1000 hours of exposure (Ekstrand et al., 2023; López-Valenciano et al., 2019). According to Waldén et al. (2023), injury is defined by evidence of tissue damage or other derangement of normal physical function resulting from the rapid or repetitive transfer of kinetic energy. Muscle injuries can be classified into two groups; direct muscle injuries (caused by external factors) and indirect muscle injuries (can also be referred to as "non-contact" and are caused by internal factors) that are identified as functional muscle disorders or structural muscle injuries (Mueller-Wohlfahrt et al., 2013). Consequently, muscle injuries have a negative impact on the physical and psychological health of soccer players (Fagundes et al., 2021). The incidence of muscle injuries in professional soccer teams is positively associated with economic costs, and inversely related to the team's success during the season (Ekstrand et al., 2023; Pulici et al., 2022). Muscle injury in soccer represents a complex process influenced by multifactorial parameters (Mandorino et al., 2023; Uchamocha et al., 2024). Recently, genetic factors have been attributed to a role in the susceptibility to muscle injury, the efficiency of recovery mechanisms, and the potential implications for athletic performance (McAuley et al., 2022; Moya, 2021). Genetic polymorphisms have been associated with non-contact muscle injuries in soccer (Lim et al., 2021, Maestro et al., 2022). In this context, evidence indicates that certain genetic polymorphisms, such as MuRF-1/TRIM63, may influence the mechanical properties of muscle fibers that express, for example, a phenotype with characteristics of delayed inflammation, resulting in slower recovery after exercise (Baumert et al., 2017; Baumert et al., 2022). The Muscle RING Finger-1 (MuRF-1) encoded by the human tripartite motif containing 63 (TRIM63) gene is located on

chromosome 1 at position 26.058.512, in which a single nucleotide polymorphism (SNP, rs2275950; AA, AG and GG) at amino acid 237 causing a change from lysine to glutamate (Bodine et al., 2001; Centner et al., 2001). MuRF-1, MuRF-2 and MuRF-3 proteins are a specific class of proteins expressed in striated and cardiac muscle tissues (Chen et al., 2012; Peris-Moreno et al., 2020). The MuRF-1 protein mediates a key role in the Ubiquitin Proteasome System (UPS), attaching ubiquitin polymers to deteriorated myofibrillar proteins after muscle damage (Centner et al., 2001; Yang et al., 2022). MuRF-1 is localized at the Z- and the M-line of the sarcomere, in which it has been found to interact and modulate the mechanical properties of titin (Baehr et al., 2021; Bodine et al., 2001).

Centner et al. (2001) stated that the relationship between the MuRF-1 and titin proteins is pivotal for the integrity of the sarcomere. This connection may provide stability and protection against muscle damage caused by eccentric damaging contractions from sports practice (Baumert et al., 2017). After this damaging process, coordinated actions are mediated by MuRF-1 and titin in the control of protein quality, protein degradation, and synthesis as well as in the process of structuring new myofibrils (Stefanetti et al., 2014; Yang et al., 2022).

A cluster of evidence has shown that MuRF-1/TRIM63 has been investigated as a regulator of muscle mass and is involved in catabolic processes identified in physiological conditions and pathological states (Peris-Moreno et al., 2020). This gene has been linked to situations such as muscle atrophy (Baehr et al., 2021), cardiomyopathies (Chen et al., 2012), and exercise-induced muscle damage (Baumert et al., 2022). Concerning exercise, researchers investigated this protein during endurance and resistance activities found that MuRF-1 mRNA protein levels increased significantly after endurance training and exercise (Baumert et al., 2022; Stefanetti et al., 2014). These studies showed consistent results revealing that the MuRF-1/TRIM63 response to exercise results in upregulation. Moreover, a study by Baumert et al. (2017) revealed that homozygous with the A-allele were significantly stronger and recovered faster after strenuous exercise than those with the G-allele who required a longer recovery period post-exercise. As a result, the A-allele may promote greater affinity for the titin strain-sensing kinase domain, suggesting that genotype-phenotype interaction may be able to tolerate a greater training and match frequency in soccer. Recent studies have identified some genetic markers that could influence the predisposition to muscle injuries in soccer (Lim et al., 2021; Maestro et al., 2022; Murtagh et al., 2023). However, to date, no study has investigated the MuRF-1/TRIM63 polymorphism in professional soccer. Furthermore, investigating this gene in the competitive environment could provide new insights into the inter-individual variability of soccer players who are submitted to frequent training and match exposures. We hypothesized that the presence of the A-allele (previously associated with greater titin stiffness) could offer muscle fibers greater resistance to eccentric damaging contractions. The present study aimed to investigate whether MuRF-1/TRIM63 genotypes were associated with the occurrence of muscle injury in professional soccer players.

Materials and methods

Participants

This is a study with a convenience sample from a first division professional soccer club in Brazil. Data were collected from 46 male Brazilian soccer players (mean ± SD, age: 21.3 ± 1.14 years, body mass 73.7 ± 6.41 kg, height 179.0 ± 6.50 cm). As inclusion criteria, the individuals assessed had to be professional soccer players with a contract with the first team, who participated in training and matches over the season at the same club and performed regular exercise training of > 1 hour per day, > 5 days per week for the prior 1 year. Exclusion criteria were contact injuries throughout the season, goalkeepers, and professional female soccer players.

Written informed consent was obtained from each player after they were informed about the advantages and potential risks. The study was performed in accordance with the ethical standards of the Helsinki Declaration and resolution 466/2012 of the National Health Council for research on humans. This study complied with the Ethical Standards in Sport and Exercise Science Research (Harriss et al., 2022), and was approved by the Research Ethics Committee (COEP-UFMG; No. 5.764.810).

Study procedures

A retrospective survey study was carried out (Isik et al., 2018; Sinovas et al., 2020). A pilot study was conducted to apply the customized semi-structured interview muscle injury for soccer players in August 2022. In November 2022, data was obtained at the club's training center in three steps. Firstly, the venipuncture for DNA was collected by the club physician (Clos et al., 2019; Pruna et al., 2013). Secondly, the club's physiologist carried out the physical assessment (Jackson & Pollock, 1978). Finally, the semi-structured interview was conducted only by the study researcher.

The semi-structured interview was individual; each player received verbal guidance from the researcher and could answer the questions without a time limit (Fagundes et al., 2021). The data was collected through a semi-structured interview using questions about the occurrence of non-contact muscle injuries and psychological aspects in the last two seasons (2021-2022). The data were transferred to a computer spreadsheet for organization, systematization, and analysis. All the procedures described happened on a single day in the season (Figure 1).

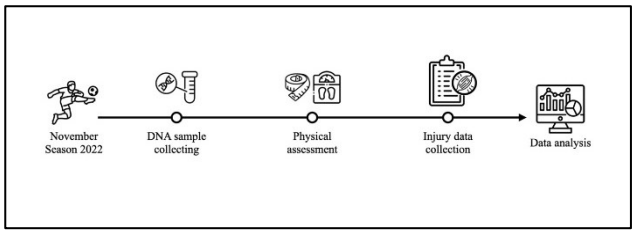

Figure 1. Study design

Physical assessment

Body mass, height, and skinfolds were measured to characterize the sample. Body mass (kg) and height (cm) were evaluated using a digital scale (Filizola®). Skinfolds were measured using a plicometer (Lange®) according to the protocol proposed (Jackson & Pollock, 1978).

DNA sample collecting

Approximately 4 ml of blood was extracted from the antecubital arm vein of each participant using EDTA tubes (BD Vaccutainer®, Brazil). Genomic DNA was extracted from 500 µl of whole blood using the salting out method (Miller et al., 1988). The quality and integrity of the samples were tested by spectrophotometry (Nanodrop®, Thermo Fisher Scientific-GE, USA). Genotyping for MuRF-1/TRIM63 was performed by polymerase chain reaction method (PCR) based on previous studies (Baumert et al., 2017; Stefanetti et al., 2014). To determine the rs2275950 polymorphism of the TRIM63 gene, the site of interest was amplified from the genomic DNA using the following primers, forward CCTGAGAGCCATTGACTTTGG and reverse CTTCCCTTCTGTGGACTCTTCCT (Applied Biosystems®, USA). Allele discrimination was performed using a genomic sequence detection system (Applied Biosystems® Steponeplus™ Real-Time PCR System, USA). In each qPCR plate, 10-15 ng of DNA (1 µL) was pipetted in addition to 12.5 µL of genotyping master mix (TaqMan Genotyping Master Mix® - 2×), 1.3 µL of specific primers and probes (TaqMan Genotyping Assay Mix® - 20×) and 11.2 µL of DNAse and RNAse free water, totaling a final volume of 25 µL for each sample. The amplification process started with denaturation at 95°C for 10 minutes, followed by 40 cycles of 94°C for 15 seconds, and a cycle of 60 seconds at 60°C.

Muscle Injuries data collection

The development of the semi-structured interview was based on the international consensus statement on epidemiological studies in soccer (Fuller et al., 2006; Hägglund et al., 2005). The mechanism of injury evaluated in the study was non-contact. This non-contact muscle injury should present structural-mechanical damage, such as partial or total muscle tears (Mueller-Wohlfahrt et al., 2013). The registration of a muscle injury was based on a clinical examination by the team medical staff. The date of muscle injury, whether the muscle injury was sustained during a match or training exposure, the date of the player's return to full participation, and the nature of the injury were registered. The medical staff described the injury report form based on the diagnosis of muscle injury (Fuller et al., 2006). In all cases, the diagnosis was supported by ultrasound and/or magnetic resonance imaging scans (Pruna et al., 2013). The data obtained from the players through the semi-structured interview and the data collected from the club's medical department (injury report form) complemented the final document (Isik et al., 2018; Sinovas et al., 2020). Injury severity was classified as mild (1-7 days), moderate (8-28 days), and severe (> 28 days) to evaluate muscle injuries in professional soccer teams (Clos et al., 2019; Larruskain et al., 2018; Pruna et al., 2013).

Statistical analysis

The Shapiro-Wilk test was performed to check the normality of the data. As the data had a normal distribution, the mean and standard deviation were used. ANOVA one-way was employed to verify differences between genotypes (AA vs AG vs GG) with the Tukey test applied post hoc for pairwise comparisons. Differences between (Injured players vs Non-injured players) were calculated using the Student's *t*-test for independent groups. The Hardy-Weinberg equilibrium and genotype distribution were determined using the Chi-Square test (χ^2). In all inferential analyses, the expected values were less than 5. Therefore, was performed Fisher's exact test to verify the association between MuRF-1/TRIM63 genotypes and models (dominant and recessive) with muscle injuries. The level of statistical significance adopted was $p < 0.05$. All data was analyzed using JASP software (Team, 2020; JASP Version 0.14).

Results

Of the 46 professional soccer players, 32 players had an injury over the seasons (69.6%) for a total of 50 muscle injuries. The other 14 players (30.4%) did not present any muscle injury during the two seasons (2021-2022). Of the 50 muscle injuries recorded, 17 (34%) occurred in official matches, while 33 (66%) happened in training.

Table 1 shows the descriptive data regarding the characterization of the study participants. The age of soccer players genotyped AA ($p = 0.004$) and GG ($p = 0.037$) were statistically significant when compared with the AG genotype. The variables compared between injured and non-injured players were not statistically significant.

Genotype frequency distribution for the MuRF-1/TRIM63 rs2275950 ($\chi^2 = 2.50$; $p = 0.285$) was in Hardy-Weinberg equilibrium. The frequency of the AA genotype was significantly higher than the AG and GG genotypes in Brazilian soccer players ($\chi^2 = 23.10$; $p < 0.001$).

Table 1.
Subjects characteristics according to MuRF-1/TRIM63 genotypes and muscle injury

Variables	MuRF-1/TRIM63			*p*-value	Muscle Injury		*p*-value
	AA (30)	AG (12)	GG (4)		IP	NIP	
n (%)	30 (65.22)	12 (26.08)	4 (8.70)		32 (69.6)	14 (30.4)	
Age (years)	21.55 ± 1.05	20.37 ± 1.03	21.88 ± 0.48	0.007*	21.10 ± 1.28	21.60 ± 0.69	0.263
Weight (kg)	73.27 ± 6.75	74.67 ± 6.48	73.67 ± 3.93	0.835	72.70 ± 6.74	75.90 ± 5.10	0.116
Height (cm)	178.11 ± 6.57	180.88 ± 7.05	175.97 ± 0.91	0.062	179.00 ± 6.39	179.00 ± 6.96	0.831
Body fat (%)	10.03 ± 2.12	10.04 ± 2.79	10.75 ± 2.07	0.824	9.72 ± 2.21	10.90 ± 2.25	0.097
Experience (years)	8.83 ± 1.70	7.83 ± 2.08	7.50 ± 1.00	0.106	8.66 ± 1.88	8.00 ± 1.62	0.262

All variables are expressed as a mean ± standard deviation. IP (Injured players), NIP (Non-injured players). * $p < 0.05$

Table 2 presents the analysis to verify whether MuRF-1/TRIM63 genotypes were associated with the occurrence of muscle injury. Of the 32 injured soccer players, 23 individuals genotyped as AA had 35 muscle injuries (70%), seven AG individuals suffered 12 muscle injuries (24%), and two GG individuals were diagnosed with three muscle injuries (6%). The MuRF-1/TRIM63 genotypes ($\chi^2 = 2.19$; $p = 0.292$), the dominant model ($\chi^2 = 1.04$; $p = 0.299$), and the recessive model ($\chi^2 = 1.94$; $p = 0.208$) showed no association with muscle injuries in soccer players.

Table 2.
Models of association between the MuRF-1/TRIM63 and muscle injury

Muscle injury	Genotype			Dominant model		Recessive model	
	AA	AG	GG	AA + AG	GG	AA	AG + GG
IP	23	7	2	30	2	23	9
NIP	7	5	2	12	2	7	7
p-value		0.356		0.659		0.876	

IP (Injured players); NIP (Non-injured players)

Table 3.
Description of muscle injuries in professional soccer players

Players	Position	Dominat leg	Location			NMIP
			1 injury	2 injury	3 injury	
2	MD	L	LA	RQ	LQ	3
3	DF	R	RA	RH		2
4	DF	R	RA	RG	LG	3
5	DF	L	LA	RQ		2
7	FW	R	LG			1
8	FW	L	LH			1
10	DF	R	LH			1
12	DF	R	LH			1
15	DF	L	LH			1
16	DF	L	RH			1
17	FW	L	LA			1
19	MD	L	LQ			1
20	FW	R	RH			1
22	FW	L	RH			1
25	FW	R	RH	LH		2
28	FW	R	RH	RA		2
29	MD	R	RH			1
30	FW	R	RH	LH	RQ	3
31	DF	R	RH	LH		2
32	FW	R	LA	RQ		2
34	MD	R	RQ			1
35	DF	R	RQ	LQ		2
37	MD	R	LH	RA		2
38	MD	R	RH			1
39	FW	R	LQ			1
40	FW	R	RA			1
41	DF	R	RA	RH		2
42	MD	L	LH			1
43	DF	R	RH	RA		2
44	MD	R	RH	LH		2
45	FW	L	LA	RH		2
46	DF	L	RA			1
Total muscle injury						50

NMIP: number of muscle injuries per players; DF: defenders; MD: midfielders; FW: forwards. R: right; L: left. LA: left adductor; RA: right adductor; LH: left hamstring; RH: right hamstring; LQ: left quadriceps; RQ: right quadriceps; LG: left gastrocnemius; RG: right gastrocnemius

Table 3 provides the characteristics of the muscle injuries suffered by professional soccer players. According to position, defenders had 40% of the muscle injuries, while midfielders 24%, and forwards 36% over two seasons.

Table 4 shows the MuRF-1/TRIM63 genotype distribution based on the severity of muscle injuries resulting in absence from training sessions and matches during treatment and return to play.

Table 4.
MuRF-1/TRIM63 genotypes distribution based on the severity of muscle injuries

Degree of severity	Number of injuries	% of total injuries	MuRF-1 genotype distribution		
			AA (%)	AG (%)	GG (%)
Mild (1-7 days)	4	8	2 (50.0)	1 (25.0)	1 (25.0)
Moderate (8-28 days)	21	42	14 (66.6)	5 (23.9)	2 (9.5)
Severe (> 28 days)	25	50	19 (76.0)	6 (24.0)	0 (0)

The anatomical locations of muscle injuries were organized as reported in Figure 2. Hamstring (48%) and adductor (26%) were quantitatively the main muscles in which soccer players suffered injuries followed by the Quadriceps (20%) and Gastrocnemius (6%).

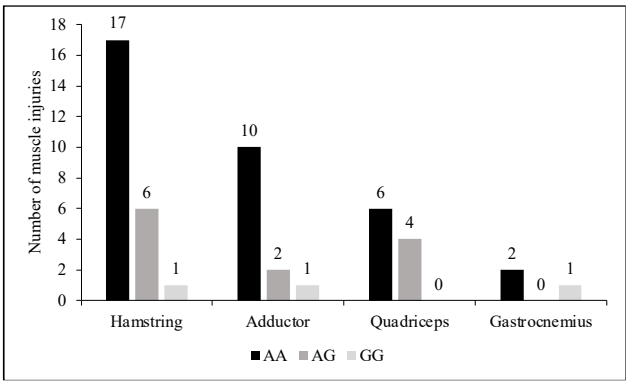

Figure 2. Muscle injury location in professional soccer players

Discussion

This study aimed to investigate whether MuRF-1/TRIM63 rs2275950 was associated with the occurrence of muscle injury in professional soccer players. The present findings indicated that the gene investigated was unable to impact the occurrence of muscle injuries. Some factors such as genes responsible for encoding soft-tissue structure and regulatory proteins are well-documented and can affect the susceptibly to muscle injury in power sports (athletics) and

intermittent sports such as soccer (Lim et al., 2021; Murtagh et al., 2023). This gene investigated in isolation showed no significant association with muscle injuries. However, polygenic analysis may present results that explain the occurrence of muscle injuries in professional soccer players (Baumert et al., 2022; Maestro et al., 2022).

As the literature is scarce on research about MuRF-1/TRIM63 with muscle injuries in sports in general, the discussion described the physiological and structural aspects to explain the results of the study partially. Meanwhile, researchers conducted a study in a controlled environment with healthy and untrained individuals who showed incipient results associated with the MuRF-1/TRIM63 gene (Baumert et al., 2022; Stefanetti et al., 2014). Different responses to eccentric training could be observed by MuRF-1/TRIM63 genotypes in exercise-induced muscle damage biomarkers (Baumert et al., 2017). From these studies, a line of reasoning has emerged that the MuRF-1/TRIM63 gene may be associated with muscle injuries in samples of trained players of certain sports. Physical exercise can alter the gene expression of proteins related to the synthesis and degradation process (Baumert et al., 2022; Peris-Moreno et al., 2020). In addition, the protein MuRF-1 plays an important role in muscle protein turnover and net protein gain that is required for the skeletal muscle adaptation process following acute and chronic exercise (Stefanetti et al., 2014). Training causes extracellular stress signals that lead to acute transient changes in intramuscular signaling, influencing the gene transcription and protein translation that participate in the repair and remodeling of skeletal muscle during the recovery periods between exercise sessions (Baumert et al., 2022; Lim et al., 2021).

Other studies have shown that increased expression of MuRF-1 after muscle damage can increase the ubiquitination process of damaged myofibrillar and sarcoplasmic proteins (Baehr et al., 2021; Yang et al., 2022), thus contributing to successful tissue repair and promoting faster recovery. However, it is hypothesized that the restricted affinity of proteins (MuRF-1 and titin) could affect the interactions between the contractile and structural properties of muscle fibers, the muscle regeneration process and contribute to a less resistant muscle. This reduces its ability to withstand damaging eccentric actions and increases the susceptibility of the soccer player to suffer more significant damage to the sarcomere structure at times of greater physical demand and mechanical overload during training and matches in the season (Larruskain et al., 2018; Pinheiro et al., 2022). In soccer, poorly controlled training combined with insufficient recovery between training sessions and matches can predispose players to muscle damage and possibly increase the risk of muscle injury if the overload is not adjusted (Murtagh et al., 2023; Uchamocha et al., 2024).

The aforementioned evidence may support our hypothesis that AA homozygotes could suffer fewer muscle injuries. Nevertheless, the results of the present study showed in percentage values that the AA genotype (70%) was the most affected by muscle injuries during the period investigated. One possible explanation is that some phenotypes may experience greater muscle damage and require longer recovery following strenuous exercise, while other players recover more quickly despite performing the same exercise at a relatively similar intensity (Baumert et al., 2022; McAuley et al., 2022). Another point that should be considered is the participation of other genes that express proteins essential in the repair and regeneration of muscle tissue, the composition and maintenance of the external matrix and genes that mediate the contractile and structural properties of muscle fibers that significantly influence the complex process involved in muscle injury (Lim et al., 2021). In summary, it should be noted that our results are inconclusive and further studies investigating the relationship between MuRF-1/TRIM63 and muscle injuries could contribute to a better understanding of this genetic polymorphism.

The present study recorded 50 muscle injuries, of which 8% were considered mild, 42% moderate, and 50% severe. When comparing this data with the literature, Larruskain et al. (2018) described a total of 160 muscle injuries were recorded as minimal (23%), mild (34%), moderate (34%) and severe (15%) over six seasons. Clos et al. (2019) found that the 146 muscle injuries identified over seven seasons were mild (63%), moderate (34.9%), and severe (2.1%). Maestro et al. (2022) described a total of 121 injuries, 71 players had a non-contact injury. In terms of severity, there were slight (21.5%), mild (10.7%), moderate (46.3%), and severe (21.5%) over the 2021–2022 season. Therefore, the data from epidemiological studies in high-performance soccer indicated the prevalence of mild and moderate injuries. The results described do not corroborate the data from the present study, in which severe muscle injuries were the most common. These percentage differences may be due to the methodological design of each study, in which soccer players live in specific contexts and are subjected to different training and match loads (Pinheiro et al., 2022). Additional data revealed that even though muscle injuries represent a severe problem in soccer, it is observed that the preventive models and risk factors existing in the literature are not yet sufficient to promote a significant reduction in the number of muscle injuries in soccer (Clos et al., 2019; Ekstrand et al., 2023). Therefore, there is evidence base supporting the integration of genetic information to help reduce muscle injury in soccer through individualized training programs based on a player's genetic predispositions (Lim et al., 2021; Maestro et al., 2022; Moya, 2021).

Some limitations must be considered in this study. The absence of other genetic biomarkers compromises a more comprehensive analysis of the main genetic interactions with muscle injury in soccer players. In addition, clubs did not provide general parameters for controlling the training load. These data could be essential to understanding the relationship between training and match exposures in developing muscle injuries according to the genetic polymorphism investigated. Past research has indicated a higher risk

of muscle injury incidence during certain moments within a season (Pinheiro et al., 2022). Another point refers to the severity and recovery time of soccer players with muscle injuries, which can be influenced by aspects such as the history of injury, genetic profile, treatment strategies (Lim et al., 2021; Maestro et al., 2022), oral health (Uchamocha et al., 2024), playing positions, injured muscle site (Sinovas et al., 2020), physiological and psychological factors (Fagundes et al., 2019; Pinheiro et al., 2022), that should be considered when analyzing the results of the present study. Moreover, the sample size was limited for genetic studies. Restricted access to professional players and the expensive cost of genetic studies can also be considered limiting factors (Fagundes et al., 2019; Murtagh et al., 2023). Therefore, the results should be interpreted with caution, and a larger sample size of soccer players is recommended so that we can make more compelling statements and robust statistical treatments to detect an association for future scientific debates.

However, to the best of our knowledge, this is the first study to investigate the association of the MuRF-1/TRIM63 gene with muscle injuries in professional soccer. This study presents new information that can be added to the sports science literature about the structural factors that may influence the association between a specific genetic polymorphism and muscle injuries in soccer players from different geographic ancestries.

We have provided preliminary results that the AA genotype might be potentially predisposed not only with a higher risk of non-contact muscle injuries but also with a longer recovery time from these conditions to return to play. Genetics studies can help coaching staff, together with other information to identify players with a possible risk of muscle injury and, using this data, individualize preventive strategies, maximizing recovery processes to contribute to lower muscle injuries in the sport and increase player availability for training and competitions. Finally, future research is recommended to investigate the association between genetic polymorphisms and physiological aspects in high performance, considering that the genotype-phenotype interaction and the environment influence the different mechanisms of muscle injuries in soccer.

Conclusion

In the present study, with Brazilian professional soccer players, it is determined that the MuRF-1/TRIM63 genotypes do not serve as an effective biomarker for assessing the connection between genetic factors and muscle injuries.

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Disclosure statement

No potential conflict of interest was reported by the author(s).

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