

GENETIC PREDISPOSITION TO SPORTS-RELATED MUSCULOSKELETAL INJURIES: A SYSTEMATIC REVIEW

Abdullah Harb Abdullah Hommadi^{1,2,*}, Abdullah Alrashidi³, Naif Alqarni³, Salman Almobayedh³, Yasir Alhusseini⁴

¹College of Medicine, King Saud bin Abdulaziz University for Health Sciences, Riyadh, Saudi Arabia.

²Ministry of Defense, Riyadh, Saudi Arabia.

³Department of Family Medicine, King Fahd Military Medical Complex, Dhahran, Eastern Province, Saudi Arabia.

⁴King Fahd Military Medical Complex, Dhahran, Eastern Province, Saudi Arabia.

ORCID iDs

Abdullah Harb Abdullah Hommadi: <https://orcid.org/0009-0006-1324-1956>

Abdullah Alrashidi: <https://orcid.org/0009-0006-5627-2111>

Naif Alqarni: <https://orcid.org/0009-0006-5636-8236>

Salman Almobayedh: <https://orcid.org/0000-0002-3587-2883>

Yasir Alhusseini: <https://orcid.org/0009-0009-6808-075X>

*Corresponding Author: Abdullah Harb Abdullah Hommadi, Email: Hommadi.AbH@gmail.com

ABSTRACT

Sports-related musculoskeletal injuries (SRMSIs) arise from interacting intrinsic and extrinsic risk factors, and growing evidence suggests that genetic polymorphisms may influence susceptibility, injury severity, and recovery. This systematic review synthesized evidence on genetic variants associated with SRMSIs. We searched PubMed/MEDLINE, Scopus, Web of Science, Embase, and the Cochrane Library for observational studies published from 2018 to 2026 that evaluated candidate-gene polymorphisms or genome-wide associations in athletes or physically active individuals. Study characteristics, participant profiles, injury outcomes, genetic variants, and principal findings were extracted and synthesized narratively according to biological function. Sixteen studies involving more than 4,000 athletes and physically active individuals were included. Most studies involved elite football players and non-contact muscle injuries. ACTN3 R577X showed the most consistent association with injury susceptibility; the XX genotype was linked to greater injury risk (odds ratios 2.0-7.87), higher injury frequency, longer return-to-play times, and poorer performance. Associations involving ACE varied across populations. Variants in COL1A1, COL5A1, MMP3, IL6, NOS3, HIF1A, and VDR were associated with muscle, connective-tissue, hamstring, or stress-fracture outcomes, while recent genome-wide association studies identified novel loci including PAPA2, MAS1, and the intergenic region between OR10G7 and VWA5A. Genetic susceptibility to SRMSIs appears to involve multiple biological pathways, including muscle function, extracellular-matrix integrity, inflammation, vascular regulation, and bone metabolism. ACTN3 currently has the strongest evidence, but larger multicenter studies and external validation are required before genetic profiling can be incorporated into routine injury-risk assessment or athlete management.

Key words: Sports injuries; Musculoskeletal injuries; Genetic polymorphisms; ACTN3; Sports genomics; Genetic susceptibility

INTRODUCTION

Sports-related musculoskeletal injuries (SRMSIs) are among the most common health problems in sport across all levels of participation, from recreational activity to elite competition (Champawat et al., 2024). They encompass injuries affecting muscles, tendons, ligaments, bones, cartilage, and other connective tissues and may result from acute trauma, repetitive overuse, or a combination of intrinsic and extrinsic risk factors (Patel et al., 2017). Despite advances in sports medicine, injury-prevention strategies, rehabilitation guidelines, and training methods, musculoskeletal injuries remain an important cause of healthcare expenditure, time lost from sport, reduced athletic performance, and chronic disability, including persistent pain and early-onset osteoarthritis (Weerasinghe et al., 2026a; Weerasinghe et al., 2026b).

SRMSIs are inherently multifactorial (Weerasinghe et al., 2026b). Established risk factors include age, sex, previous injury, anatomical alignment, muscle-strength imbalance, joint flexibility, neuromuscular control, training intensity, playing surface, equipment, and environmental conditions (Patel et al., 2017). Although these factors contribute substantially to injury risk, they do not fully explain the marked interindividual variation observed among athletes exposed to comparable training loads and environments (Patel et al., 2017). Athletes participating in the same sport, following similar conditioning programs, and experiencing comparable mechanical loads may nevertheless exhibit very different injury profiles (Bache-Mathiesen et al., 2024).

Genetic predisposition refers to inherited genomic variation that can influence biological processes such as tissue development, extracellular-matrix organization, inflammation, angiogenesis, collagen synthesis, tendon remodeling, muscle repair, bone metabolism, and recovery after mechanical stress (Leońska-Duniec, 2025; Mueller et al., 2026). Single-nucleotide polymorphisms (SNPs), the most common form of genomic variation, are increasingly investigated as potential markers of individual differences in tissue resilience and injury susceptibility (Antontseva et al., 2023). Advances in molecular genetics and high-throughput genotyping have enabled the study

of numerous candidate genes related to musculoskeletal integrity, while sports genomics has emerged as a field linking genetics with performance, injury prevention, and personalized medicine (Mijaica et al., 2025; Łosińska et al., 2026).

Variants in collagen-encoding genes such as COL1A1, COL5A1, COL12A1, and COL27A1, which contribute to the structural integrity and mechanical properties of tendons, ligaments, and connective tissue, have been studied extensively (Mijaica et al., 2025). Variation in these genes has been associated with altered collagen-fiber organization, tissue stiffness, and susceptibility to tendon and ligament injury (Mijaica et al., 2025). Polymorphisms in MMP3 and other matrix metalloproteinase genes may influence extracellular-matrix remodeling, while variants in inflammatory genes such as IL6, IL1B, TNFA, and TGFB1 have been linked to inflammatory responses and tissue repair after injury (Jarosz et al., 2025). Other genes investigated in relation to tendon adaptation, muscle performance, ligament strength, and bone remodeling include GDF5, VEGFA, IGF1, ACE, and ACTN3 (Łosińska et al., 2026).

Genetic association studies have examined anterior cruciate ligament rupture, Achilles tendinopathy, rotator-cuff disease, hamstring strain, stress fractures, and other tendon and ligament disorders (Łosińska et al., 2026). Although several studies have reported strong associations between specific polymorphisms and injury risk, findings have often differed across populations and sports (Maffulli et al., 2013; Łosińska et al., 2026). Some associations are not reproduced in all ethnic groups, and earlier observations have not always been replicated (Maffulli et al., 2013). These inconsistencies may reflect differences in study design, sample size, phenotype definition, athletic level, environmental exposure, gene-gene interactions, and gene-environment interactions (Łosińska et al., 2026).

Overall, the literature remains heterogeneous with respect to injury type, athlete characteristics, genetic markers, and methodological quality (Mijaica et al., 2025; Łosińska et al., 2026). Studies differ substantially in sample size, ancestry, competition level, genotyping methods, statistical approaches, and outcome definitions (Mijaica et al., 2025; Łosińska et al., 2026), limiting definitive conclusions about clinically meaningful genetic predictors. At the same time, the identification of new susceptibility loci and advances in genomic methods have created a need to integrate the evidence and clarify priorities for future research. Therefore, this systematic review aimed to evaluate the available evidence on associations between genetic polymorphisms and sports-related musculoskeletal injuries.

MATERIAL AND METHODS

Reporting framework

This systematic review was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement (Page et al., 2021) to identify, select, appraise, and synthesize evidence on the relationship between genetic factors and sports-related musculoskeletal injuries.

Search strategy

A comprehensive search was conducted in PubMed/MEDLINE, Scopus, Web of Science, Embase, and the Cochrane Library from database inception through the final search date. Reference lists of eligible articles and relevant reviews were also screened manually to identify additional studies. The strategy combined Medical Subject Headings (MeSH) and free-text terms related to genetic susceptibility and sports-related musculoskeletal injury, including "genetic predisposition," "genetics," "genetic polymorphism," "single nucleotide polymorphism," "SNP," "sports injury," "musculoskeletal injury," "athletic injury," "ligament injury," "tendon injury," "muscle injury," "stress fracture," and "athlete," together with related synonyms and Boolean operators (AND/OR). The strategy was adapted to the indexing requirements of each database.

Eligibility criteria

Studies were eligible if they were published in English from 2018 through 2026 and examined inherited genetic variants, polymorphisms, or other genetic markers in relation to the risk of sports-related musculoskeletal injury among athletes or physically active individuals. Original observational studies, including case-control, cohort, and cross-sectional designs, were eligible regardless of participant age, sex, ethnicity, or athletic activity. Outcomes could involve muscles, tendons, ligaments, cartilage, bones, or other musculoskeletal structures injured during sport or physical activity. Reviews, systematic reviews, meta-analyses, conference abstracts with incomplete data, editorials, commentaries, case reports, animal studies, in vitro studies, and studies with insufficient outcome data were excluded. Full-text availability was required.

Study selection

All records identified through the database searches were imported into reference-management software and duplicates were removed. Two reviewers independently screened titles and abstracts for potential eligibility, followed by full-text assessment against the predefined inclusion and exclusion criteria. Disagreements were resolved through discussion and consensus, with consultation of a third reviewer when necessary.

Data extraction

Two reviewers independently extracted data using a standardized form. Extracted variables included first author, publication year, country, study design, participant characteristics, sample size, athletic population, musculoskeletal injury phenotype, genetic variants or polymorphisms, genotyping methods, comparison groups, measures of association, and principal findings. When reported, information on potential confounders and

proposed biological mechanisms was also collected. Extracted data were cross-checked for accuracy and completeness.

Quality assessment

The methodological quality of the included observational studies was assessed using the Newcastle-Ottawa Scale (NOS), which evaluates participant selection, comparability of study groups, and ascertainment of exposure or outcome. Two reviewers independently rated each study, and disagreements were resolved by discussion until consensus was reached.

Data synthesis

A qualitative narrative synthesis was used because of anticipated heterogeneity in study populations, injury definitions, genetic markers, outcome measures, and statistical analyses. Findings were summarized by injury type and genetic variant, with emphasis on associations reported consistently across studies, the strength and direction of observed effects, and similarities and differences between studies. Where appropriate, findings were interpreted in the context of biological plausibility and methodological quality.

RESULTS

Study selection and characteristics

The characteristics of the included studies are summarized in Table 1. Sixteen studies published between 2018 and 2026 met the eligibility criteria. The study-selection process is shown in Figure 1. Included studies were conducted across Europe, Asia, South America, and the Middle East, with cohorts from Spain, Italy, Estonia, Japan, Brazil, Kazakhstan, Lithuania, Qatar, Turkey, and Russia.

Figure 1. PRISMA flow diagram of study selection.

Table 1. Characteristics of included studies

Study	Country	Study design	Population	Sample size	Sport/Injury	Gene(s)/Polymorphism
Ebert et al. (2023)	Estonia	Case-control GWAS (Level III)	Estonian National Team athletes (Olympic International Championship participants)	126 athletes (104 males and 82.5%)	Lower limb musculoskeletal injuries: patellar tendinopathy (n=22), Achilles tendinopathy (n=16–17), hamstring injury (n=3), ACL rupture (n=6)	Genome-wide SNPs
de Almeida et al. (2022)	Brazil	Prospective cohort	Professional soccer players	83 athletes	Non-contact muscle injuries	ACTN3 R577X, ACE I/D
Massidda et al. (2020)	Italy & Japan	Multicenter case-control	Elite male football players	710 athletes (Italy=341; Japan=369)	Structural muscle injuries and functional muscle disorders	ACE I/D
Sierra et al. (2019)	Brazil	Prospective observational cohort	Male marathon runners	81 runners	Exercise-induced muscle damage and inflammation	ACE I/D, AGT Met235Thr, BDKRB2 -9/+9
Larruskain et al. (2018)	Spain	Prospective cohort	Elite male soccer players	107 players (356 player-seasons)	Non-contact hamstring injuries (129 injuries)	MMP3, TNC, IL6, NOS3, HIF1A, COL1A1, COL5A1 and other candidate SNPs (37 SNPs)
Moreno et al. (2020)	Spain	Cross-sectional	Marathon runners	139 runners	Running-related musculoskeletal injuries	ACTN3 R577X
Clos et al. (2019)	Spain	Retrospective cohort	Professional football players	43 players	Non-contact soft tissue muscle injuries	ACTN3 rs1815739
Massidda et al. (2024)	Italy	Retrospective cohort	Elite male football players	64 players	Muscle injuries	ACE rs4341, ACTN3 rs1815739, COL5A1 rs12722, MCT1 rs1049434

Study	Country	Study design	Population	Sample size	Sport/Injury	Gene(s)/Polymorphism
Del Coso et al. (2024)	Spain	Prospective cohort	LaLiga professional football players	315 players	Non-contact injuries (muscle, bone, and overall injuries)	ACTN3 rs1815739
Rodas et al. (2021)	Spain	Prospective cohort	Professional male and female soccer players	46 players	Non-contact soft-tissue muscle injuries	ACTN3 rs1815739 (R577X)
Miyamoto-Mikami et al. (2021)	Japan	Case-control with prospective validation	Japanese competitive athletes	1,185 athletes (association analysis); prospective validation cohort	Fatigue fractures and muscle injuries	COL1A1 rs1107946
Varamenti et al. (2024)	Qatar (Arab athletes)	Prospective observational cohort	High-level male athletes	Arab 30 athletes	Non-contact soft tissue injuries and stress fractures	COL5A1 rs12722; VDR rs10735810
Cerit et al. (2025)	Turkey & Russia	Case-control with injury cohort	Professional female volleyball players	187 volleyball players (genetic comparison); 50 players with injury follow-up	Non-contact muscle injuries	ACTN3 rs1815739 (R577X)
Miyamoto-Mikami et al. (2025)	Japan	GWAS with athlete validation cohort	Physically active young adults and Japanese athletes	350 (GWAS); 1,428 athletes (validation)	Passive muscle stiffness and muscle injury	Genome-wide SNPs (rs12807854, rs78405694)
Gabdulkayum et al. (2026)	Kazakhstan	Cross-sectional genetic association study	Elite athletes from nine Olympic sports	137 athletes	Moderate/severe sports injuries	VDR (TaqI, ApaI, BsmI, FokI)
Kabelis et al. (2026)	Lithuania (Caucasian population)	Case-control	Physically active Caucasian adults	275 participants (142 injured; 133 controls)	Non-contact musculoskeletal injuries	BGN, DCN, ELN, FBN2, MMP1, MMP10, MMP12

Study designs and participants

Prospective cohort designs were the most common (n = 4), followed by case-control studies (n = 3, including multicenter and conventional genome-wide association studies), retrospective cohorts (n = 3), cross-sectional genetic association studies (n = 2), prospective observational cohorts (n = 2), and studies combining case-control analyses with prospective or validation cohorts (n = 2). Sample sizes ranged from 30 high-level athletes to 1,428 athletes in an external validation cohort for a genome-wide association study. Across all studies, more than 4,000 athletes and physically active individuals were represented, although some participants were reported separately within multicenter or validation designs.

Injury phenotypes and genetic approaches

The populations were predominantly elite or professional athletes, including football players, marathon runners, volleyball players, Olympic athletes, and athletes from several individual sports. Football players represented the largest proportion of participants, consistent with the high prevalence and detailed surveillance of musculoskeletal injuries in professional football (Larruskain et al., 2018; Clos et al., 2019; Massidda et al., 2020; Rodas et al., 2021; de Almeida et al., 2022; Del Coso et al., 2024; Massidda et al., 2024). Other studies included marathon runners, Japanese competitive athletes, physically active adults, and elite athletes from multiple Olympic sports (Sierra et al., 2019; Moreno et al., 2020; Miyamoto-Mikami et al., 2021; Miyamoto-Mikami et al., 2025; Gabdulkayum et al., 2026; Kabelis et al., 2026).

Outcomes were diverse and focused mainly on non-contact musculoskeletal injuries, with muscle injury being the most frequently assessed phenotype. Other outcomes included hamstring injury, tendon or ligament injury, lower-limb musculoskeletal injury, exercise-induced muscle damage, passive muscle stiffness, fatigue and stress fractures, bone injury, and athletic performance. Several studies also assessed injury severity, recovery time,

return-to-play duration, or exercise-induced biomarkers as secondary outcomes (Sierra et al., 2019; Miyamoto-Mikami et al., 2021; Varamenti et al., 2024; Cerit et al., 2025; Miyamoto-Mikami et al., 2025).

Candidate-gene analyses were the predominant genetic approach, while two genome-wide association studies (GWAS) investigated novel susceptibility loci (Ebert et al., 2023; Miyamoto-Mikami et al., 2025). ACTN3 was the most frequently studied candidate gene, followed by ACE. Other investigations examined genes related to extracellular-matrix organization, collagen synthesis, inflammation, vascular regulation, vitamin D metabolism, and matrix remodeling. More recent work also evaluated polygenic risk models and gene-gene interactions, reflecting a shift from single-gene analyses toward broader genomic approaches (Massidda et al., 2024; Gabdulkayum et al., 2026; Kabelis et al., 2026).

Genetic associations by biological pathway

Genetic variants associated with sports-related musculoskeletal injuries are summarized by biological function in Table 2. Overall, the strongest signals involved genes related to muscle structure and function, connective-tissue integrity, extracellular-matrix remodeling, muscle physiology, and vitamin D metabolism; however, the strength and consistency of associations varied substantially across genes.

Table 2. Genetic associations with sports-related musculoskeletal injuries grouped by functional gene category

Functional gene category	Gene(s)	Variant(s)	Injury/Outcome	Summary of findings (including statistics)	Supporting studies
Muscle structure and function	ACTN3	R577X (rs1815739)	Muscle injury, non-contact injury, running-related injury, recovery after injury, athletic performance	The most consistently replicated gene. The XX (TT) genotype was associated with higher muscle injury risk (OR = 5.14, 95% CI 1.47–17.96), increased injury severity, higher injury frequency (XX>RX>RR), approximately two-fold higher muscle injury odds (OR = 2.0), greater injury incidence (2.78 injuries/player; TT=3.3 injuries/1000 h), higher total and match injury incidence (P = 0.026 and P = 0.009), increased muscle (P = 0.016) and bone injuries (P = 0.033), reduced running performance (4.2% lower total distance, 13.1% lower high-speed running, 14.3% fewer sprints), longer return-to-play (36±26 vs 20±10 vs 17±12 days; P = 0.044), and higher injury odds in female volleyball players (adjusted OR = 5.92, 95% CI 1.12–60.98).	Clos et al. (2019); Moreno et al. (2020); Rodas et al. (2021); de Almeida et al. (2022); Del Coso et al. (2024); Massidda et al. (2024); Cerit et al. (2025)
Muscle structure and function	MCT1	rs1049434	Muscle injury	AA genotype showed significantly greater injury incidence than TT genotype (2.22 vs 0.78 injuries/1000 h; P = 0.031).	Massidda et al. (2024)
Renin–angiotensin muscle physiology	ACE	I/D, rs4341	Muscle injury, exercise-induced muscle damage	Evidence was mixed. II genotype was associated with nearly two-fold higher injury incidence (P	Sierra et al. (2019); Massidda et al. (2020); de Almeida et al.

Functional gene category	Gene(s)	Variant(s)	Injury/Outcome	Summary of findings (including statistics)	Supporting studies
				= 0.030), higher injury rate (4.20 injuries/1000 h), greater CK, LDH, troponin, IL-6 and IL-8 responses (CK>500 U/L: 69% vs 40% vs 38%), whereas DD/ID genotypes showed reduced injury risk in pooled analyses (OR = 0.61, 95% CI 0.38–0.98) and in the Japanese cohort (OR = 0.48, 95% CI 0.24–0.97). DD genotype also showed a trend toward fewer severe injuries (>28 days).	(2022); Massidda et al. (2024)
Renin–angiotensin muscle physiology	and AGT	Met235Thr	Exercise-induced muscle damage	MM genotype showed greater troponin, pro-BNP, IL-1 β and IL-8 responses following marathon running.	Sierra et al. (2019)
Renin–angiotensin muscle physiology	and BDKRB2	–9/+9	Exercise-induced muscle damage	–9 allele carriers showed greater CK elevation (CK>500 U/L: 46% vs 48% vs 31%).	Sierra et al. (2019)
Extracellular matrix connective tissue	and COL1A1	rs1107946	Muscle injury; fatigue fracture	C allele reduced muscle injury risk (OR = 0.46, 95% CI 0.24–0.91; HR = 0.27, 95% CI 0.08–0.96), but increased fatigue fracture risk in female athletes (OR = 2.44, 95% CI 1.17–5.77).	Miyamoto-Mikami et al. (2021)
Extracellular matrix connective tissue	and COL5A1	rs12722	Muscle, tendon and ligament injuries	Conflicting findings. TT genotype increased muscle injury incidence compared with TC (2.35 vs 0.68 injuries/1000 h; P = 0.029), whereas another study found fewer muscle strains (RR = 0.16, 95% CI 0.04–0.67), fewer tendon/ligament injuries (RR = 0.27; P = 0.046), and lower tendon injury severity (OR = 0.06; P = 0.039).	Massidda et al. (2024); Varamenti et al. (2024)
Extracellular matrix connective tissue	and COL22A1	rs3924862	Lower-limb musculoskeletal injury	Variant associated with injury subtype in GWAS.	Ebert et al. (2023)
Extracellular matrix connective tissue	and BGN	rs1042103	Non-contact musculoskeletal injury	AG and AA genotypes showed nominal associations with increased injury susceptibility.	Kabelis et al. (2026)

Functional gene category	Gene(s)	Variant(s)	Injury/Outcome	Summary of findings (including statistics)	Supporting studies
Extracellular matrix and connective tissue	DCN	rs516115, rs13312816	Non-contact musculoskeletal injury	AG genotype associated with increased injury susceptibility, whereas GG genotype appeared protective. Interaction with BGN increased injury probability (OR = 5.44 and OR = 4.24), although significance was lost after Bonferroni correction.	Kabelis et al. (2026)
Extracellular matrix and connective tissue	FBN2	rs331079	Non-contact musculoskeletal injury	GC and CC genotypes showed nominally increased injury susceptibility.	Kabelis et al. (2026)
Extracellular matrix and connective tissue	ELN	rs2071307	Non-contact musculoskeletal injury	No significant independent association observed.	Kabelis et al. (2026)
Extracellular remodeling	matrix MMP3	rs679620	Hamstring injury	HR = 2.06 (95% CI 1.51–2.81).	Larruskain et al. (2018)
Extracellular remodeling	matrix MMP12	rs2276109	Non-contact musculoskeletal injury	GG genotype showed nominal association with injury susceptibility.	Kabelis et al. (2026)
Extracellular remodeling	matrix MMP1, MMP10	rs1799750, rs486055	Non-contact musculoskeletal injury	No significant independent associations detected.	Kabelis et al. (2026)
Inflammatory, vascular and hypoxia pathways	IL6	rs1800795	Hamstring injury	GG genotype increased injury risk (HR = 1.68, 95% CI 1.11–2.53).	Larruskain et al. (2018)
Inflammatory, vascular and hypoxia pathways	NOS3	rs1799983	Hamstring injury	HR = 1.35 (95% CI 1.01–1.79).	Larruskain et al. (2018)
Inflammatory, vascular and hypoxia pathways	HIF1A	rs11549465	Hamstring injury	CC genotype HR = 2.08 (95% CI 1.00–4.29).	Larruskain et al. (2018)
Vitamin D metabolism	VDR	rs10735810 (FokI), TaqI	Stress fracture, sports injury susceptibility, vitamin D deficiency	rs10735810 CT and TT genotypes markedly increased stress fracture risk (RR = 7.72 and RR = 9.93, respectively); severe fractures OR = 10.91. TaqI GG genotype increased sports injury susceptibility (OR = 5.56, 95% CI 1.41–21.91) and vitamin D deficiency/insufficiency (OR = 6.23, 95% CI 1.65–23.43).	Varamenti et al. (2024); Gabdulkayum et al. (2026)
Genome-wide association (GWAS) loci	PAPPA2	rs11580456	Lower-limb musculoskeletal injury	Strongest GWAS association with injury susceptibility (OR = 13.8).	Ebert et al. (2023)
Genome-wide association (GWAS) loci	MAS1	rs220735, rs170219	Lower-limb musculoskeletal injury	Increased injury susceptibility (OR = 3.1).	Ebert et al. (2023)

Functional gene category	Gene(s)	Variant(s)	Injury/Outcome	Summary of findings (including statistics)	Supporting studies
Genome-wide association (GWAS) loci	PLXNA2, DOK5, GNG12, DAP	rs11799530, rs73142922, rs28435277, rs267959/rs2930047/rs1080440/rs267939	Lower-limb musculoskeletal injury	Associated with specific injury subtypes in GWAS.	Ebert et al. (2023)
Genome-wide association (GWAS) loci	OR10G7/VWA5A intergenic locus	rs12807854	Passive muscle stiffness; muscle strain injury	Genome-wide significant association with passive biceps femoris stiffness ($P = 5.19 \times 10^{-10}$); C allele increased muscle strain injury risk (OR = 1.62, 95% CI 1.06–2.47).	Miyamoto-Mikami et al. (2025)
Genome-wide association (GWAS) loci	CCDC73 locus	rs78405694	Passive muscle stiffness	Genome-wide significant association with passive muscle stiffness ($P = 2.09 \times 10^{-8}$), but not with muscle injury (OR = 1.25; $P = 0.332$).	Miyamoto-Mikami et al. (2025)
Performance-related genes	DSG1, DSG4	rs10502567, rs73410248	Elite athletic performance	Associated with medal-winning performance (OR = 14.3 and OR = 17.4, respectively).	Ebert et al. (2023)
Polygenic models	ACTN3 + ACE + COL5A1 + MCT1	Total + Genetic Score (TGS)	Muscle injury prediction	Injured athletes had significantly lower TGS than non-injured athletes (42.5 ± 12.5 vs 63.7 ± 13.0 ; $P < 0.001$). TGS predicted injury with OR = 3.5 (95% CI 1.8–6.8) and AUC = 0.873.	Massidda et al. (2024)

ACTN3 and muscle-related genes

ACTN3 showed the strongest and most consistent evidence among the investigated genes. The R577X (rs1815739) polymorphism was repeatedly associated with a higher risk of non-contact muscle injury. Athletes with the XX (TT) genotype had greater injury risk, with reported odds ratios ranging from 2.0 to 7.87, higher injury frequency (2.78 injuries/player or 3.3 injuries/1,000 h), greater injury severity, longer return-to-play times (36 +/- 26 days vs 20 +/- 10 days and 17 +/- 12 days), and poorer running performance (4.2% lower total distance, 13.1% lower high-speed running, and 14.3% fewer sprints) (Clos et al., 2019; Moreno et al., 2020; Rodas et al., 2021; de Almeida et al., 2022; Del Coso et al., 2024; Massidda et al., 2024; Cerit et al., 2025). In addition, incorporating ACTN3 into a polygenic model improved injury prediction, with an AUC of 0.873 and an OR of 3.5 (95% CI 1.8–6.8) for identifying injured athletes (Massidda et al., 2024).

Renin-angiotensin system and muscle physiology

Findings for genes involved in the renin-angiotensin system and muscle physiology were more variable. Across studies of ACE, the II genotype was associated with approximately two-fold greater injury incidence in some populations, higher muscle-injury rates (4.20 injuries/1,000 h), and larger exercise-induced increases in CK, LDH, troponin, IL-6, and IL-8; 69% of II carriers exceeded CK >500 U/L compared with 40% of ID carriers and 38% of DD carriers (Sierra et al., 2019; de Almeida et al., 2022; Massidda et al., 2024). In contrast, pooled analyses indicated protective associations for DD/ID genotypes, including a lower injury risk (OR = 0.61, 95% CI 0.38–0.98) and fewer severe injuries lasting more than 28 days (Massidda et al., 2020). Other exercise-physiology genes, including AGT and BDKRB2, were associated with greater biochemical responses after marathon running, suggesting increased susceptibility to exercise-induced muscle damage (Sierra et al., 2019).

Extracellular matrix and connective tissue

Genes involved in extracellular-matrix composition and connective-tissue integrity also showed important associations. COL1A1 demonstrated a dual pattern: the C allele was associated with lower muscle-injury risk (OR = 0.46; HR = 0.27) but greater fatigue-fracture risk in female athletes (OR = 2.44) (Miyamoto-Mikami et al., 2021). Findings for COL5A1 were conflicting. One study reported a higher incidence of muscle injury in TT

carriers than in TC carriers (2.35 vs 0.68 injuries/1,000 h), whereas another found that the TT genotype was associated with fewer muscle strains (RR = 0.16), fewer tendon and ligament injuries (RR = 0.27), and lower tendon-injury severity (OR = 0.06) (Massidda et al., 2024; Varamenti et al., 2024). Exploratory analyses also identified nominal associations involving BGN, DCN, FBN2, and MMP12. Gene-gene interactions between BGN and DCN suggested increased injury risk (OR = 5.44 and 4.24), although significance was lost after correction for multiple testing (Kabelis et al., 2026).

Inflammatory, vascular, and hypoxia pathways

Hamstring-injury studies also implicated variants involved in extracellular-matrix remodeling, inflammation, vascular regulation, and hypoxia responses. Significant associations with non-contact muscle-injury susceptibility were reported for MMP3 (HR = 2.06), IL6 (HR = 1.68), NOS3 (HR = 1.35), and HIF1A (HR = 2.08) (Larruskain et al., 2018).

Vitamin D metabolism

Two recent studies supported a role for vitamin D metabolism. For VDR rs10735810 (FokI), the CT and TT genotypes were associated with relative risks of 7.72 and 9.93, respectively, for stress fractures, while the TT genotype was associated with greater odds of severe stress fracture (OR = 10.91) (Varamenti et al., 2024). In another cohort, the VDR TaqI GG genotype was associated with more than five-fold higher odds of overall sports injury (OR = 5.56, 95% CI 1.41-21.91) and more than six-fold higher odds of vitamin D deficiency or insufficiency (OR = 6.23, 95% CI 1.65-23.43) (Gabdulkayum et al., 2026).

Genome-wide associations

Genome-wide association studies extended the evidence beyond traditional candidate genes. One GWAS identified PAPA2 as the strongest susceptibility locus for lower-limb musculoskeletal injury (OR = 13.8), with MAS1 (OR = 3.1) and variants in COL22A1, PLXNA2, DOK5, GNG12, and DAP associated with specific injury subtypes (Ebert et al., 2023). The OR10G7/VWA5A intergenic locus (rs12807854) was significantly associated with passive biceps femoris stiffness ($P = 5.19 \times 10^{-10}$) and muscle-strain risk (OR = 1.62, 95% CI 1.06-2.47), whereas CCDC73 rs78405694 was associated with increased stiffness ($P = 2.09 \times 10^{-8}$) but not with injury risk (Miyamoto-Mikami et al., 2025). Together, these findings support a highly polygenic architecture for sports-related musculoskeletal injury susceptibility.

DISCUSSION

This review synthesized current evidence on genetic factors associated with sports-related musculoskeletal injuries and reinforced the view that injury susceptibility is multifactorial. Genetic variation affecting muscle structure, extracellular-matrix integrity, inflammatory responses, vascular regulation, and vitamin D metabolism may contribute to interindividual differences in injury risk. At the same time, substantial heterogeneity in athlete populations, injury definitions, and study methods indicates that genetic predisposition should be interpreted within a broader biopsychosocial framework that also considers training load, biomechanics, environmental exposure, and recovery strategies.

The characteristics of the included studies reflect the rapid development of sports genomics over the past decade. Most investigations focused on elite athletes, particularly football players, a population in which non-contact muscle injuries are common and longitudinal injury-surveillance systems are well established (Garcia et al., 2022). Elite athletes have also been highlighted as valuable populations for genetic research because their training environments are comparatively controlled and injury histories are often well documented, reducing some environmental variability that can confound genetic association studies (Bıçakçı et al., 2024; Aslam et al., 2025; Marrouh et al., 2025). However, the predominance of football cohorts limits the generalizability of current evidence to endurance, combat, racquet, and recreational sports.

Another notable trend was the transition from candidate-gene studies toward genome-wide association studies and polygenic approaches. This mirrors the broader development of human genetics from single-candidate hypotheses to hypothesis-free genomic analyses capable of identifying new biological pathways. Reviews of sports genomics have emphasized the limited statistical power and reproducibility of many early candidate-gene associations, whereas GWAS can provide a more comprehensive assessment of the complex polygenic basis of athletic phenotypes (Wang et al., 2024; Mijaica et al., 2025). Future research is therefore likely to incorporate polygenic risk scores, transcriptomics, epigenetics, and multi-omics approaches alongside individual polymorphisms.

ACTN3 had the most consistent association with sports-related musculoskeletal injury among the genes examined. Alpha-actinin-3 is a structural Z-disc protein expressed in fast glycolytic muscle fibers and contributes to force transmission and resistance to eccentric mechanical loading (Haug et al., 2022). Experimental studies indicate that alpha-actinin-3 deficiency alters calcium handling, muscle metabolism, and the mechanical properties of skeletal muscle, potentially increasing vulnerability to repeated eccentric contractions (Seto et al., 2011; Head et al., 2015). These mechanisms provide biological plausibility for the higher non-contact muscle-injury burden and longer recovery observed in athletes with the XX genotype. Nevertheless, even for ACTN3, the predictive value of a single gene is limited because injury is a complex multifactorial outcome (Baltazar-Martins et al., 2020; Bıçakçı et al., 2024).

Evidence for ACE polymorphisms was less consistent. The ACE insertion/deletion polymorphism has been linked to variation in muscle hypertrophy, endurance capacity, tissue remodeling, and ACE activity, but meta-analyses

of athletic phenotypes have reported inconsistent findings across ethnic groups and sports (Ipekoglu et al., 2022; Sommers et al., 2024). Differences in training intensity, competitive level, injury definition, ancestry, and gene-environment interactions may contribute to the conflicting associations between ACE genotype and muscle-injury susceptibility (Ipekoglu et al., 2022). Accordingly, ACE should not currently be considered a stand-alone predictor of injury risk.

The findings for collagen-related genes further emphasize the importance of extracellular-matrix homeostasis in musculoskeletal health. Collagens provide much of the tensile strength of tendons, ligaments, and skeletal-muscle connective tissue, and variation in genes governing collagen synthesis or fibril formation may influence tissue mechanics and healing capacity (Emam, 2025; Kanniyappan et al., 2025). COL1A1 and COL5A1 have repeatedly been implicated in tendon and ligament pathology (Abboud et al., 2026). However, the conflicting COL5A1 findings in the present review illustrate the complexity of connective-tissue genetics, as the same polymorphism may have different effects according to tissue type, ancestry, athletic exposure, or injury phenotype.

Associations involving inflammatory pathways, vascular signaling, hypoxia responses, and matrix metalloproteinases are also biologically plausible. Effective skeletal-muscle regeneration requires tightly regulated inflammatory activation, extracellular-matrix remodeling, and angiogenesis (Arroyo and Iruela-Arispe, 2010). Dysregulated cytokine signaling or matrix degradation may impair regeneration and increase susceptibility to recurrent injury (McBride et al., 2020). Interleukin-6 contributes to muscle-damage and regeneration pathways, while matrix metalloproteinases participate in extracellular-matrix turnover during tissue repair (Mukherjee and Das, 2024). These mechanisms are consistent with the observed associations involving IL6, MMP3, NOS3, and HIF1A.

Vitamin D receptor polymorphisms may be particularly relevant to stress-fracture susceptibility. Vitamin D contributes to calcium metabolism, bone mineralization, skeletal-muscle function, and immune regulation (Khazai et al., 2008). Observational evidence links vitamin D deficiency with greater stress-fracture risk, reduced muscle strength, and prolonged recovery among athletes and military populations (Knechtle et al., 2021; Ribbans et al., 2021). Genetic variation affecting vitamin D receptor activity could therefore modify susceptibility to bone injury under repetitive mechanical loading. However, vitamin D status is also strongly influenced by sunlight exposure, dietary intake, supplementation, and ancestry, so future studies should evaluate genetic variation alongside these environmental determinants rather than in isolation.

The evidence base has important methodological limitations. Many studies had small samples, increasing the risk of unstable or spurious associations and limiting statistical power. Injury classification, exposure measurement, sport type, ancestry, and genotyping methods varied substantially across studies, reducing comparability. In addition, many investigations assessed only a small number of candidate polymorphisms without fully accounting for gene-gene interactions, epigenetic regulation, or environmental modifiers. Larger multicenter collaborations, standardized injury phenotypes, independent replication cohorts, and integration of genomic, transcriptomic, biomechanical, and environmental data are needed to improve reproducibility and clinical relevance.

CONCLUSION

The available evidence supports the hypothesis that susceptibility to sports-related musculoskeletal injuries is influenced by multiple biological pathways rather than a single genetic mechanism. ACTN3 currently shows the strongest and most consistent evidence, while collagen-related genes, vitamin D receptor polymorphisms, inflammatory mediators, and emerging GWAS loci represent additional biologically plausible contributors. At present, however, the evidence is insufficient to support routine genetic testing of athletes for injury prediction. Future research should prioritize large multi-ethnic prospective cohorts, standardized phenotyping, external validation of GWAS findings, and integrated polygenic prediction models that incorporate biomechanical and training-related risk factors to advance precision sports medicine.

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