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Genetic Testing for Injury Prevention in Sport: Hype or Reality?

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ABSTRACT

Participation in physical activity and sport confers numerous health benefits but is frequently associated with acute and chronic musculoskeletal injuries, including tendon, ligament, muscle, and bone injuries. While rare musculoskeletal conditions result from single-gene mutations, most sports injuries are multifactorial, arising from interactions between intrinsic and extrinsic factors. Increasing evidence suggests a polygenic component, with heritability contributing to individual variation in injury susceptibility. Research has explored genetic risk factors using candidate gene studies, genome-wide association studies, and next-generation sequencing. However, these approaches have limitations: associations are often not independently replicated, risk alleles may be rare or population-specific, and clinical sensitivity and specificity are generally inadequate. Non-coding RNAs and epigenetic modifications further complicate the genetic architecture of injury susceptibility. Despite this, direct-to-consumer genetic tests claim to predict musculoskeletal injury risk and concussion susceptibility, yet scientific evidence does not support their clinical validity. Personalized medicine, however, is achievable without genetic testing, through individualized assessment and management of modifiable risk factors. Large, well-powered, and rigorously phenotyped studies with replication across diverse populations are required before genetic tests can be considered reliable predictors of injury risk. Until then, clinicians can implement precision strategies to reduce injury risk, highlighting that personalized injury prevention is feasible today, even in the absence of validated genetic markers.

Introduction

Although physical activity offers numerous health benefits, participation is commonly associated with acute and chronic musculoskeletal injuries [1]. Tendon and ligament injuries, for instance, have been reported to account for approximately 30 to 50% of all sports-related injuries, while muscle injuries account for approximately 10 to 55% of acute injuries [2,3]. While some rare musculoskeletal conditions result from single-gene mutations [4-6], most sports injuries are multifactorial, arising from a combination of intrinsic and extrinsic risk factors [7]. A growing body of evidence indicates that these injuries also have a polygenic component, with significant heritability contributing to the observed inter-individual variation in injury susceptibility [8-11]. For example, heritability estimates for anterior cruciate ligament (ACL) rupture have been reported to reach up to 69%, while estimates for tennis elbow are 40% [12,13]. Traditionally, injury risk is considered to result from complex gene-gene interactions and the interplay between an individual's genetic makeup and factors such as training load, nutrition, and other sport-specific variables, with the role of epigenetic modifications in this model briefly discussed in this perspective. Over the last two decades, research has begun to identify genetic risk factors associated with common tendon, ligament, bone, and muscle injuries [8-11]. Progress, however, has been slow, and a complete genetic profile of injury risk remains elusive. Consequently, the specific genes that most strongly modulate injury susceptibility have yet to be determined.

Despite limited scientific knowledge and the essential role of health care professionals in this process, several direct-to-consumer (DTC) genetic testing companies have emerged claiming to assess susceptibility to sport- and exercise-related injuries. These claims range from broad non-specific statements, such as "soft tissue injury risk", "injury predisposition" and "risk for inflamation and injury" to assertions about specific injuries, including "risk of stress fracture", "Achilles injury risk," and "ACL injury risk" [14]. While numerous articles describe and warn against the growth of this industry, few critically examine the scientific validity of the genetic markers included in these commercial tests [15-17]. Nevertheless, some authors have expressed support for the industry and there have been reports that a professional football club has used genentic testing for injury prevention [18,19]. Although many companies do not disclose which markers they assess, a recent review evaluated the evidence for commonly used polymorphisms in DTC musculoskeletal injury tests. The authors concluded that it is currently premature to market any commercial genetic test as a reliable predictor of susceptibility to common musculoskeletal injuries. [14].

Methodologies used to Identify Genetic Markers

Two broad approaches have been used to identify genetic markers associated with sports injuries. To date, most studies have employed a hypothesis-driven candidate gene, case-control genetic association study approach, although genome-wide association study (GWAS) and next-generation sequencing (NGS) approaches have been used more recently.

In the candidate gene approach, investigators select genes and polymorphisms based on known biological function and the hypothesis that the gene product is involved in the biological mechanisms underlying injury pathogenesis. For example, early studies reporting genetic associations with chronic Achilles tendinopathy and ACL rupture initially focused on collagen-encoding genes, specifically *COL5A1* and *COL1A1* [20-23]. These genes were selected because they encode the α1 chains of type V and type I collagen, respectively. Types I and V, as well as other collagen types, are key structural components of the collagen fibril, which is the basic building block of tendons, ligaments, and other musculoskeletal connective tissues [10]. These genes were also considered strong candidates because mutations in *COL5A1* and *COL1A1* cause Mendelian connective tissue disorders, such as Ehlers-Danlos syndrome and osteogenesis imperfecta, respectively. It was therefore argued that, while rare mutations cause severe connective tissue disorders, common polymorphisms within the same genes may contribute to susceptibility to multifactorial connective tissue injuries [24]. The polymorphisms selected in candidate gene studies are typically functional; for example, the polymorphisms investigated within *COL1A1* and *COL5A1* are located in regulatory regions of these genes [25,26]. Even though several DTC companies include *COL1A1* and *COL5A1* polymorphisms as risk markers for susceptibility to musculoskeletal injuries, a recent review of the scientific evidence did not support their inclusion in these commercial tests [14].

Since the initial focus on collagen-encoding genes, subsequent studies have examined a small number of other genes regulating tendon and ligament structure and function, including glycoproteins, proteoglycans, matrix-degrading enzymes, cytokines, and proteins involved in apoptosis and angiogenesis [10]. To date, only a limited number of genes have been associated with muscle injuries. These genes encode, among other things, structural components of skeletal muscle and the associated extracellular matrix, as well as regulators of muscle growth, development, regeneration, metabolism and perfusion [9].

While the human genome contains approximately 20,000 protein-coding genes, representing only 1 to 2% of the entire genome, as mentioned above, the association between a small subset of these genes and musculoskeletal injuries has been investigated primarily using candidate gene approaches. In addition, it is estimated that over 85% of the human genome is transcribed into non-coding RNAs (ncRNAs). Non-coding RNAs, such as microRNAs (miRNAs) and long non-coding RNAs (lncRNAs), also influence musculoskeletal development, injury, and repair, and polymorphisms in these genes can also affect injury susceptibility [11,27]. However, candidate gene approaches to date have not focused on these genes. Because musculoskeletal injuries are polygenic and multifactorial, commercial tests that assess only a few protein-coding genes lack clinical value.

In hypothesis-free GWAS and NGS approaches, entire genomes or targeted regions are sequenced, and millions of polymorphisms are analyzed across cases and controls to identify variants associated with injuries. Currently,

only a single company markets DTC tests using injury-specific polymorphisms identified through GWAS. A recent review of the scientific evidence for tests related to rotator cuff injury, runner's knee, Achilles tendon injury, cruciate ligament injury, ankle injury, plantar fasciitis, iliotibial band syndrome, patellar tendinopathy, de Quervain's tenosynovitis, medial collateral ligament injury, and shoulder instability did not support their clinical validity [14]. A major concern identified was that the associations of the polymorphisms had not been independently confirmed, and most of the risk alleles were rare or absent in several non-European populations. In addition, the sensitivity of many markers was low and not clinically relevant.

Sport-Related Concussion

In addition to musculoskeletal injuries, athletes participating in combat and collision sports are at a high risk of sports-related concussion. These injuries are potentially serious and there is evidence to suggest a genetic contribution to concussion susceptibility and recovery [11,28,29]. However, the genetic literature in this area remains limited and is dominated by candidate gene studies, many of which examine polymorphisms in genes related to concussion risk, personality traits, injury severity, neural plasticity and repair, and post-concussion cognitive and behavioural outcomes [11,28]. Although two genetic markers - rs144663795 (G>C) in the *SPATA5* gene and rs117985931 (A>G) in the *PLXNA4* gene - have been identified using a GWAS approach [30], their clinical relevance remains unclear. Specifically, the effective rs144663795 C allele is rare and is present only in 0.8% of the European and 0.4% of the South Asian populations, while the effective rs117985931 G allele is also rare and is present only in the 0.1% of the European population, 0.1% of one African subpopulation, and 0.3 % of the one American sub-population (www.ensembl.org, accessed 10 February 2026). Despite this, these markers are reportedly incorporated into a direct-to-consumer commercial genetic test marketed for concussion risk assessment (www.axgen.us/science; www.axgen.us/what-you-get; accessed 4 February 2026). The prevailing consensus within the literature is that genetic testing for sports-related concussion is premature, and that substantial further research, including large, well-powered, and independently replicated studies, is required before genetic markers can be meaningfully applied to concussion risk stratification or management [11,28,29,31-33].

Reproducibility, Specificity, Sensitivity and Other Considerations

The inclusion of genetic markers in sport injury tests is only warranted if there is strong, reproducible, reliable, and independent scientific evidence supporting the association. Markers also need to have been tested in multiple population groups and shown to be universally applicable. Unfortunately, this is currently not the case, as many published candidate gene studies are limited by small sample sizes, often using predominantly European populations, and have frequently failed to be consistently reproduced in subsequent studies [14]. Similarly, as previously mentioned, the GWAS approach also has limitations: larger sample sizes are often achieved at the expense of clinically well-defined, homogeneous cases and controls [14]. This is illustrated by GWAS findings in which three polymorphisms were associated with anterior and posterior cruciate ligament injuries [34]. Although the cruciate ligament injury cases were heterogeneous, two of the polymorphisms were included in a DTC test to assess anterior and posterior cruciate ligament injury risk [14]. We recently showed that none of the three polymorphisms were associated with injury in multiple clinically well-characterised cohorts of 906 participants with ACL rupture [35]. In addition, when more than one GWAS has been published, different polymorphisms have been identified for the same injury, highlighting the importance of confirming associations in clinically well-characterized cases [34,36]. To my knowledge none of the other injury tests using GWAS findings have been idependently confirmed.

Many of the effective alleles or genotypes included as injury markers in several DTC tests are rare in a limited number of populations and/or absent in some population groups [14]. For example, the functional *COL1A1* rs1800012 TT genotype, which has been associated with a reduced risk of ACL rupture in some studies, is absent in the East Asian population (www.ensembl.org, accessed 10 February 2026) [37]. Although the TT genotype is rare in European populations, only 4.4%, it is even rarer in American, only 2.3%, South Asian, only 1.2%, and African, only 0.5%, populations (www.ensembl.org, accessed 10 February 2026). This limited distribution likely explains why the TT genotype has only been associated with a reduced risk of ACL rupture in European populations [37]. Any genetic marker included in a clinical test should be informative across all population groups.

Although rare effective alleles and genotypes can be highly specific, they typically have low sensitivity, leading to false negatives - that is, they fail to identify the majority of individuals at risk. For example, the specificity of the *COL1A1* polymorphism has been estimated at approximately 97%, while its sensitivity is less than 1% [14]. Genetic tests are most reliable when based on polymorphisms with high sensitivity and specificity >90%, which minimize false negatives and false positives. Polymorphisms with lower accuracy of <80% are unreliable and should not guide risk assessment or preventive measures [38].

Although there can be some similarities, the pathogenesis of different musculoskeletal injuries is complex and not identical [39]. Therefore, any test claiming that a limited number of genetic markers can determine “soft tissue injury risk” or “injury predisposition” lacks scientific merit. It is essential to evaluate both what the test claims to measure and which markers are being used to determine susceptibility.

Epigenetic Modifications and Injury

This perspective has primarily focused on the important role of inherited polymorphisms in protein-coding and non-coding genes. Although not its main focus, this perspective would be incomplete without consideration of the effects of inherited and transient epigenetic changes in response to sport-related injuries [40]. Epigenetics was defined by the Centers for Disease Control and

Prevention (CDC) as follows: "Epigenetics is the study of how your behaviours and environment can cause changes that affect the way your genes work. Unlike genetic changes, epigenetic changes are reversible and do not change your DNA sequence, but they can change how your body reads a DNA sequence" (www.cdc.gov/genomics/disease/epigenetics.htm, access on 3 May 2023). During injury and repair, gene expression in skeletal muscle, tendon, bone, brain, and other tissues is regulated by three main epigenetic mechanisms: (i) DNA methylation, (ii) histone modification and (iii) miRNA expression [40]. As illustrated in Figure 1, epigenetic changes add an additional layer of complexity to susceptibility to sports injuries, further calling into question the validity of using a small number of inherited genetic polymorphisms as clinically relevant markers of injury susceptibility [41].

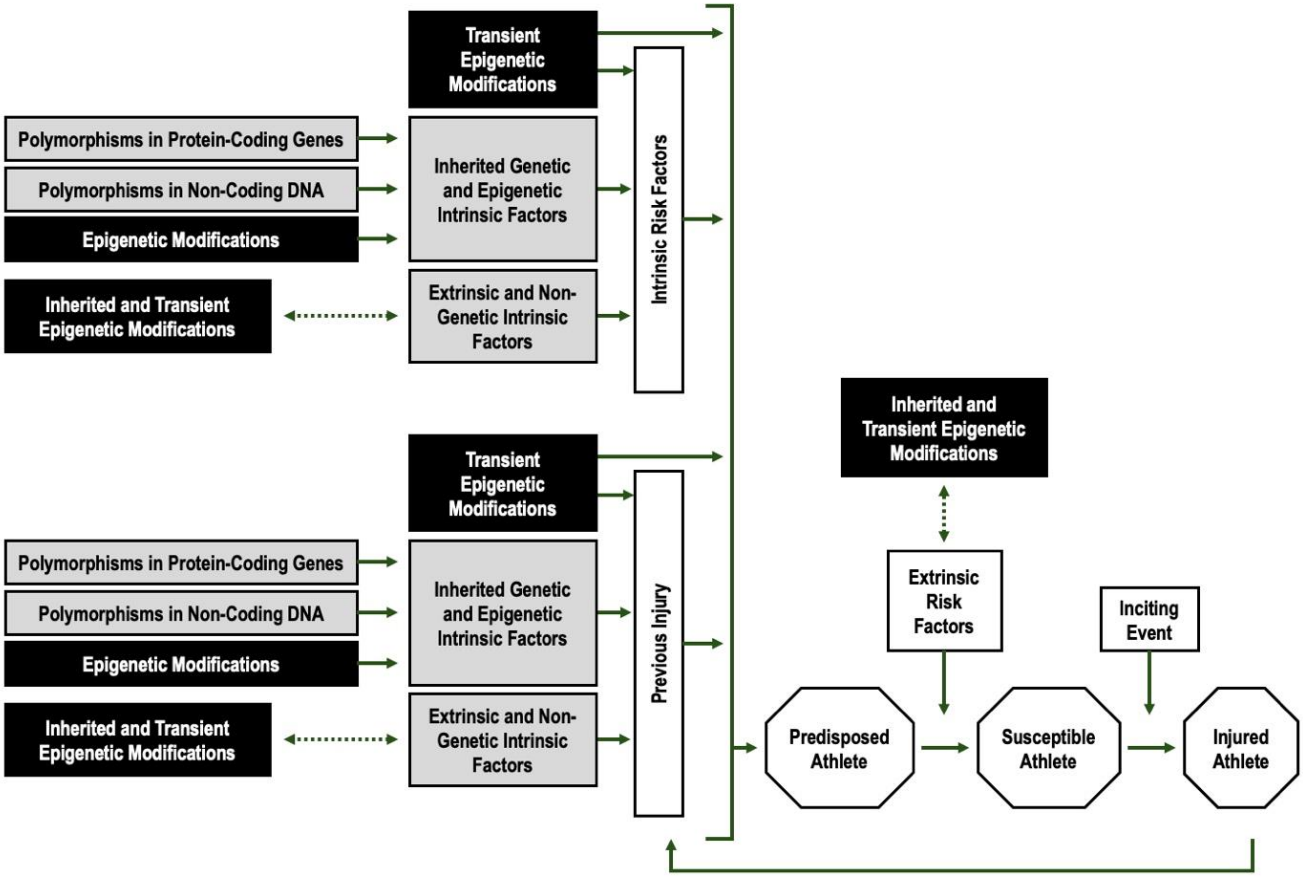

Figure 1: The schematic diagram illustrates the complex relationship between intrinsic and extrinsic risk factors, as well as the role of the inciting event in the etiology of multifactorial sporting injuries (white boxes represents the classical model) [42]. For acute injuries, the inciting event is the macro-traumatic event that directly causes the injury, whereas for chronic (overuse) injuries, it represents the point at which accumulated micro-traumatic damage to the tissue becomes symptomatic. Each injury has a unique set of intrinsic and extrinsic risk factors. Only a previous injury - which is common to many injuries - is specifically shown as an intrinsic risk factor. Many individual intrinsic risk factors are themselves also multifactorial phenotypes, determined by their own unique sets of extrinsic and intrinsic factors [43]. These are represented by as two grey boxes: (i) extrinsic and non-genentic intrinsic factors and (ii) inherited genentic and epigenetic intrinsic factors. The inherited genetic and epigenentic factors consist of (i) polymorphisms in protein coding genes (grey boxes), (ii) polymorphisms in non-coding genes/DNA (grey boxes) and (iii) inherited epigenentic modifications (black boxes with solid arrows). In the revised model, transient non-inherited epigenetic modifications, such as histone modifcations, (black boxes with solid arrows) are proposed to directly influence susceptibility to injury and the individual intrinsic risk factors. The model also highlightes the possibility complex relationship of extrinsic and non-genentic intrinsic factors with inherited and transcient epigenentic modifications (black box with dashed arrows) [41].

Inherited Disorders and Exercise

While this perspective has focused on common multifactorial injuries in which genetic polymorphisms modulate susceptibility but do not directly cause injury, it is important to acknowledge the existence of rare inherited musculoskeletal, cardiac, and other conditions. In these cases, participation in exercise and sport requires careful clinical management, and genetic diagnostic testing may be appropriately recommended by a clinician [44,45].

Personalized Medicine Already a Reality

Even though I personally believe that there is currently no scientific evidence to support the use of specific genetic markers to determine susceptibility to exercise- and sport-related injuries, the following example illustrates why I believe personalized medicine is possible without a genetic test.

A professional athlete has a history of multiple tendon ruptures. During his most recent rupture, he was not taking

any quinolone antibiotics and has no previous history of using this type of medication. The question asked was: "Do you think he carries a genetic risk factor and can you perhaps guide us on how to reduce the risk in the future?"

A typical answer might be:

1. It is reasonable to conclude that the athlete is predisposed to tendon ruptures once the possible involvement of all non-genetic risk factors has been considered and excluded.
2. Currently, it is not possible to identify the exact predisposing factor, as this is a multifactorial injury, likely resulting from a combination of inherited and non-inherited factors.
3. Prescribe supervised prehabilitation exercises to reduce the risk of tendon ruptures and other common acute injuries associated with the sport.

Conclusion

As highlighted throughout this perspective and a recent review, there is currently no robust scientific evidence to support the use of commercial genetic tests for predicting susceptibility to common multifactorial exercise- and sports-related injuries [14]. The limitations are clear: most studies focus on a small number of candidate genes, findings are often not reproducible across independent cohorts, risk alleles are frequently rare or population-specific, and the clinical sensitivity and specificity of reported markers are generally inadequate. Large, well-powered, and rigorously phenotyped studies with replication across diverse populations are urgently needed before any genetic test can be considered clinically valid. Importantly, personalized medicine in sports injury prevention does not depend on genetic testing. Clinicians can already apply individualized strategies to reduce injury risk by assessing and managing established modifiable factors such as training load and biomechanics. In this way, precision care can be achieved today, while future research may eventually clarify the role of genetics and epigenetics in injury susceptibility.

References

1. Wang C, Stovitz SD, Kaufman JS, Steele RJ, Shrier I. Principles of musculoskeletal sport injuries for epidemiologists: a review. *Inj Epidemiol*. May 27 2024;11(1):21. doi:10.1186/s40621-024-00507-3
2. Jarvinen TA, Kannus P, Maffulli N, Khan KM. Achilles tendon disorders: etiology and epidemiology. *Foot Ankle Clin*. Jun 2005;10(2):255-66. doi:10.1016/j.fcl.2005.01.013
3. Maffulli N, Del Buono A, Oliva F, et al. Muscle Injuries: A Brief Guide to Classification and Management. *Transl Med UniSa*. May-Aug 2015;12(4):14-8.
4. Symoens S, Syx D, Malfait F, et al. Comprehensive molecular analysis demonstrates type V collagen mutations in over 90% of patients with classic EDS and allows to refine diagnostic criteria. *Hum Mutat*. Oct 2012;33(10):1485-93. doi:10.1002/humu.22137
5. Judge DP, Dietz HC. Marfan's syndrome. *Lancet*. Dec 3 2005;366(9501):1965-76. doi:10.1016/S0140-6736(05)67789-6
6. Gajko-Galicka A. Mutations in type I collagen genes resulting in osteogenesis imperfecta in humans. *Acta Biochim Pol*. 2002;49(2):433-41.
7. Bahr R, Krosshaug T. Understanding injury mechanisms: a key component of preventing injuries in sport. *Br J Sports Med*. Jun 2005;39(6):324-9. doi:10.1136/bjsm.2005.018341
8. Herbert AJ, Williams AG, Hennis PJ, et al. The interactions of physical activity, exercise and genetics and their associations with bone mineral density: implications for injury risk in elite athletes. *Eur J Appl Physiol*. Jan 2019;119(1):29-47. doi:10.1007/s00421-018-4007-8
9. Leonska-Duniec A. Genetic Susceptibility to Sport-Related Muscle Injuries: Insights from the Literature and Novel Gene Candidates. *Int J Mol Sci*. Nov 19 2025;26(22)doi:10.3390/ijms262211175
10. Ribbans WJ, September AV, Collins M. Tendon and Ligament Genetics: How Do They Contribute to Disease and Injury? A Narrative Review. *Life (Basel)*. Apr 29 2022;12(5):663. doi:10.3390/life12050663
11. Marrouh A, El Haddouchi A, Kartti S, et al. The role of genetic and epigenetic factors in sports-related muscle, bone, and brain injuries. *Sci Prog*. Oct-Dec 2025;108(4):368504251385937. doi:10.1177/00368504251385937
12. Magnusson K, Turkiewicz A, Hughes V, Frobell R, Englund M. High genetic contribution to anterior cruciate ligament rupture: Heritability ~69. *Br J Sports Med*. Dec 7 2020;doi:10.1136/bjsports-2020-102392
13. Hakim AJ, Cherkas LF, Spector TD, MacGregor AJ. Genetic associations between frozen shoulder and tennis elbow: a female twin study. *Rheumatology (Oxford)*. Jun 2003;42(6):739-42. doi:10.1093/rheumatology/keg159
14. Collins M, September AV. Are commercial genetic injury tests premature? *Scand J Med Sci Sports*. Sep 2023;33(9):1584-1597. doi:10.1111/sms.14406
15. Williams AG, Wackerhage H, Day SH. Genetic Testing for Sports Performance, Responses to Training and Injury Risk: Practical and Ethical Considerations. *Med Sport Sci*. 2016;61:105-19. doi:10.1159/000445244
16. Webborn N, Williams A, McNamee M, et al. Direct-to-consumer genetic testing for predicting sports performance and talent identification: Consensus statement. *Br J Sports Med*. Dec 2015;49(23):1486-91. doi:10.1136/bjsports-2015-095343
17. Vlahovich N, Fricker PA, Brown MA, Hughes D. Ethics of genetic testing and research in sport: a position statement from the Australian Institute of Sport. *Br J Sports Med*. Jan 2017;51(1):5-11. doi:10.1136/bjsports-2016-096661
18. Hadley M, McNamara D. Genetic testing in sport: Considerations for young athletes. *Alternative Law Journal*. 2020;45(4):303-308. doi:10.1177/1037969x20956260
19. Goodlin GT, Roos AK, Roos TR, et al. Applying personal genetic data to injury risk assessment in athletes. *PLoS One*. 2014;10(4):e0122676. doi:10.1371/journal.pone.0122676
20. Mokone GG, Schwellnus MP, Noakes TD, Collins M. The COL5A1 gene and Achilles tendon pathology. *Scand J Med Sci Sports*. Feb 2006;16(1):19-26. doi:10.1111/j.1600-0838.2005.00439.x
21. Posthumus M, September AV, Keegan M, et al. Genetic risk factors for anterior cruciate ligament ruptures: COL1A1 gene variant. *Br J Sports Med*. May 2009;43(5):352-6. doi:10.1136/bjsm.2008.056150
22. Posthumus M, September AV, O'Cuinneagain D, van der Merwe W, Schwellnus MP, Collins M. The COL5A1 gene is associated with increased risk of anterior cruciate ligament ruptures in female participants. *Am J Sports Med*. Nov 2009;37(11):2234-40. doi:10.1177/0363546509338266
23. Khoschnau S, Melhus H, Jacobson A, et al. Type I collagen alpha1 Sp1 polymorphism and the risk of cruciate ligament ruptures or shoulder dislocations. *Am J Sports Med*. Dec 2008;36(12):2432-6. doi:10.1177/0363546508320805
24. Collins M, Posthumus M. Type V collagen genotype and exercise-related phenotype relationships: a novel hypothesis. *Exerc Sport Sci Rev*. Oct 2011;39(4):191-8. doi:10.1097/JES.0b013e318224e853
25. Laguette MJ, Abrahams Y, Prince S, Collins M. Sequence variants within the 3'-UTR of the COL5A1 gene alters mRNA stability: implications for musculoskeletal soft tissue injuries. *Matrix Biol*. Jun 2011;30(5-6):338-45. doi:10.1016/j.matbio.2011.05.001
26. Skorupski P, Krol J, Starega J, Adamiak A, Jankiewicz K, Rechberger T. An alpha-1 chain of type I collagen Sp1-binding site polymorphism in women suffering from stress urinary incontinence. *Am J Obstet Gynecol*. Feb 2006;194(2):346-50. doi:10.1016/j.ajog.2005.07.034
27. Wang B, Chen Q, Zou X, Zheng P, Zhu J. Advances in non-coding RNA in tendon injuries. *Frontiers in Genetics*. 2024;15doi:10.3389/fgene.2024.1396195
28. Antrobus MR, Brazier J, Callus PC, et al. Concussion-Associated Polygenic Profiles of Elite Male Rugby Athletes. *Genes (Basel)*. May 4 2022;13(5):820. doi:10.3390/genes13050820

29. Guth T, Ketcham CJ, Hall EE. Influence of Concussion History and Genetics on Event-Related Potentials in Athletes: Potential Use in Concussion Management. *Sports (Basel)*. Jan 19 2018;6(1) doi:10.3390/sports6010005
30. Kim SK, Roche MD, Fredericson M, et al. A Genome-wide Association Study for Concussion Risk. *Med Sci Sports Exerc*. Apr 1 2021;53(4):704-711. doi:10.1249/MSS.0000000000002529
31. Kerr H, Bakken B, House G. Future Directions in Sports-Related Concussion Management. *Clin Sports Med*. Jan 2021;40(1):199-211. doi:10.1016/j.csm.2020.08.009
32. McGrew CA. Sports-related Concussion - Genetic Factors. *Curr Sports Med Rep*. Jan 2019;18(1):20-22. doi:10.1249/JSR.0000000000000555
33. Tabor JB, Brett BL, Nelson L, et al. Role of biomarkers and emerging technologies in defining and assessing neurobiological recovery after sport-related concussion: a systematic review. *Br J Sports Med*. Jun 2023;57(12):789-797. doi:10.1136/bjsports-2022-106680
34. Kim SK, Nguyen C, Avins AL, Abrams GD. Three genes associated with anterior and posterior cruciate ligament injury: A genome-wide association analysis. *Bone Jt Open*. Jun 2021;2(6):414-421. doi:10.1302/2633-1462.26.BJO-2021-0040.R1
35. Phipson C. *Investigating the Association of Genetic Variants, Previously Identified by a Genome-Wide Association Study, with ACL Rupture in Multiple Cohorts*. University of Cape Town; 2026.
36. Kim SK, Roos TR, Roos AK, et al. Genome-wide association screens for Achilles tendon and ACL tears and tendinopathy. *PLoS One*. 2017;12(3):e0170422. doi:10.1371/journal.pone.0170422
37. Gibbon A, Raleigh SM, Ribbans WJ, Posthumus M, Collins M, September AV. Functional COL1A1 variants are associated with the risk of acute musculoskeletal soft tissue injuries. *J Orthop Res*. Oct 2020;38(10):2290-2298. doi:10.1002/jor.24621
38. Tabangin ME, Woo JG, Martin LJ. The effect of minor allele frequency on the likelihood of obtaining false positives. *BMC Proc*. Dec 15 2009;3 Suppl 7(Suppl 7):S41. doi:10.1186/1753-6561-3-S7-S41
39. Bahr R, Alfredson H, Järvinen M, et al. Types and Causes of Injuries. *The IOC Manual of Sports Injuries*. 2012:1-24.
40. Tarnowski M, Tomasiak P, Tkacz M, Zgutka K, Piotrowska K. Epigenetic Alterations in Sports-Related Injuries. *Genes (Basel)*. Aug 17 2022;13(8):1471. doi:10.3390/genes13081471
41. Ehlert T, Simon P, Moser DA. Epigenetics in sports. *Sports Med*. Feb 2013;43(2):93-110. doi:10.1007/s40279-012-0012-y
42. Meeuwisse WH. Assessing causation in sport injury: A multifactorial model. *Clin J Sport Med*. 1994;4:166-170.
43. Tucker R, Collins M. Athletic performance and risk of injury: can genes explain all? *Dialogues in Cardiovascular Medicine*. 2012;17(1):3-11.
44. Rye EE, Mitchell AM, Santiago CF, La Gerche A, Fatkin D. Exercise in Inherited Cardiomyopathies: Optimizing the Dose-Response Curve. *Circ Res*. Jul 7 2025;137(2):316-334. doi:10.1161/CIRCRESAHA.125.326396
45. Jouini S, Milleron O, Eliahou L, Jondeau G, Vitiello D. Is physical activity a future therapy for patients with Marfan syndrome? *Orphanet J Rare Dis*. Feb 10 2022;17(1):46. doi:10.1186/s13023-022-02198-9