Proceedings of the 6th International Conference on Biological Engineering and Medical Science
DOI: 10.54254/2753-8818/2026.38673

Genetic and Environmental Factors Underlying Tennis Performance and Injury Risk: A Structured Review

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Abstract. This structured review integrates evidence regarding genetic and exercise-related factors associated with tennis performance and injury risk, focusing on genes involved in explosive power, endurance, connective-tissue integrity, and recovery. The literature was systematically searched across PubMed and Google Scholar, with PubChem and the AlphaFold Protein Structure Database used as supplementary resources for chemical and structural information. Ten candidate genes were examined across four functional categories: ACTN3 and ACE (explosive power), PPARGC1A, PPARA, and NOS3 (endurance), COL5A1, COL1A1, and MMP3 (connective-tissue health), and IL6 and IGF1 (recovery and inflammation). Environmental factors, including training, nutrition, sleep, and heat exposure, were also considered for their potential influence on relevant physiological pathways. Although several genetic variants have been associated with athletic traits in general athletic populations, direct evidence in tennis-specific cohorts remains limited. Overall, the findings are consistent with a polygenic, environment-modulated model, but the reviewed evidence does not establish any single candidate gene as a reliable predictor of tennis performance or injury risk.

Keywords: sports genetics, tennis, gene-environment interactions, injury risk, athletic performance

1. Introduction

Tennis is a physically demanding sport that requires a combination of explosive power, aerobic and anaerobic endurance, agility, and fine motor control, often during matches lasting two to three hours or longer. Repetitive high-intensity movements, including rapid acceleration, deceleration, and overhead serving, place substantial stress on muscular and connective tissues. As a result, tennis players are susceptible to overuse injuries such as lateral epicondylitis (“tennis elbow”), rotator cuff pathology, and Achilles tendinopathy. Lower-extremity injuries account for approximately 31–67% of tennis-related injuries, followed by upper-extremity injuries at 20–49% and trunk injuries at 3–21% [1]. Injury patterns also differ by age. In young tennis players, lower-extremity injuries, particularly ankle sprains, occur more frequently, while chronic overuse conditions such as tennis elbow are less common [2].

In the past, research and coaching have mainly focused on modifiable factors such as training, nutrition, and recovery. However, sports genomics has shown that genetic variation may also

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DOI: 10.54254/2753-8818/2026.38673

contribute to athletic traits such as power, endurance, and tissue recovery. Yang et al. (2003) found that the ACTN3 R577X polymorphism was associated with elite sprint and power performance, with the 577R allele more common among power athletes and the 577X allele more common among endurance athletes [3]. Pickering and Kiely (2017) further suggested that ACTN3 may also influence exercise adaptation, muscle recovery, and injury risk [4]. These findings suggest that athletic performance and injury are influenced by both genetic and environmental factors.

This review focuses on four categories of genes related to tennis: power (ACTN3 and ACE), endurance (PPARGC1A, PPARA, and NOS3), connective-tissue health (COL5A1, COL1A1, and MMP3), and recovery and inflammation (IL6 and IGF1). The proteins encoded by these genes are shown in Figures 1–4, respectively. However, most studies have examined general athletic populations rather than tennis players, and many have focused on adult athletes. This creates a gap in understanding how these genetic factors may apply to young tennis players, whose physical development, training load, and injury patterns differ from those of adults [2].

This structured review asks: What evidence links genetic variants and exercise-responsive genes involved in power, endurance, connective-tissue health, and recovery to physical traits relevant to tennis players? We hypothesize that tennis performance and injury risk are polygenic and influenced by gene-environment interactions, rather than predicted by a single gene.

2. Literature review

This structured review used a focused literature search to identify studies relevant to genetic factors in athletic performance, injury risk, and recovery. PubMed and Google Scholar were searched in August 2026 using terms including “tennis genes”, “physical performance genetics”, “genotype athletes”, and “sports genes”. PubMed records were assessed for relevance to athletes or athletic performance, then checked for whether they investigated specific genes or genetic variants related to performance, injury risk, or recovery. We screened first twenty Google Scholar search results per query. Studies were included if they met these criteria: if they investigated specific genes or genetic variants associated with athletic performance, injury risk, or recovery. PubChem verified chemical and nutritional information related to environmental factors, while AlphaFold predicted protein structures of selected candidate genes and provide insight into their potential biological functions [5, 6].

3. Genes related to tennis performance

3.1. Power related genes

Tennis strokes such as the serve and forehand require rapid, explosive contractions that rely heavily on fast-twitch (type II) muscle fibers. As shown in Figure 1, ACTN3 encodes α-actinin-3, a structural protein expressed in fast-twitch fibers. Its R577X (rs1815739) variant determines whether functional α-actinin-3 is produced, with the XX genotype lacking the protein. Yang et al. found the R allele to be overrepresented among elite sprint and power athletes, while the XX genotype was more common among endurance athletes [3]. However, Moreno-Pérez et al. found no significant association between ACTN3 R577X and elite performance in professional and amateur tennis players [7]. Pickering and Kiely also suggested that ACTN3 may influence recovery and injury, indicating that its role extends beyond explosive power [4].

ACE encodes angiotensin-converting enzyme and regulates cardiovascular and muscular responses to exercise. The protein structure is also shown in Figure 1. Its I/D polymorphism has

DOI: 10.54254/2753-8818/2026.38673

been associated with endurance and power phenotypes, with the D allele linked to power and strength in some athletic populations. Eider et al. found a higher D-allele frequency among elite Polish power athletes, increasing from sub-elite to elite and top-elite levels [8], but this association has not been established in tennis players.

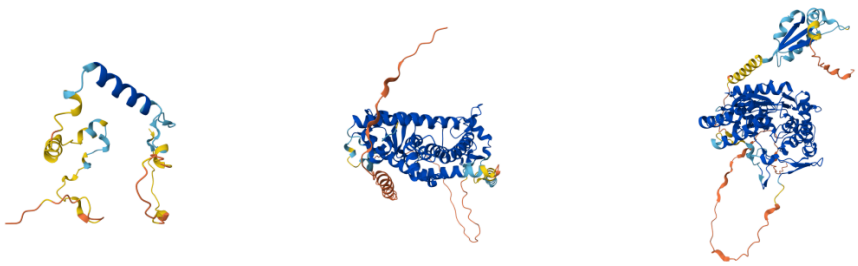


(a) α-actinin-3 (b) Angiotensin-converting enzyme

Figure 1. AlphaFold-predicted structures of power-related proteins: (a) α-actinin-3 (ACTN3; UniProt Q08043) and (b) angiotensin-converting enzyme (ACE; UniProt P12821-2). Blue generally indicates higher model confidence; yellow/orange/red indicates lower confidence

3.2. Endurance-related genes

PPARGC1A encodes PGC-1α, a key regulator of mitochondrial biogenesis and energy metabolism. The Gly482Ser (rs8192678) variant has been associated with endurance performance, with the Gly allele overrepresented among Polish and Russian endurance athletes [9]. PPARA regulates fatty-acid oxidation, and a meta-analysis of 760 endurance athletes and 1,792 controls associated the G allele of rs4253778 with endurance status (OR = 1.65) [10]. NOS3 encodes endothelial nitric oxide synthase, which supports vasodilation and oxygen delivery; the Genathlete study associated an intron 13 variant with elite endurance status [11]. As shown in Figure 2, PGC-1α has a relatively flexible structure with both ordered and less-structured regions, PPARα displays a compact, predominantly helical fold, and NOS3 has a larger structure with multiple folded domains [5, 6]. These differences reflect their distinct protein architectures. No tennis-specific studies of these genes were identified in this review.



(a) PGC-1α (b) PPARα (c) endothelial nitric oxide synthase

Figure 2. AlphaFold-predicted structures of endurance-related proteins: a)PGC-1α (PPARGC1A; UniProt Q9UBK2), b)PPARα (PPARA; UniProt Q07869), and c)endothelial nitric oxide synthase (NOS3; UniProt P29474). Blue generally indicates higher model confidence; yellow/orange/red indicates lower confidence

3.3. Tendon Health and Flexibility related genes

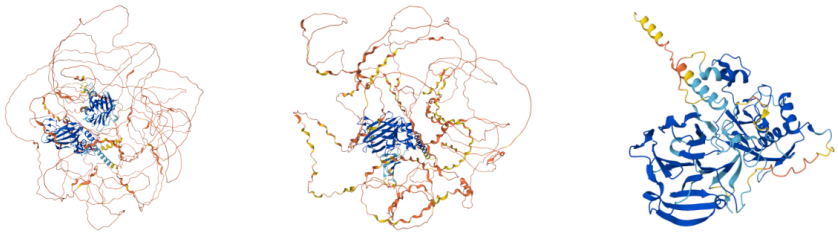
In addition to explosive power and endurance, tennis subjects' tendons and ligaments to substantial repetitive mechanical stress, particularly during overhead serves and rapid lateral movements.

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Therefore, connective-tissue integrity represents a third key dimension in tennis-related genetic research, with a more direct association with injury risk than athletic performance itself.

COL5A1 encodes a type V collagen chain involved in regulating type I collagen fiber assembly; the A2 allele was associated with lower risk of chronic Achilles tendon disease [12]. COL1A1 encodes a major structural collagen in tendons and ligaments; a meta-analysis found a protective association for the TT genotype and included studies of tennis elbow, providing injury-related evidence potentially relevant to tennis [13]. The MMP3 rs679620 GG genotype was associated with Achilles tendinopathy (OR = 2.5), and an interaction with COL5A1 was reported [14].

The predicted structures in Figure 3 represent two collagen chains with lower confidence and one blue matrix-remodeling enzyme. Their functions are complementary, but they should not be interpreted as having the same overall protein fold.



(a) Collagen alpha-1(V) chain (b) Collagen alpha-1(I) chain (c) Stromelysin-1

Figure 3. AlphaFold-predicted structures of connective-tissue-related proteins: a) collagen α-1(V) chain (COL5A1; UniProt P20908), b) collagen α-1(I) chain (COL1A1; UniProt P02452), and c) matrix metalloproteinase-3 (MMP3; UniProt P08254). Blue generally indicates higher model confidence; yellow/orange/red indicates lower confidence

3.4. Recovery genes

Recovery between training sessions and matches represents a fourth genetically influenced dimension relevant to tennis performance, particularly for younger athletes balancing training loads with ongoing physical development.

The IL6 gene encodes interleukin-6, a cytokine involved in the acute inflammatory response to exercise-induced muscle injury. Although this evidence is not from tennis-specific cohorts, IL6 signaling pathway is relevant to recovery after repeated high-intensity exercise.



(a) Interleukin-6 (b) Insulin-like growth factor 1

Figure 4. AlphaFold-predicted structures of recovery-related proteins: a) interleukin-6 (IL6; UniProt P05231) and b) insulin-like growth factor 1 (IGF1; UniProt P05019). Blue generally indicates higher model confidence; yellow/orange/red indicates lower confidence.

IGF1 encodes insulin-like growth factor 1, a key regulator of muscle repair and anabolic signaling following exercise-induced damage. A promoter CA-repeat polymorphism region has been studied in relation to motor skill development in a study of 350 young athletes found an association

DOI: 10.54254/2753-8818/2026.38673

between this polymorphism and strength-oriented motor skill profiles [15]. An earlier study of 272 Polish youth athletes found that variants in the same promoter region were more frequent in athletes than non-athletes (9.2% vs. 2.4%), with a higher proportion among strength-sport athletes than team-sport athletes (11.0% vs. 7.8%) [16].

4. Results

Table 1 summarizes the candidate genes, selected variants, and corresponding proteins across four functional categories.

Table 1. Summary of candidate genes examined in this review, grouped by functional category

Category	Gene	Key variant	Encoded proteins
Power	ACTN3	R577X (rs1815739)	α-actinin-3
Power	ACE	I/D polymorphism	Angiotensin-converting enzyme
Endurance	PPARGC1A	Gly482Ser (rs8192678)	PGC-1α
Endurance	PPARA	Intron 7 G/C (rs4253778)	PPARα
Endurance	NOS3	Intron4 VNTR (4a/4b); intron 13 variant	Endothelial nitric oxide synthase
Tendon Health	COL5A1	BstUI RFLP (3' UTR)	Type V collagen α-1 chain
Tendon Health	COL1A1	Sp1 site (rs1800012)	Type I collagen α-1 chain
Tendon Health	MMP3	rs679620	Matrix metalloproteinase 3
Recovery	IL6	-174 G/C (IL6)	Interleukin-6
Recovery	IGF1	Promoter CA repeat	Insulin-like growth factor 1

Environmental and lifestyle factors, including training, nutrition, sleep, and heat exposure, may modify pathways related to the genes discussed above. Endurance training favors the PGC-1α1 isoform, which supports aerobic adaptation, while resistance training favors PGC-1α4 and increases IGF1 expression, promoting muscle growth [17]. Nutrition may also affect exercise-related inflammation and recovery; omega-3 supplementation has been shown to reduce biomarkers of exercise-induced muscle damage [18]. Sleep is another important factor in recovery. In a rodent model, REM sleep deprivation reduced IGF1 expression and delayed muscle regeneration, although sleep compensation restored IGF1 levels without fully restoring regeneration [19]. Heat exposure provides another example of environmental regulation. Following Finnish sauna exposure, non-athletes showed higher expression of heat-shock proteins and IL6, IL10, and C-reactive protein than athletes, suggesting that training status can modify the response to heat exposure [20]. These findings are not tennis-specific and do not yet support genotype-based recommendations.

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5. Discussion

A key finding is that ACTN3 is one of the most extensively studied genes related to explosive power in the general athletic population and showed no significant association with elite tennis performance when tested directly in a tennis-specific cohort [3, 7]. This result supports the central hypothesis of this review that tennis performance and injury risk are polygenic traits influenced by multiple pathways rather than a single gene. A genetic variant associated with performance in sprint or power athletes may have less predictive value in tennis, which also requires a combination of explosive power, endurance, agility, technical skill, and tactical ability.

The connective-tissue category provides the evidence of associations with tendon and ligament injuries, particularly because COL1A1 includes data on tennis elbow and the MMP3-COL5A1 interaction supports a polygenic model of tendon injury risk [13, 14]. The recovery category also provides evidence from young athletes, with IGF1 associated with strength-oriented motor skills in two studies [15, 16]. The endurance-related genes PPARGC1A, PPARA, and NOS3 have shown associations with endurance traits in other athletic populations [9-11], but no tennis-specific studies of these genes were identified in this review. This finding is consistent with a multifactorial interpretation of tennis performance, although a null association for one variant does not establish a polygenic mechanism. External factors like training, nutrition, sleep, and heat exposure may influence pathways involving PGC-1α, IGF1, and IL6, indicating that genetic susceptibility does not act independently of an athlete's environment. Research in youth tennis cohorts should assess variants alongside training load, diet, sleep, recovery and other factors before genotype-specific advice is considered.

Several limitations affect interpretation of the evidence. Most genetic evidence came from non-tennis populations. Among the genetic association studies discussed, reference [7] directly examined tennis players, whereas reference [14] included tennis-elbow outcomes without establishing a tennis-player cohort. Many genetic studies also focused on adults, whereas adolescent players differ in growth, training load, and injury patterns. In addition, some environmental evidence, particularly the sleep-related findings, came from animal models rather than human athletes, limiting direct translational confidence.

6. Conclusion

This review supports a polygenic and environment-modulated model of tennis performance and injury risk. No single candidate gene reliably predicts tennis performance or injury susceptibility. COL1A1 and the MMP3-COL5A1 interaction provide the evidence of associations with tendon and ligament injuries, while power and endurance genes remain largely untested in tennis-specific populations. Training, nutrition, sleep, and recovery may modify genetic effects. Future research should prioritize tennis-specific youth cohorts and directly examine gene-environment interactions.

Acknowledgement

The author gratefully acknowledges Li Zhu for mentorship and guidance throughout this research project.

References

[1] Abrams, G. D., Renstrom, P. A., & Safran, M. R. (2012). Epidemiology of musculoskeletal injury in the tennis player. *British Journal of Sports Medicine, 46*(7), 492–498.

DOI: 10.54254/2753-8818/2026.38673

[2] Bylak, J., & Hutchinson, M. R. (1998). Common sports injuries in young tennis players. *Sports Medicine, 26*(2), 119–132.
[3] Yang, N., MacArthur, D. G., Gulbin, J. P., Hahn, A. G., Beggs, A. H., Easteal, S., & North, K. (2003). ACTN3 genotype is associated with human elite athletic performance. *American Journal of Human Genetics, 73*(3), 627–631.
[4] Pickering, C., & Kiely, J. (2017). ACTN3: More than just a gene for speed. *Frontiers in Physiology, 8*, 1080.
[5] Jumper, J., Evans, R., Pritzel, A., et al. (2021). Highly accurate protein structure prediction with AlphaFold. *Nature, 596*, 583–589.
[6] Varadi, M., Anyango, S., Deshpande, M., et al. (2022). AlphaFold protein structure database: Massively expanding the structural coverage of protein-sequence space with high-accuracy models. *Nucleic Acids Research, 50*(D1), D439–D444.
[7] Moreno-Pérez, V., Machar, R., Sanz-Rivas, D., & Del Coso, J. (2020). ACTN3 R577X genotype in professional and amateur tennis players. *Journal of Strength and Conditioning Research, 34*(4), 952–956.
[8] Eider, J., Cieszczyk, P., Ficek, K., Leonska-Duniec, A., Sawczuk, M., Maciejewska-Karlowska, A., & Zarebska, A. (2013). The association between D allele of the ACE gene and power performance in Polish elite athletes. *Science & Sports, 28*(6), 325–330.
[9] Maciejewska, A., Sawczuk, M., Cieszczyk, P., Mozhayskaya, I. A., & Ahmetov, I. I. (2012). The PPARGC1A gene Gly482Ser in Polish and Russian athletes. *Journal of Sports Sciences, 30*(1), 101–113.
[10] Lopez-Leon, S., Tuvblad, C., & Forero, D. A. (2016). Sports genetics: The PPARA gene and athletes' high ability in endurance sports. A systematic review and meta-analysis. *Biology of Sport, 33*(1), 3–6.
[11] Wolfarth, B., Rankinen, T., Muhlbauer, S., Ducke, M., Rauramaa, R., Boulay, M. R., Perusse, L., & Bouchard, C. (2008). Endothelial nitric oxide synthase gene polymorphism and elite endurance athlete status: The Genathlete study. *Scandinavian Journal of Medicine & Science in Sports, 18*(4), 485–490.
[12] Mokone, G. G., Schwellnus, M. P., Noakes, T. D., & Collins, M. (2006). The COL5A1 gene and Achilles tendon pathology. *Scandinavian Journal of Medicine & Science in Sports, 16*(1), 19–26.
[13] Wang, C., Li, H., Chen, K., Wu, B., & Liu, H. (2017). Association of polymorphisms rs1800012 in COL1A1 with sports-related tendon and ligament injuries: A meta-analysis. *Oncotarget, 8*(16), 27627–27634.
[14] Raleigh, S. M., Van Der Merwe, L., Ribbans, W. J., Smith, R. K. W., Schwellnus, M. P., & Collins, M. (2009). Variants within the MMP3 gene are associated with Achilles tendinopathy: Possible interaction with the COL5A1 gene. *British Journal of Sports Medicine, 43*(7), 514–520.
[15] Karpowicz, K., Krych, K., Karpowicz, M., Nowak, W., & Gronek, P. (2018). The relationship between CA repeat polymorphism of the IGF-1 gene and the structure of motor skills in young athletes. *Acta Biochimica Polonica, 65*(1), 43–50.
[16] Krych-Garsztka, K., Mizgajska-Wiktor, H., & Gozdzicka-Jozefiak, A. (2011). An analysis of the regulatory region of the IGF1 gene in professional athletes in youth sports teams. *Human Movement, 12*(3), 216–222.
[17] Ruas, J. L., White, J. P., Rao, R. R., et al. (2012). A PGC-1alpha isoform induced by resistance training regulates skeletal muscle hypertrophy. *Cell, 151*(6), 1319–1331.
[18] Fernandez-Lazaro, D., Arribalzaga, S., Gutierrez-Abejon, E., Azarbayjani, M. A., Mielgo-Ayuso, J., & Roche, E. (2024). Omega-3 fatty acid supplementation on post-exercise inflammation, muscle damage, oxidative response, and sports performance in physically healthy adults: A systematic review of randomized controlled trials. *Nutrients, 16*(13), 2044.
[19] Monico-Neto, M., Dattilo, M., Ribeiro, D. A., Lee, K. S., de Mello, M. T., Tufik, S., & Antunes, H. K. M. (2017). REM sleep deprivation impairs muscle regeneration in rats. *Growth Factors, 35*(1), 12–18.
[20] Zychowska, M., Polrola, P., Chruscinski, G., Zielinska, J., & Goral-Polrola, J. (2017). Effects of sauna bathing on stress-related genes expression in athletes and non-athletes. *Annals of Agricultural and Environmental Medicine, 24*(1), 104–107.