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Title page

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Genetic variants associated with on-water sports performance: A systematic review

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Genetic variants associated with on-water sports performance: A systematic review

Abstract

Background Genetic studies are conducted in athletic populations to understand the influence of genotype on phenotype, with the prospects of identifying talent, designing individualized training programmes, and predicting injuries in sports. Despite extensive research on improving water sports performance, the effects of various genetic polymorphisms on competitive performance remain unclear.

Objective This systematic review aims to identify key gene variants associated with on-water sports and assess their influence on performance.

Methods To fulfill this aim, a systematic search was conducted in Web of Science, Scopus, and PubMed until 6th October 2024, along with a secondary search of the included full-text articles. Studies examining the associations between genetic variants and on-water sports performance (rowing, canoeing, kayaking, sailing, surfing, and rafting) and meeting the Hardy–Weinberg equilibrium criteria were included, and the risk of bias was determined.

Results A total of 28 articles (6 high risk-of-bias) were included and reviewed (22 rowing, 4 kayaking, 5 canoeing, 1 each on surfing and rafting and none on sailing) that examined 17 different variants (ACE, ACTN3, AMPD1, CKMM, GABPB1, GDF8, HFE, HIF1A, MCT1, NOS3, PPARA, PPARGC1A, TFAM, UCP2, UCP3, VEGFR, and VEGFR2) from 8 different populations (Russians, Polish, Spanish, Brazilians, Hungarian, Australian, Turkish, and Chinese) associated with performance. The available evidence was inadequate for performing a meta-analysis and determining whether any of the genetic variants identified in this review were associated with sports performance.

Conclusion Although several structural and functional genes and their polymorphisms have been identified that are associated with on-water sports performance, further collaborative genome-wide association studies and gene expression and gene–gene interaction studies are needed in larger populations of different ethnicities to investigate additional possible genetic variants and the role of these gene polymorphisms in enabling success in sports.

Key words: Single-nucleotide polymorphism; rowing; canoeing; kayaking; ACTN3; ACE.

Background

In today's world, sports serve as one of the biggest platforms for individuals to participate and showcase their talents through physical prowess and skills. Sports performance is an intricate blend of strength, speed, endurance, and coordination, all of which are essential for executing technical and tactical skills effectively. Water sports encompass a wide range of recreational and competitive activities performed in, on, or around water. These include sports played in water, such as swimming and water polo; underwater sports, such as deep sea diving and underwater rugby; and on-water sports, such as rowing, kayaking, canoeing, sailing, surfing, and rafting. Our focus in this systematic review is on on-water sports.

On-water sports encompass a diverse range of activities, each demanding a unique combination of strength, endurance, skill, and balance. Rowing, one of the oldest and popular on-water sports, involves propelling a boat using oars. It is a strength-endurance sport that lasts approximately 6-8 minutes, depending on the level of performance [1]. Canoeing and kayaking rely on single-bladed and double-bladed paddles, respectively, to navigate water, necessitating significant cardiovascular and upper body strength and endurance for sustaining efforts and propelling the boat [2, 3]. Sailing harnesses wind power to drive boats, where both strength and endurance are crucial for managing sails and maintaining performance during long races. Other popular on-water sports, such as surfing and rafting, emphasize skill, balance, and speed, with surfing making its Olympics debut in 2020 [4]. Rafting, not yet an Olympic sport, is known for its adventurous nature and demands aerobic endurance and upper body strength [5].

Previous studies have reported that multiple factors influence success in sports, including genetics, technique, tactics, training, nutrition, psychology, and social support [6-8]. Among these factors, genetics plays a significant role, with approximately 66% of the variance in athletic status explained by genetic factors [9]. Heritable traits and genetic factors significantly impact sports performance, with an individual's genotype influencing the percentage of slow- and fast-twitch fibers [10, 11], heart and lung size [12], height, weight, maximal volume of oxygen consumption [13-16], mental toughness and other physiological characteristics [17]. The heritability of various muscular strength- and power-related phenotypes varies between 30% and 80%, whereas endurance-related phenotypes have a heritability of 44%-68% [14, 18]. The interplay of genetically determined physical and mental traits with the ideal environment results in

1 elite athletic status. Identifying genetic variants contributing to successful sports
2 performance has led to the discovery of 251 DNA polymorphisms associated with athletic
3 performance or related phenotypes [19]. Moreover, 200 genetic polymorphisms located
4 in the nuclear and mitochondrial genomes are positively associated with athlete status
5 [20, 21].). At least 155 genetic variants were found to be linked to elite athlete status, of
6 which 62 were power/strength-related genetic markers and 93 were endurance-related
7 genetic markers [22]. Case-control studies and cross-sectional association studies, which
8 have been the most common study designs in sports genomics, have focused primarily on
9 single-nucleotide polymorphisms (SNPs) [19, 23]. SNPs are the most common type of
10 genetic variation among people. An SNP occurs when a single nucleotide in the genome
11 is altered. Currently, the idea of using SNP and genetic test data to identify talent and
12 predict future sports performance is increasingly common and relevant throughout the
13 world. With the increasing popularity of competitive water sports, extensive research has
14 focused on improving water sports performance, including genetics, which plays a key
15 role in these efforts. Despite the high heritability of athletic status and related
16 phenotypes, specific genetic variants associated with performance have still not been
17 identified. Additionally, the genes and genetic variants associated with water sports
18 performance are still scarce and inconclusive. Systematic reviews and meta-analysis have
19 been conducted to find genetic variants associated with the performance of swimmers,
20 endurance athletes, elite combat sports athletes, and footballers [15, 24-27]. Despite
21 extensive research, no systematic review has comprehensively synthesized evidence
22 concerning genes associated with on-water sports performance. We addressed this gap
23 by systematically reviewing genes and its polymorphism associated with performance.

24 By identifying the genetic factors that contribute to elite performance in on-water sports,
25 this knowledge can be utilized to tailor training programs, optimize talent identification,
26 and enhance athlete selection processes. Furthermore, insights into the genetic
27 mechanisms influencing physical and physiological traits crucial for on-water sports can
28 lead to early screening and personalized interventions aimed at maximizing athletic
29 potential. Therefore, the objective of this systematic review was to identify key genes
30 associated with on-water sports performance and examine the influence of these gene
31 variants on performance. We hypothesize that numerous structural and functional genes
32 and their variants are associated with on-water sports performance.

Methods

Protocol

We followed the Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) checklist for all key aspects of this review (e.g., conducting searches, reporting, etc.) [28] but were unable to register the review in PROSPERO due to its current inclusion criteria, which exclude systematic reviews focused on sporting performance.

Search strategy

The systematic search of the articles used for this review was conducted in the Scopus, PubMed, and Web of Science online databases until 6th October 2024. In addition to these databases, a secondary search was performed by screening the references of the included full-text articles. The PECO (population, exposure, comparison, and outcome) tool was used to identify the research question and the framework used in the search strategy to search terms that were combined with “AND” or “OR”, Boolean operators. No additional filters were applied apart from the keywords while searching. The following search strategy was used: i) (“gene” OR “genes” OR “genetics” OR “gene variants” OR “genetic variants” OR “polymorphism” OR “genotype” OR “snp” OR “single nucleotide polymorphism” OR “allele” OR “alleles”) AND (“rowing” OR “rowers” OR “rower”); ii) (“gene” OR “genes” OR “polymorphism” OR “genotype” OR “snp” OR “allele” OR “alleles”) AND (“canoeing” OR “canoe*”); iii) (“gene” OR “genes” OR “polymorphism” OR “genotype” OR “snp” OR “allele” OR “alleles”) AND (“kayaking” OR “kayak*”); iv) (“gene” OR “genes” OR “polymorphism” OR “genotype” OR “snp” OR “allele” OR “alleles”) AND (“sailing” OR “sail” OR “sailor” OR “sailors”); v) (“gene” OR “genes” OR “polymorphism” OR “genotype” OR “snp” OR “allele” OR “alleles”) AND (“surfing” OR “surfer” OR “surfers” OR “surf”), and vi) (“gene” OR “genes” OR “polymorphism” OR “genotype” OR “snp” OR “allele” OR “alleles”) AND (“rafting” OR “rafter” OR “rafters” OR “raft”).

Eligibility criteria

Studies were considered for inclusion in our evaluation if they met the following criteria: (1) they were conducted on human subjects; (2) they were related to any of the following sports - rowing, canoeing, kayaking, sailing, surfing, or rafting; (3) they examined the associations between a genetic variant and water-sports performance; (4) their genotype data could be acquired in detail to validate the key findings; and (5) they were published

in English. The records were excluded if they met the following exclusion criteria: (1) studies conducted on animals; (2) they were review articles or editorials; (3) they did not examine associations between genotypes and on-water sports performance; (4) we were unable to obtain detailed genotype data to validate key findings; (5) studies which were in languages other than English; and (6) the full text was not available.

Study selection

EndNote reference management software was used to integrate the search results from three separate databases and delete duplicates. The Rayyan Intelligent Systematic Review web tool was used to manage and screen all the articles after duplicate removal [29]. Two authors blindly screened the results by article title and abstract. The selected full-text articles were also screened by the same independent authors. Studies that did not fulfill the eligibility criteria and were not relevant to our research were excluded, and the reasons for exclusion were recorded (Fig. 1). Any conflict or disagreement was resolved by discussion and mutual consensus.

----- Fig. 1 about here -----

Data extraction

Two authors independently gathered and summarized data from the included studies. The extracted data were crosschecked, and disagreements were resolved by discussion between two authors. The following data were collected from the selected studies on prespecified forms, if available: 1) basic information: title, name of the authors, and year published; 2) participant characteristics: country of origin, gender, and sports; 3) study characteristics: study design, number of cases and controls, gene name, and genetic variant; and 4) outcomes: association with sports performance.

Risk of bias assessment

We used the Newcastle–Ottawa quality assessment scale (NOS) for case–control and cohort studies to assess the risk of bias and quality of the included studies. This scale is a 9-star scale with 3 domains: study selection, comparability, and exposure. According to the guidelines, selection was graded with a maximum of 4 stars and included the selection and definition of controls and the definition and representativeness of the cases.

Comparability was awarded a maximum of 2 stars, and age, sex, and fulfilling the Hardy–Weinberg equilibrium (HWE) of the controls were considered in comparability. Exposure has a maximum of 3 stars and includes ascertainment and nonresponse rates. Details of the scale are available at http://www.ohri.ca/programs/clinical_epidemiology/oxford.asp. Two authors independently and blindly performed the quality assessment, and any disagreements were resolved by consensus or by a third author. Studies that received a score of 0–2 were considered poor quality, those with a score of 3–5 were considered fair quality, and those with a score of 6 or above were considered high quality.

Statistical analysis

We chose not to perform a statistical pooling of the data and do meta-analysis because of the variation in genetic variants studied, the failure of HWE, and the heterogeneity in the risk of bias across the studies (Additional file 2). Instead, we opted to present a narrative summary of the findings.

Results

Study selection

From the initial search results across three databases, a total of 2247 articles were found for this systematic review, supplemented by one additional record from another source. The details of the search results are shown in additional file 1. After duplicate records were removed (n=1407), 1341 articles remained. Following the title screening, 797 articles were excluded, and 544 articles remained for abstract screening, 41 of which were eligible for full-text viewing. Thirteen articles did not meet the inclusion criteria and were excluded for the following reasons: 3 articles were review type, could not extract genotype data from 4 studies, 2 studies were not in English, 2 studies focused on gene expression, 1 study focused on gene mutation, and the full text of 1 study was not available. Finally, 28 articles were eligible and included in this review. Fig. 1 depicts a flow diagram illustrating the article selection process at each stage.

Study characteristics

A summary of the 28 included articles is presented in Table 1. Among these, 19 were case–control studies, 6 were cross–sectional studies, 2 were cohort studies, and 1 was a

pilot study. These studies examined 29 different populations. Specifically, 11 studies focused on Russian populations, 10 on Polish populations, 2 each on Spanish and Brazilian populations, and 1 each on Hungarian, Belarusian, Australian, and Turkish populations (Fig. 2). The studies covered various sports, with 22 studies on rowing, 4 on kayaking, 5 on canoeing, 1 on surfing, and 1 on rafting, but no studies were found on sailing. Elite, non-elite, college, national, international and world class athletes were found in the studies across the included records. A total of 17 genes and their different variants were identified after a systematic search and included in this review. The genes associated with water sports are ACE, ACTN3, AMPD1, CKMM, GABPB1, GDF8, HFE, HIF1A, MCT1, NOS3, PPARA, PPARGC1A, TFAM, UCP2, UCP3, VEGFR, and VEGFR2 (Fig. 2).

----- Table 1 and Fig. 2 about here -----

Genetic variants associated with rowing performance

Several studies have investigated the associations of various gene polymorphisms with rowing performance. Six studies revealed that the ACTN3 RR genotype of the rs1815739 R577X polymorphism was associated with better rowing performance than the XX or RX genotypes were [30-35]. Rowers were shown to have an excess ACE I allele and the ACE II genotype of the ACE rs1799752 Alu I/D gene polymorphism, which was linked to high aerobic capacity and athletic excellence in six studies [30, 34, 36-39]. However, conflicting evidence has also been reported. Muniesa et al. (2010) studied the same ACE gene variants and reported that the ACE II genotype was relatively low in elite lightweight male rowers [40]. They also studied the same ACTN3 rs1815739 variant and reported no significant association between ACTN3 genotype and rowing performance. Ciężczyk et al. (2011) reported that the AMPD1 TT genotype and T allele were negatively associated with athletic performance. [41]. However, Muniesa et al. (2010) studied the same variant and reported no association of AMPD1 genotypes with rowing performance [40].

Conflicting evidence was found for the CKMM rs8111989 and NOS3 rs1799983 G/T polymorphisms. Two studies reported that the CKMM A/G polymorphism was not an important determinant of endurance performance in Polish or Spanish rowers [40, 42]. Another study reported that endurance athletes' CKMM A allele and AA genotype frequencies were significantly greater [43]. One study reported no significant association

of the NOS3 G/T polymorphism with the physical performance status of Polish rowers [44]. Szelid et al. (2015) performed a study on the Hungarian population and reported that rowers with Asp allelic variant carriers had greater right ventricular mass and stroke volume indices than athletes with a Glu/Glu genotype did. The G allele of the GABPB1 rs7181866 A/G polymorphism was found to be associated with greater endurance capability in rowers, as evidenced by higher frequencies of the AG genotype and G allele [45].

Limited evidence was found for a significant association between HIF1A, MCT1, PPAR α , UCP2, and UCP3 gene polymorphisms and rowing performance. The HIF1A Ser allele and Pro/Ser genotype [46], the AA genotype and A allele of the MCT1 gene [47], the PPAR α GG genotype [48], the UCP2 Val allele and the UCP3 T allele [30] were significantly prevalent in elite rowers. One study reported that the VEGF C allele was significantly greater in endurance athletes, especially rowers [49].

Genetic variants associated with canoeing performance

Orysiak et al. (2018) reported that ACTN3 and ACE gene polymorphisms were not associated with the power or strength of canoyers, irrespective of sex [50]. However, in a previous study, Orysiak et al. (2015) reported that the ACTN3 XX genotype was advantageous for endurance canoe performance [51]. They also reported that individuals with the RR genotype presented greater power output in countermovement jumps than those with other genotypes did, presenting contradictory findings. In contrast, Szelid et al. (2015) reported no association between the NOS3 G/T polymorphism and athletic performance [45].

Genetic variants associated with kayaking performance

Orysiak et al. (2015) reported that the ACTN3 XX genotype provides an advantage for endurance kayakers [51]. The TFAM Ser12Thr polymorphism is also associated with the physical performance of kayakers [52]. The VEGF G-634C polymorphism is related to the performance of athletes, and the C allele was markedly greater in the group of athletes who participated in endurance events [49].

Genetic variants associated with surfing

Limited evidence was found for an association between gene polymorphisms and surfing performance. Only one study reported that the ACTN3 X allele is associated with the endurance capacity of Turkish windsurfers [53].

Genetic variants associated with rafting

One study reported an association between the ACE I/D gene polymorphism and rafting performance. The study reported that high-level rafters with the ACE ID and DD genotypes contribute to being champions [5].

Quality assessment

The studies demonstrated variable risk-of-bias scores, with the majority achieving high scores, reflecting generally robust methodologies (Table 2). Notably, 22 studies were of high quality, highlighting their rigorous design and low risk of bias. Six studies were found to have a higher risk of bias due to limitations in selection, comparability, and exposure assessment. Overall, the assessment reveals a predominance of high-quality studies, although attention should be given to the lower-scoring studies when interpreting the overall findings.

----- Table 2 about here -----

Discussion

This systematic review attempted to examine articles and summarize the influence of genes and their polymorphisms associated with various on-water sports performances, including rowing, canoeing, kayaking, surfing, sailing, and rafting. The purpose of this study was to investigate and validate how genes are involved in impacting athletic performance and whether the impact is positively associated, i.e., performance-enhancing, or if it negatively impacts athletes. We found that several genes and their variants associated with on-water sports performance have been identified by previous researchers. The majority of gene associations identified in our study pertained to rowers and involved genes such as ACE, ACTN3, AMPD1, CKMM, NOS3, GABPB1, GDF8, HFE, HIF1A, MCT1, PPARA, PPARGC1A, TFAM, UCP2, UCP3, and VEGFR. This was followed by associations found in canoers (ACE, ACTN3, AMPD1, and NOS3) and kayakers (ACTN3,

NOS3, and TFAM). Fewer studies have been conducted on surfers (ACTN3) and rafters (ACE), and no studies have linked genes to sailing.

Association of ACTN3 gene polymorphism with on-water sports performance

The ACTN3 rs1815739 R577X polymorphism is the most studied gene among all the other genes. Studies have shown that the ACTN R/X genotype is associated with better rowing performance than the RR and XX genotypes are [30, 31, 33, 35, 38]. Ulucan et al. (2013) reported that the XX genotype was linked to prolonged endurance performance in Turkish windsurfers [53]. The same conclusions were reached by Orysiak et al. (2015) for kayakers and canoers [51]. Orysiak et al. (2015) reported an association of the RR genotype in canoers with strength and power development in SBJ and CMJ in canoers, which was later contradicted by other studies [50, 54]. ACTN3 is a sarcomeric protein present only in type 2 (fast, glycolytic) fibers. α -actinin-3 plays an important role in muscle contraction, cytoskeletal organization in skeletal muscle, maintaining the structural integrity of the Z-line, regulating calcineurin signaling and calcium (Ca^{++}) handling, and skeletal muscle metabolism. The nonfunctional X allele leads to α -actinin3 deficiency [55]. Previous studies have reported that the presence of the ACTN3 R allele is beneficial for sprint and power performance, whereas the ACTN3 XX genotype is beneficial for endurance performance [51, 56-59].

Association of ACE gene polymorphism with on-water sports performance

Another most extensively researched polymorphism found was the ACE rs1799752 I/D variant. Studies on rowers reported an association between the ACE I allele and II genotype with high endurance and athletic success [30, 36-39], the latter being the same for rafters where the ACE ID and DD genotypes were linked to success [5]. In contrast, a lower ACE II genotype was also reported in rowers [40]. ACE is a zinc metallopeptidase widely distributed on the surface of endothelial and epithelial cells. ACE plays an important role in two physiological systems: the renin–angiotensin system (RAS) and the kinin–kallikrein system [60]. It converts inactive angiotensin I to the active octapeptide and potent vasoconstrictor angiotensin II, which is the main active product of the RAS. It also acts on the adrenal cortex, causing the release of aldosterone, which stimulates tubules in the kidneys, allowing them to reabsorb more sodium and water from the urine. ACE metabolizes bradykinin, which is a strong vasodilator, forming the inactive

metabolite bradykinin. These effects directly act to increase the amount of fluid in the blood, compensating for a loss in volume and increasing blood pressure [60]. Previous studies on athletes of various sports reported that the presence of the ACE DD genotype demonstrated better results in sports that require speed, such as sprinting [61-63]. On the other hand, individuals with the II genotype, and therefore a lower ACE activity in blood, demonstrate better results in sports requiring endurance [36, 61]. Subjects with the ID genotype had intermediate levels, indicating codominance [60].

Association of NOS3 gene polymorphism

Previous studies have explored the associations of NOS3 gene polymorphisms with athletic performance, particularly in sports such as rowing and canoeing. Cieszczyk et al. (2010) reported no association between the NOS3 G/T polymorphism and physical performance in rowers [44]. Similarly, Szelid et al. (2015) reported that there was no genetic association between the NOS3 polymorphism and performance in both rowing and canoeing [45]. The NOS3 gene encodes endothelial nitric oxide synthase (eNOS), an enzyme crucial for the production of nitric oxide (NO) in blood vessels. NO plays a significant role in vasodilation and angiogenesis, which increases oxygen delivery to muscles during exercise, potentially enhancing performance [64]. However, on the basis of the current evidence, findings suggest that the NOS3 gene G/T variant genotype does not appear to be a predictor of performance in these specific sports and requires further research.

Gene variants in energy metabolism and association with performance

Few studies have investigated the role of genetic variants related to energy production and their association with on-water athletic performance. Researchers have reported that the AMPD1 gene T allele and TT genotype are negative factors for athletic performance, whereas others have reported that the AMPD1 gene T allele and TT genotype are not associated with performance overall [40, 41]. The AMPD1 gene encodes adenosine monophosphate deaminase 1, an enzyme crucial in the purine nucleotide cycle that plays a significant role in muscle energy metabolism during exercise. It is involved in the anaplerotic replenishment of tricarboxylic acid (TCA) cycle intermediates and the regulation of nucleotide levels in the cell [65-67].

Another study reported that the AG genotype and G allele of the GA binding protein transcription factor subunit beta 1 (GABPB1) gene (rs7181866) were associated with greater endurance in rowers [68]. The GABPB1 gene is involved in the regulation of oxidative phosphorylation in muscles [68, 69]. The increased oxidative phosphorylation efficiency could explain the improved endurance observed in individuals with these genetic variants. Furthermore, Muniesa et al. (2010) and Eider et al. (2015) reported that CKMM gene variants were not associated with rowing performance [40, 42]. The CKMM gene encodes muscle-specific creatine kinase, an enzyme essential for the rapid regeneration of ATP during high-intensity, short-duration exercise [43, 70]. Despite its critical role in energy metabolism, the specific CKMM variants studied did not appear to significantly influence rowing performance.

The PPARGC1A gene (location: 4p15.1) Gly482Ser polymorphism (rs8192678 G/A) is not associated with rowing performance [40]. This transcriptional coactivator of the PPAR family is involved in mitochondrial biogenesis, glucose utilization, fatty acid oxidation, angiogenesis, thermogenesis, and muscle fiber-type conversion toward slow-twitch type I fibers [71-74]. However, the PPAR α GG genotype was found to be prevalent in elite Polish rowers and beneficial for endurance performance [48]. PPAR α plays a critical role in lipid metabolism and energy homeostasis [75, 76]. The TFAM gene Ser12Thr polymorphism is associated with the physical performance of kayakers [32]. TFAM is encoded by the TFAM gene (location: 10q21) and is important for mtDNA transcription, replication, and maintenance. Different types of exercise, particularly endurance training increase TFAM mRNA levels and enhance mtDNA replication [77-80]. This in turn contributes to an increase in mitochondrial mass in human skeletal muscles.

The UCP2 Val allele and UCP3 T-allele were found to be associated with high aerobic performance in rowers [30]. Melnikov et al. (2019) also reported that the UCP2 Val/Val genotype and the UCP3 T/T genotype are genetic predictors of increased HRV in highly trained athletes [81]. The literature suggests that UCP2 and UCP3 regulate the mitochondrial membrane potential and help manage reactive oxygen species [82-84]. A common UCP2 gene (location: 11q13) Ala55Val polymorphism (rs660339 C/T) has been variably associated with altered body mass index, physical activity, and changes in energy expenditure [85]. More specifically, the Val/Val genotype has been reported to be associated with increased exercise efficiency, increased metabolic efficiency and physical

activity, and increased volume of maximum oxygen consumption [30, 86]. Acute exercise upregulates UCP3 as a result of increased plasma-free fatty acid levels. The T allele of UCP3 is reportedly associated with increased skeletal muscle UCP3 mRNA expression and increased aerobic capacity in Russian female rowers [30].

Association of growth factor gene variants with on-water sports performance

Previous studies have indicated a significant association between certain gene polymorphisms and sports performance, particularly in kayakers and rowers. The VEGF G-634C polymorphism is associated with the physical performance of kayakers [49, 87]. Compared with other athletes, endurance athletes, including rowers, were found to have a greater frequency of the VEGF C allele [49]. Additionally, the VEGFR2 gene was found to be associated with the endurance performance of female rowers [87]. VEGF is a major growth factor for endothelial cells and regulates angiogenesis [88, 89]. Thus, it plays an important role in delivering oxygen to tissues during prolonged physical activity and enhancing endurance. VEGFR2, also known as the kinase insert domain receptor, is essential for introducing the full spectrum of VEGF angiogenic responses to aerobic training [87, 90].

In contrast, the GDF8 (myostatin) gene variant K153R was not significantly associated with rowing performance [40]. These findings suggest that myostatin, which negatively regulates skeletal muscle cell proliferation and differentiation, may not directly influence rowing performance. Additionally, the MCT1 A allele and AA genotype were significantly more common in top elite and elite rowers [47]. The monocarboxylate transporter-1 (MCT1) gene, located on chromosome 1p12, encodes MCT1, which is mostly present in slow-twitch muscle fibers. MCT1 is necessary for lactate transport to myocytes for oxidation, which is critical for energy production during intense physical activity [91-93].

HIF-1 α , encoded by the HIF1A gene located at 14q23.2, plays a key role in the body's response to hypoxia by regulating key glycolytic enzymes and glucose transporters, such as GLUT1 and lactate dehydrogenase, facilitating increased glucose uptake and anaerobic metabolism during hypoxia [94, 95]. The Pro/Ser genotype and Ser allele of the HIF1A gene were significantly common in rowers [46]. Collectively, these findings underscore the complex interplay between various genetic factors and their

contribution to sport performance. Understanding these associations can aid in identifying potential genetic markers for athletic talent and performance optimization.

The selected studies also have limitations which should be considered while interpreting the results. In our systematic review, we only included studies published in English and search to specific databases, which may have led to the exclusion of relevant studies published in other languages and indexed elsewhere. Since studies related to some genes have conflicting opinions, further research is needed. Factors other than genes, such as environmental factors and gene–environment interaction factors, should be considered when studying genetic influences in sports. Some studies included a smaller number of athletic populations, which makes them statistically weak compared with control groups. Most studies have focused on a single polymorphism, which is a limiting factor, as different genetic components combine to influence athletic performance. For some studies, it was not practically possible to obtain a larger number of athletic populations, as the number or competition level of athletes for that specific sport is lower. This makes the sample size of the athletic population smaller.

Future perspectives

Genetic testing of athletes is now a part of sports talent identification, athletic training and performance enhancement, allowing athletes to understand their genetic predispositions and tailor training programs to maximize their potential. Identifying and selecting athletes through genotyping at a young age might allow earlier adoption of specialized and dedicated training. Understanding the role of genetics in water sports performance would also help coaches bring the best out of genetically gifted athletes with proper strategies and help researchers understand the extent to which each of these factors affects performance outcomes among naturally gifted versus normal or trained athletes. Commercial sports companies may potentially misuse genetic testing of athletes with preferences being given to some athletes over others, which would be a violation of fundamental human rights.

Conclusions

Several genes and their polymorphisms have been associated with athletic performance in water sports, such as rowing, canoeing, kayaking, surfing, and rafting. This review comprehensively summarizes numerous structural and functional genes and their

variants associated with water sports performance. Among the 17 different genes from 8 different populations and 28 studies, the genes ACTN3, ACE, AMPD1, GABPB1, PPARA, TFAM, VEGF, VEGFR2, UCP2, UCP3, HIF1A, and MCT1 were associated with water sports performance. However, several risks of bias have been identified in these studies. Notably, 22 studies were of high quality, and 6 studies were found to have a higher risk of bias. No studies have been conducted on sailors, whereas conflicting and limited evidence has also been reported for certain games, such as surfing and rafting, which needs to be studied further. More studies are needed in different ethnicities with larger sample sizes to support a better understanding of the etiology. However, these genetic variant data can be utilized for the talent identification and nurturing of water sports athletes. Collaborative genome-wide association studies are also advised to investigate additional possible genetic variations. Future studies on gene expression and gene–gene interactions might explain the specific role of these gene polymorphisms in enabling success in these sports.

Abbreviations

ACE	Angiotensin converting enzyme
ACTN3	Alpha-actinin-3
AMPD1	Adenosine monophosphate deaminase-1
CKMM	Muscle creatine kinase
GABPB1	GA-binding protein transcription factor beta-1
GDF8	Growth differentiation factor-8 or myostatin
HFE	Hereditary Hemochromatosis
HIF1A	Hypoxia-Inducible Factor 1 Alpha
MCT1	Monocarboxylate Transporter 1
NOS3	Nitric oxide synthase 3
PPARA	Peroxisome proliferator-activated receptor Alpha
PPARGC1A	Peroxisome proliferator-activated receptor gamma co-activator 1 alpha
SNP	Single-nucleotide polymorphism
TFAM	Mitochondrial transcription factor A
UCP2	Uncoupling protein 2
UCP3	Uncoupling protein 3
VEGFR	Vascular endothelial growth factor receptor

VEGFR2 Vascular endothelial growth factor receptor 2

Declarations

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Competing Interests

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Ethics approval and consent to participate

Not applicable.

Consent for publication

Not applicable.

Data availability

The datasets are available from the corresponding author on reasonable request.

Author contributions

All authors contributed to the conception and methodology design of the systematic review. AD and VR performed the database search. VR and RK performed data screening and data extraction. Validation of the included and excluded articles was done by VKG, PM, and AD. Data visualisation was done by VKG and AD. The first draft of the manuscript was written by VR and RK. VKG, PM, and AD commented on previous versions of the manuscript and did review and editing. All authors read and approved the final manuscript.

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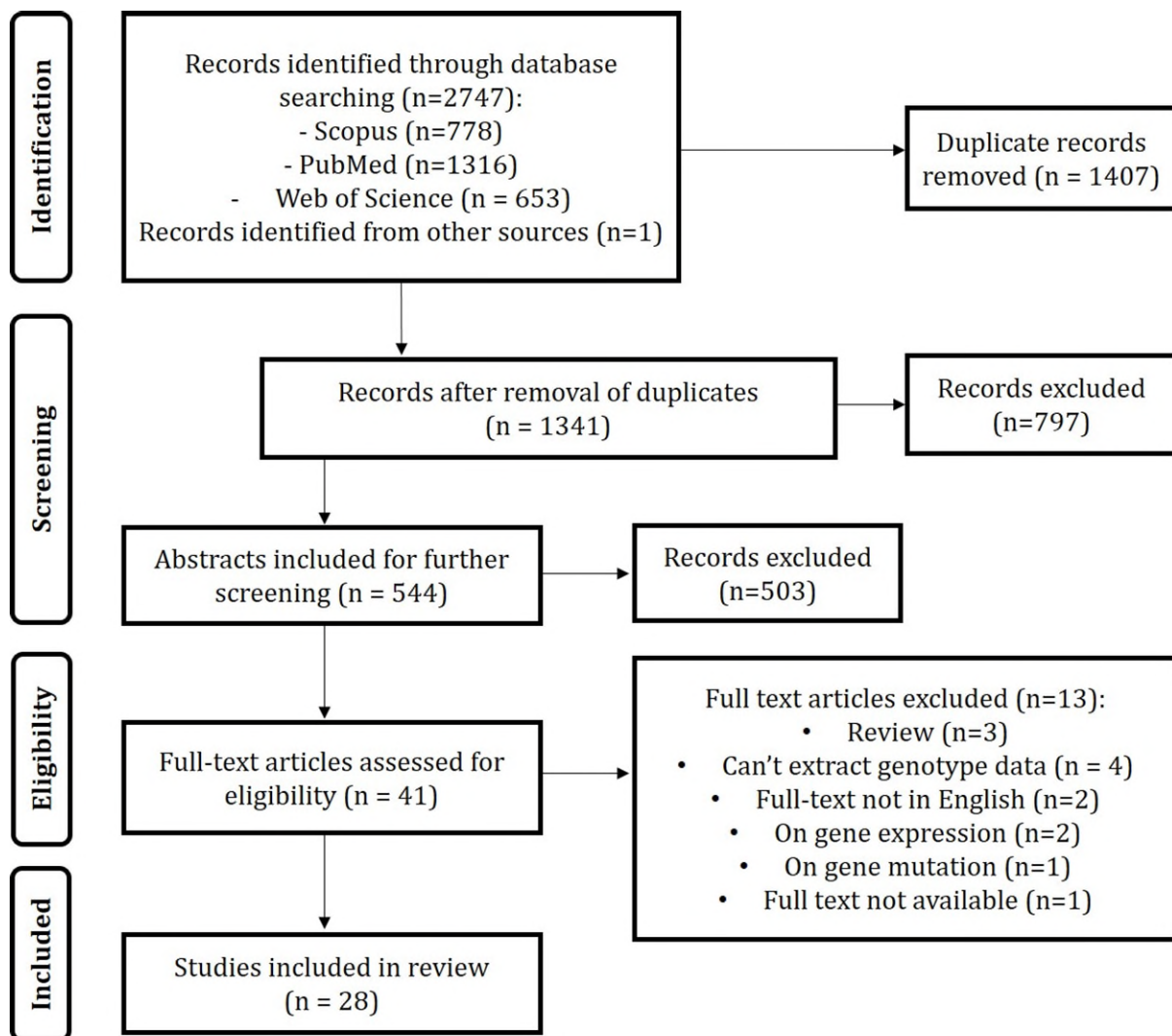


Fig. 1 Showing the study selection process (according to PRISMA checklist)

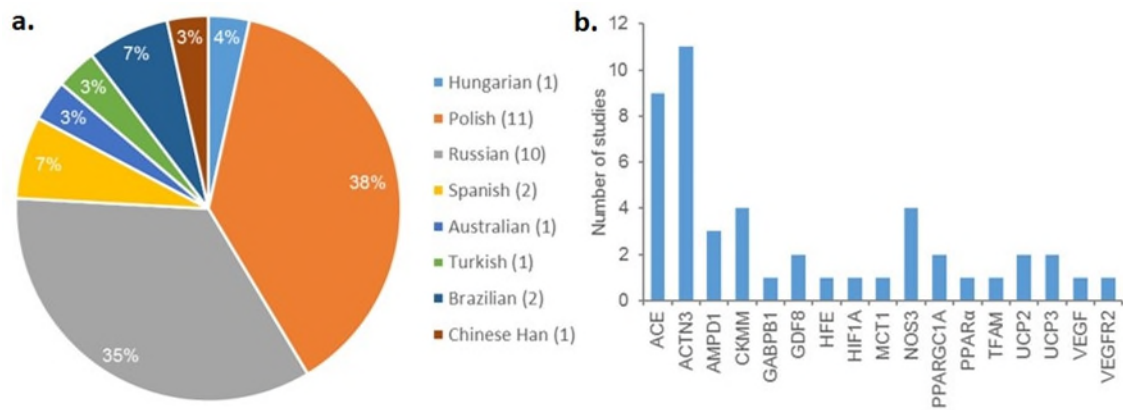


Fig. 2 Demographics of the included studies (a) ethnicity of the participants and (b) number of studies on genetic polymorphisms related to on-water sports performance

Table 1 Summary of the included studies

Author/s (Year)	Study design	Population studied	Athlete's level of performance	Sport	Control (n)	Case (n)	Gene Name	Variant	Associated with performance?
Szelid et al. 2015 [45]	Cross- sectional	Hungarian	International	Kayaking Rowing Canoeing	155 (M 109; F 46)	21 22 19	NOS3	rs1799983	No / May be
Orysiak et al. 2015 [51]	Case-control	Polish	National	Canoeing Kayaking	354 (M)	32 (M) 54 (M)	ACTN3	rs1815739	Yes
Orysiak et al. 2015 [54]	Cross- sectional	Polish	National	Canoeing	--	185 (86 M Canoers)	ACTN3	rs1815739	Yes
Melnikov et al. 2019 [81]	Pilot Study	Russian	National	Rowing	--	23	UCP2 UCP3	rs660339 rs1800849	Yes
Melnikov et al. 2018 [39]	Case-control	Russian	College	Rowing	855 633	22 (M)	ACE NOS3	rs1799752 4b/4a	Yes
Eider et al. 2015 [42]	Case-control	Polish Russian	Elite	Rowing	684 1170	127 (M) 93	CKMM	rs8111989	No
Fedotovskaya et al. 2014 [47]	Case-control	Russian	National	Rowing	467	101	MCT1	rs1049434	Yes
CieSzczyk et al. 2012 [46]	Case-control	Polish	Elite Non-elite	Rowing	160 (M)	44 (M) 83 (M) 127 (M)	HIF1A	rs11549465	Yes
Maclejewska- Karłowska et al. 2012 [68]	Case-control	Polish	Elite Non-elite	Rowing	130 (M)	30 (M) 25 (M) 55 (M)	GABPB1	rs7181866	Yes
CieSzczyk et al. 2012 [35]	Case-control	Polish	Elite Non-elite	Rowing	204 (M)	37 (M) 46 (M) 80 (M)	ACTN3	rs1815739	Yes
Fedotovskaya et al. 2012 [43]	Case-control	Russian	College	Boat racing	1116	95	CKMM	rs8111989	Yes
CieSzczyk et al. 2011 [41]	Case-control	Polish	Elite Non-elite	Rowing	251 (M)	44 (M) 83 (M)	AMPD1	rs17602729	Yes

						127 (M)			
Maciejewska et al. 2011 [48]	Case-control	Polish	Elite Non-elite	Rowing	115 (M)	30 (M) 25 (M) 55 (M)	PPARα	rs4253778	Yes
Muniesa et al. 2010 [40]	Case-control	Spanish	Elite	Rowing	123 (M)	39 (M)	ACTN3 ACE PPARGC1A AMPD1 CKMM GDF8	rs1815739 rs1799752 (K153R)	No Yes No No No
Ahmetov et al. 2010 [31]	Case-control	Russian	Elite	Rowing	1211 (M 532; F 679)	187	ACTN3	rs1815739	Yes
Akhmetov et al. 2010 [52]	Case-control	Russian	--	Boat racing Kayaking	1113 (M 526; F 587)	193 35	TFAM	rs1937	Yes
Cieřarczyk et al. 2010 [44]	Case-control	Polish	Elite & non- elite	Rowing	160 (M)	63 (M; 34 elite; 29 non-elite)	NOS3	rs1799983	No
Ahmetov et al. 2009 [87]	Case-control	Russian	National	Rowing	603 (M 202; F 401)	106	VEGFR2	rs1870377	Yes
Cieřarczyk et al. 2009 [37]	Case-control	Polish	Elite & non- elite	Rowing	115 (M)	55 (M; 30 elite; 25 non-elite)	ACE	rs1799752	Yes
Ahmetov et al. 2008 [49]	Case-control	Russian	--	Rowing Kayaking	1073 (M 488; F 585)	192 35	VEGF	rs2010963	Yes
Ahmetov et al. 2008 [30]	Case-control	Russian	College/ National	Rowing	855 (M 292; F 563)	230 (M 164; F 66)	ACE ACTN3 NOS3 UCP2 UCP3	rs1799752 rs1815739 -- -- --	Yes

Gayagay et al. 1998 [36]	Case-control	Australian	National	Rowing	114	64 (M 43; F 21)	ACE	rs1799752	Yes
Ulucan et al. 2013 [53]	Cohort	Turkish	National	Surfing	--	8 (M 5; F 3)	ACTN3	rs1815739	Yes
Oishi et al. 2016 [5]	Cohort	Brazilian	National	Rafting	--	13 (M)	ACE	rs1799752	--
Orysiak et al. 2018 [50]	Cross-sectional	Polish	National	Canoeing	--	104 (M); 38 (F)	ACE ACTN3	rs1815739	No
Miarka et al. 2017 [33]	Cross-sectional	Brazilian	Elite and Non-elite	Rowing	--	100 (M); 37 (F)	ACTN3	rs1815739	Yes
Santiago et al. 2010 [38]	Cross-sectional	Spanish	World class & national	Rowing	123 (M)	39 (world class); 15 (national)	ACE	rs1799752	Yes
							ACTN3	rs1815739	
							AMPD1	s17602729	
							CKMM	--	
							HFE	s1799945	
							GDF8	rs1805086	
Wei Q 2021 [34]	Cross-sectional	Chinese Han	National elite rowers	Rowing	--	109 (M); 134 (F)	PPARGC1A	rs819267	Yes
							ACE ACTN3	rs1799752 rs1815739	

M = male, F = female. ACE = angiotensin converting enzyme, ACTN3 = alpha-actinin-3, AMPD1 = Adenosine monophosphate deaminase-1, CKMM = Muscle creatine kinase, GABPB1 = GA-binding protein transcription factor beta-1, GDF8 = Growth differentiation factor-8 or myostatin, HFE = Hereditary Hemochromatosis, HIF1A = Hypoxia-Inducible Factor 1 Alpha, MCT1 = Monocarboxylate Transporter 1, NOS3 = Nitric oxide synthase 3, PPARGC1A = Peroxisome proliferator-activated receptor gamma co-activator 1 alpha, TFAM = Mitochondrial transcription factor A, UCP2 = Uncoupling protein 2, UCP3 = Uncoupling protein 3, VEGFR = Vascular endothelial growth factor receptor, VEGFR2 = Vascular endothelial growth factor receptor 2.

Table 2 Risk-of-bias of included studies assessed by the Newcastle-Ottawa Scale (NOS)

Study	Selection	Comparability of cases	Exposure	Total score
Szelid et al. 2015 [45]	★★★★	★	★★★	8
Orysiak et al. 2015 [51]	★★★★	★★	★★★	9
Orysiak et al. 2015 [54]	★★	★	★★	5
Melnikov et al. 2019 [81]	★★	-	★★	4
Melnikov et al. 2018 [39]	★★★	★★	★★	7
Eider et al. 2015 [42]	★★★★	★★	★★★	8
Fedotovskaya et al. 2014 [47]	★★★★	★★	★★★	8
CieSzczyk et al. 2012 [46]	★★★★	★★	★★★	9
Maclejewska-Karłowska et al. 2012 [68]	★★★★	★★	★★★	9
CieSzczyk et al. 2012 [35]	★★★★	★★	★★★	9
Fedotovskaya et al. 2012 [43]	★★★	★	★★★	7
Cięszczyk et al. 2011 [41]	★★★★	★★	★★★	9
Maciejewska et al. 2011 [48]	★★★★	★★	★★★	9
Muniesa et al. 2010 [40]	★★★★	★★	★★★	9
Ahmetov et al. 2010 [31]	★★★	★	★★★	7
Akhmetov et al. 2010 [52]	★★★	★★	★★★	8
Cięszczyk et al. 2010 [44]	★★★★	★★	★★★	9
Ahmetov et al. 2009 [87]	★★★	★	★★★	7
CieSzczyk et al. 2009 [37]	★★★★	★★	★★★	9
Ahmetov et al. 2008 [49]	★★★	★	★★★	7
Ahmetov et al. 2008 [30]	★★★	-	★★★	6
Gayagay et al. 1998 [36]	★★★★	★★	★★★	9
Ulucan et al. 2013 [53]	★★	-	★★	4
Oishi et al. 2016 [5]	★	-	★★	3
Orysiak et al. 2018 [50]	★★	★	★★	5
Miarka et al. 2017 [33]	★★	★	★★	5
Santiago et al. 2010 [38]	★★★★	-	★★	6
Wei Q 2021 [34]	★★	★★	★★	6

Study selection: 4 items, 1 star can be awarded for each item; comparability of cases: 2 stars can be awarded; exposure: 2 items; 1 star can be awarded for each item. A maximum score of 9 is possible (eight items in total).

Supplementary Files

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