Genetic Influence on Athletic Performance

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***Abstract*—Athletic performance is a multifactorial trait shaped by the complex interplay of genetic, environmental, and behavioral influences. While training, nutrition, and psychological resilience have long been considered the primary factors behind athletic success, recent advances in genomics highlight the significant role of genetic variation in determining physical capabilities. This study explores the genetic basis of athletic performance, focusing on key genes such as ACTN3 and ACE, which are associated with muscle strength, power, and endurance. Utilizing a bioinformatics approach, the research aims to develop a lightweight model capable of classifying genotype based on individuals' performance analysis.**

***Index Terms*—Genetics, athletic performance, ACTN3, ACE gene, bioinformatics, genotype classification.**

I. Introduction

The pursuit of athletic excellence has captivated humanity for millennia, driving individuals to push the boundaries of physical capability through rigorous training, strategic nutrition, and unwavering dedication. While the impact of these environmental factors is undeniable, a growing body of evidence underscores the significant role that our inherited genetic blueprint plays in shaping the traits that underpin athletic prowess.

From the explosive power of a sprinter to the enduring stamina of a marathon runner, the variation observed in athletic aptitude among individuals is, in part, a reflection of their unique genetic makeup. This paper will delve into the intricate relationship between genetics and athletic performance, examining key genes and their polymorphisms associated with different physiological attributes relevant to sport.

II. Background

Athletic performance is a complex, multifactorial trait influenced by a combination of genetic, environmental, and behavioral factors. Traditionally, excellence in sport has been attributed largely to intense physical training, optimal nutrition, coaching, and mental resilience. However, as our understanding of human genetics has advanced, it has become increasingly evident that genetic factors play a critical role in determining an individual's physical capabilities and athletic potential.

Scientific research has revealed that traits such as aerobic capacity, muscle strength, power, endurance, flexibility, and recovery time show varying degrees of heritability. For example, studies estimate that aerobic capacity (VO_2max), a key determinant of endurance performance, is about 50% heritable. Muscular strength and power also show heritability estimates ranging from 30% to 80%, depending on the muscle group and type of contraction studied. Additionally, body morphology traits such as height and limb length — which influence performance in sports like basketball or swimming — are up to 80% heritable.

This research aims to investigate the influence of genetic variation on athletic performance using bioinformatics methods. By focusing on key genes such as *ACTN3* and *ACE*, and leveraging genomic datasets, the study will explore how specific genetic markers correlate with traits relevant to sports performance.

III. Literature Review

A. Genetic Basis of Physical Performance

Elite performance is a polygenic trait.The genes that have been associated with performance or performance-related phenotypes to date have been extensively reviewed. Generally speaking, different sets of genetic sequence variants have been associated with endurance performance and sprint/power events.

Rather than merely listing and describing specific genes that have been associated with performance and performance-related traits, we will discuss the current knowledge of the role that genes play in determining four of the many intrinsic traits known to contributes to elite performance phenotypes. These are sex, height, skeletal muscle properties and VO2max [1]. As mentioned, not all of these traits necessarily affect performance in all sporting codes or to the same extent, but they illustrate the complexity of genetic factors on single phenotypic traits, and thus on the complexity of exercise performance.

B. Key Genes Associated with Athletic Traits

Genes are important in determining physical and physiological traits, including those associated with athletic performance. In recent years, researchers have focused on the genetic basis of athletic performance, with the goal of identifying genes that may influence an individual's ability to excel in sports. The genetic basis of athletic performance has significant implications for athletes, coaches, and sports organizations. It may be possible to develop personalized training and nutrition plans

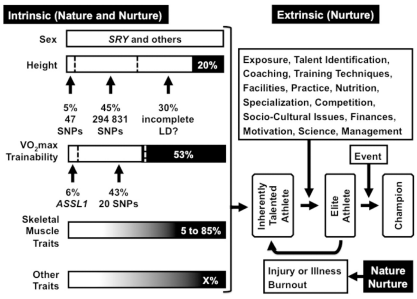

Fig. 1. Genetic and environmental factors

that maximize an athlete's potential for success by identifying genetic factors that may impact athletic ability.

Genes Role in determining traits: Genes are the fundamental units of heredity that determine an individual's traits. They are DNA segments found on chromosomes that encode proteins that are involved in various physiological processes within the body. Genes play a critical role in determining an individual's physical abilities, such as muscle strength, endurance, and agility, in the context of athletic performance.

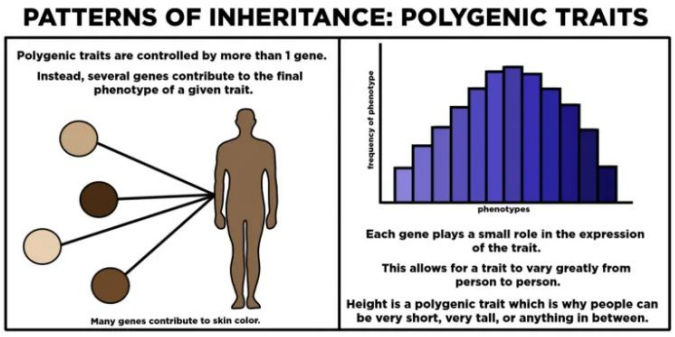

Fig. 2. Polygenic inheritance

Polygenic Inheritance and Athletic Performance: The concept of polygenic inheritance holds that many different genes contribute to the expression of a specific trait. Many genes are likely to play a role in determining an individual's physical abilities in the context of athletic performance. A single gene, for example, may influence an individual's muscle strength, whereas another gene may influence their endurance. As a result, the combined effect of multiple genes determines an individual's overall physical capabilities.

IV. Key Performance Genes

There are several types of genes that can influence athletic performance. Though many specific genes and sequence variants (polymorphisms) within genes have been associated with performance, many of the findings to date have not been adequately replicated. Two notable exceptions are the angiotensin-1 converting enzyme insertion/deletion (ACE I/D) polymorphism, and the α-actinin-3 (ACTN3) R577X polymorphism, both of which have been examined in several populations using a variety of experimental approaches.

The ACTN3 gene, which codes for a protein found in fast-twitch muscle fibers, is one example. This gene has been linked to muscle strength and power, especially in sprint and power athletes [2].

Another gene that may influence athletic performance is the ACE gene, which is involved in the production of angiotensin-converting enzyme in the body. This enzyme is involved in blood pressure regulation and may also affect an individual's ability to use oxygen during exercise.

A. ACTN3

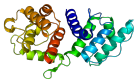

Fig. 3. Alpha-actinin-3

The ACTN3 gene is found on chromosome 11 and encodes the protein alpha-actinin-3, which is found primarily in fast-twitch muscle fibers. The R and X alleles of the gene, which determine whether or not a person produces alpha-actinin-3 in their muscle fibers, are the most common variants [2]. Individuals with the RR genotype (two copies of the R allele) outperformed those with the XX genotype (two copies of the X allele) in endurance performance. This is thought to be due to the lack of alpha-actinin-3 in fast-twitch muscle fibers, which may shift the muscle fiber composition towards a more oxidative, slow-twitch phenotype that is better suited for endurance exercise.

B. ACE

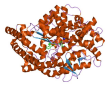

Fig. 4. Angiotensin-converting enzyme.

The ACE gene, which is found on chromosome 17, encodes the enzyme angiotensin-converting enzyme (ACE). The ACE protein helps to regulate blood pressure and fluid balance in the body. The I and D alleles are common variants of the gene that influence ACE protein levels. Individuals with the II genotype (two copies of the I allele) have higher endurance capacity than those with the DD genotype (two copies of the D allele) [2]. This is due to the fact that the II genotype is linked to lower ACE activity and higher levels of bradykinin, a vasodilator that increases blood flow to the muscles during exercise.

V. Genes and Endurance Sports

Endurance sports necessitate the efficient delivery of oxygen to the muscles as well as the use of energy sources. By regulating these physiological processes, genes play a significant role in determining an individual's ability to perform endurance activities. Several genes have been identified in research

that may influence an individual's ability to participate in endurance sports. The ACE gene, for example, regulates the levels of the enzyme angiotensin-converting enzyme in the body. Individuals with a specific variant of the ACE gene have been found to have higher endurance capacity.

The Relationship Between ACE Activity and Endurance Performance: Insights from South African Ironman Triathletes

A study investigated the association between plasma ACE activity and endurance capabilities in a cohort of 145 South African-born Caucasian male triathletes participating in the demanding South African Ironman Triathlon (either the 2000 or 2001 event) [3].

Their findings revealed a tendency for athletes with lower mean plasma ACE activity to achieve faster overall finishing times. Notably, a statistically significant positive correlation emerged between plasma ACE activity and total finishing time among participants who completed the gruelling event in under 15 hours. This suggests that higher ACE activity was associated with longer completion times within this performance group.

Furthermore, the study delved into the relationship between ACE activity and performance in the individual disciplines of the triathlon. Positive correlations were observed between plasma ACE activity and the finishing times for both the cycling and running segments of the race, indicating that higher ACE activity was associated with slower performance in these endurance-dependent stages. Interestingly, no significant correlation was found between plasma ACE activity and performance in the swimming portion of the triathlon.

In conclusion, this study provides evidence suggesting a link between systemic ACE activity and endurance performance in humans engaged in ultra-endurance events. The observed trend towards lower ACE activity in faster finishers, coupled with the significant positive correlations with overall and discipline-specific (cycling and running) finishing times, warrants further investigation into the underlying mechanisms through which ACE influences endurance capacity.

Human sporting phenotypes result from the interaction of genetic variation with environmental stimuli. The ACE I/D polymorphism is but one such genetic factor –the D allele tending to be associated with power/sprint performance,and the I allele with endurance sports. The prevalence of both the D and I alleles in populations worldwide suggest that they may both have offered different survival advantages.That of the I allele may relate to improved endurance performance, and enhanced oxygen utilization in times of both exercise and illness. The D allele, being associated with gains in strength with training, may offer separate advantages related directly to strength itself, but also to the acquisition of increased muscle bulk in response to muscle strength training/high loading. In addition, how-ever, ACE genotype influences a variety of other phenotypes, such as haemorrhage response to the outcome from infection-all of which may offer separate evolutionary selection pressures beyond those exerted through 'fitness phenotypes'alone.

Table II. Studies in athletes associating the *ACE* insertion/deletion (I/D) genotype with sporting performance

Study	Cohort	No. of subjects and ethnicity	Performance[a]	Outcome measure[b]	Association with performance	I/D associations
Cam et al.[45]	Sprinters	88 Caucasian	Non-elite	Performance	Yes	D and short distance
Cerit et al.[46]	Army	186 Caucasian	Army	Performance	Yes	D and short duration
Colakoglu et al.[47]	Athletes	99 Caucasian	Non-elite	Performance	Yes	D and strength
Juffer et al.[48]	Footballers	52 mixed	Elite	Prevalence	Yes	D more prevalent
Lucia et al.[49]	Cyclists	50 Caucasian	Elite	Performance	Yes	D/D and endurance
Munisea et al.[50]	Mixed	141 mixed	Elite	Prevalence	Yes	D/D and endurance rowers
Winnicki et al.[51]	Mixed	233 mixed	Sedentary	Performance	Yes	D/D and sedentary lifestyle
Nazarov et al.[9]	Mixed	217 Caucasian	Elite	Sport performance	Yes	D/D and short distance
Costa et al.[52]	Swimmers	72 Caucasian	Elite	Prevalence and performance	Yes	D/D and short distance
Woods et al.[34]	Swimmers	102 Caucasian	Elite	Prevalence and performance	Yes	D/D and short distance
Giaccaglia et al.[53]	Elderly	213 mixed	Sedentary	Training	Yes	D/D and strength
Zhao et al.[54]	Army	67 Chinese	Army	Performance	Yes	D/D and VO_{2max}
Tsianos et al.[55]	Climbers	284 mixed	Elite	Performance	Yes	I and high ascent
Gayagay et al.[31]	Rowers	64 Caucasian	Elite	Prevalence	Yes	I and endurance
Myerson et al.[29]	Runners	91 Caucasian	Elite	Sport performance	Yes	I and endurance
Montgomery et al.[1]	Army	78 Caucasian	Army	Performance	Yes	I and endurance
Collins et al.[33]	Triathletes	166 Caucasian	Elite	Performance	Yes	I and endurance
Hruskovicova et al.[56]	Runners	445 Caucasian	Elite	Performance	Yes	I and endurance
Cieszczyk et al.[57]	Rowers	55 Caucasian	Elite	Prevalence	Yes	I and endurance
Min et al.[58]	Track and field	277 Japanese	Non-elite	Prevalence	Yes	I and endurance
Rankinen et al.[36]	Mixed	192 Caucasian	Elite	Prevalence and performance	Mixed	I and endurance

Fig. 5. ACE Gene and Human Performance

A. ACE Insertion/Deletion Polymorphism and Human Physical Performance

The ACE gene contains a well-characterized insertion/deletion (I/D) polymorphism in intron 16, consisting of the presence (insertion, I) or absence (deletion, D) of a 287 base pair Alu repeat. This polymorphism has a significant impact on circulating and tissue ACE activity, with the D allele being associated with elevated ACE levels. Consequently, individuals homozygous for the D allele (D/D) exhibit markedly greater ACE activity compared to I/I or I/D genotypes, with implications for cardiovascular, muscular, and metabolic functions.

Multiple studies have confirmed the association between ACE genotype and athletic performance. The I allele is more commonly found in elite endurance athletes and is correlated with enhanced endurance capacity, particularly in events requiring prolonged aerobic effort such as marathons, triathlons, and long-distance rowing. Conversely, the D allele tends to be overrepresented in strength and power athletes, including sprinters and short-distance swimmers. The proposed mechanism behind these genotype-performance associations includes ACE-mediated regulation of bradykinin degradation and angiotensin II production, influencing vasodilation, blood pressure, and muscle perfusion during exercise [4].

B. ACE Polymorphism and Cardiac Muscle

The ACE D allele has been linked to increased left ventricular (LV) hypertrophy in response to training, likely mediated by enhanced angiotensin II signaling through the AT1 receptor. Angiotensin II functions as a trophic factor in cardiac tissue, promoting myocardial growth and remodeling. In a cohort of 140 male military recruits, D/D individuals demonstrated a significantly larger increase in LV mass after 10 weeks of physical training compared to I/I genotypes. This pattern was replicated in elite endurance athletes and wrestlers, where D allele carriers exhibited higher LV mass indices and a greater prevalence of ECG-defined LV hypertrophy. training [4].

C. ACE Genotype and Skeletal Muscle

In skeletal muscle, the ACE D allele has been associated with greater gains in muscle strength and mass in response to resistance training. This may be attributable to increased angiotensin II levels, which enhance protein synthesis and hypertrophic signaling pathways. For example, D allele carriers showed significantly greater improvements in quadriceps strength following a 9-week resistance training program. This genotype-performance relationship has also been observed in patients with chronic diseases such as COPD, indicating a robust influence of ACE polymorphism on muscle phenotype.

On a cellular level, the I allele has been linked to a higher proportion of type I (slow-twitch) muscle fibers, which are more fatigue-resistant and suited for prolonged aerobic activity. This fiber-type distribution supports the association of the I allele with superior endurance performance. Moreover, bradykinin may mediate metabolic effects in skeletal muscle by enhancing insulin sensitivity, glucose uptake, and substrate utilization. These kinin-mediated effects may partly explain why I allele carriers demonstrate improved muscle metabolic efficiency and less fatigue under prolonged exercise conditions [4].

D. ACE and Maximal Oxygen Consumption (VO_2max)

Despite its established role in performance phenotypes, the relationship between ACE genotype and maximal oxygen uptake (VO_2max) remains inconsistent across studies. VO_2max is a key determinant of aerobic performance and is governed by both central (cardiac output) and peripheral (oxygen extraction) factors. While some studies have reported higher VO_2max in I/I individuals—particularly in postmenopausal women and patients with cardiac dysfunction—other research involving larger and more diverse cohorts failed to replicate this finding [4].

One potential explanation lies in the heterogeneity of study populations, including differences in baseline fitness, age, sex, and training status. In highly trained individuals, where cardiac structural adaptations are maximized, the genotype effect may be less pronounced. Additionally, studies in high-altitude environments have shown a higher frequency of the I allele among elite mountaineers, suggesting a potential advantage in oxygen-limited conditions. The I allele may support enhanced ventilatory responses and improved arterial oxygenation during hypoxia, further reinforcing its relevance in endurance-related contexts.

VI. Genes and Power Sports

Genetics play a significant role in determining an individual's potential in power sports such as weightlifting, sprinting, and jumping—activities that demand explosive strength and high power output. While environmental factors like training, nutrition, and recovery are crucial, genetic factors are estimated to account for up to 50% [5] of the variation in power performance among individuals. Skeletal muscle traits, including strength and lean mass, are highly heritable, with studies showing heritability estimates ranging from 30–85% [5] for muscle strength and 50–80 percent for lean muscle mass.

This strong genetic influence highlights the importance of understanding specific genes and biological pathways involved in muscle development and performance. For athletes and coaches, insights into these genetic components can guide personalized training strategies, optimize performance, and potentially identify talent early in athletic development.

A. The Genetic Edge of ACTN3 in Power Sports

One of the most widely studied genes in relation to power performance is ACTN3, which encodes the protein α-actinin-3. This protein is expressed in fast-twitch (type II) muscle fibers, which are responsible for generating rapid and forceful contractions necessary for explosive movements such as sprinting, jumping, and weightlifting. Individuals who possess the functional variant of the ACTN3 gene (the R allele) are able to produce α-actinin-3, potentially giving them a performance advantage in power-based sports. In contrast, individuals with the non-functional variant (the X allele) lack this protein but may excel in endurance activities.

Although the exact functions of α-actinin-3 are still being researched, it is believed to play a structural role in maintaining muscle fiber integrity, as well as potential roles in muscle signaling and metabolism. The strong link between ACTN3 and athletic performance makes it a key genetic factor of interest for sports scientists, coaches, and athletes aiming to optimize training and talent identification in power sports.

B. A Study on the ACTN3 R577X Polymorphism and Its Association with Power and Endurance Athletic Performance

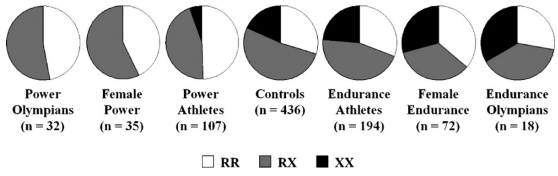

Fig. 6. R577X genotype frequencies

Researchers investigated the impact of a common genetic variation in the ACTN3 gene, specifically the R577X polymorphism, which affects the production of α-actinin-3, a protein found in fast-twitch muscle fibers. Individuals with two functional R alleles (RR genotype) produce normal α-actinin-3, those with one functional and one non-functional allele (RX) produce reduced amounts, and those with two non-functional alleles (XX) produce no α-actinin-3 at all. Despite its absence in about 18 of the European population, lack of this protein does not cause muscle disease, though it may influence muscle function [6].

To test this, scientists conducted case-control studies comparing elite athletes (both power and endurance) to non-athlete controls. The results, shown in Figure 3, revealed a significantly lower frequency of the XX genotype in power athletes—only 6% compared to 18% in controls. Notably, none

of the power Olympians or female power athletes had the XX genotype, suggesting a strong link between the presence of α-actinin-3 and enhanced performance in sprinting, jumping, and other power-based sports. In contrast, endurance athletes showed a slightly higher frequency of the XX genotype, though this trend was not always statistically significant [6].

These findings suggest that α-actinin-3 plays a key role in fast muscle fiber function, making the RR genotype more favorable for power-based activities, while XX individuals may have a slight advantage in endurance sports. Although the association with endurance is less clear, the consistent results across studies in both Australian and Finnish athletes support the conclusion that ACTN3 is a meaningful genetic factor in determining athletic potential, particularly for power performance.

C. Genetic Influence of the ACE I/D Polymorphism on Athletic Power Performance

The ACE gene, which encodes the angiotensin-converting enzyme involved in blood pressure regulation and cardiovascular function, has also been explored as a potential genetic factor influencing power performance. This gene exists in two main variants: insertion (I) and deletion (D). Some studies suggest that individuals with the I allele may have a genetic advantage in developing greater muscle strength and power output compared to those with the D allele. For instance, research has shown that I allele carriers often display enhanced performance in strength-related activities [7].

However, the evidence is not entirely consistent; other studies have failed to find a significant difference in power or strength between individuals with the I and D variants [8]. These mixed results indicate that while the ACE gene may play a role, its effect on power performance is likely influenced by other genetic, environmental, and training-related factors.

VII. Implementation Overview

In this project, we aim to analyze and classify gene sequences related to **ACTN3** and **ACE**, two genes known for their association with athletic performance and physiological traits. Our implementation consists of two main components: data labeling and model training.

A. *Idea 1: Sequence Labeling*

In the context of genotype classification, sequence labeling can be achieved using various methods if a reference dataset or variant information is available. One such method is:

- **Naïve String Matching:** This approach scans gene sequences for known genetic motifs to classify them into genotypes such as RR, RX, or XX. It is computationally efficient and effective when the target markers are well-defined. We tested this method using two sequences from the ACTN3 gene obtained from the NCBI dataset:
  - `NC_000011.10:66546395-66563334`
  - `NC_060935.1:66542637-66559581`

  The Naïve String Matching approach successfully labeled the genotypes as **RR** for both sequences based on known allelic variants at the target positions.

These technique allow us to generate labeled data from raw sequences, which is critical for downstream analysis.

B. *Idea 2: Model Training for Genotype Classification*

In this study, we aimed to predict the ACTN3 genotypes (RR, RX, XX) based on performance analysis data. To achieve this, we utilized several machine learning models and optimized their hyperparameters using Grid Search Cross-Validation. Furthermore, we employed Principal Component Analysis (PCA) for dimensionality reduction and visualization of the data, which helped us gain insights into the distribution of genotypes in the high-dimensional feature space.

1) Dataset and Preprocessing: The dataset used in this study is derived from the ACTN3 study, available at https://zenodo.org/records/5626501. It includes performance metrics across multiple athletic activities, such as Squat (Sq), Countermovement Jump (CMJ), Drop Jump (DJ), and Sprint. The features provided include maximal velocity, maximal acceleration, jump height, rate of force development, and power.

Data preprocessing was a crucial step in preparing the dataset for modeling:

- **Data Cleaning:** We ensured that there were no missing or null values in any of the columns.
- **Feature Scaling:** To standardize the data, all continuous features were scaled to ensure that they contributed equally during model training.

2) Model Selection and Hyperparameter Optimization: We explored several machine learning models for genotype classification, including:

- **K-Nearest Neighbors (KNN)**
- **Random Forest (RF)**
- **Decision Tree (DT)**
- **Naive Bayes (GNB)**

Each of these models was optimized using Grid Search Cross-Validation to identify the best-performing hyperparameter configurations. The Grid Search process involves systematically testing a range of hyperparameter values and selecting the ones that maximize model performance.

The hyperparameters of each model were tuned as follows:

- **KNN:** The optimal number of neighbors (K) and distance metrics were explored.
- **Random Forest:** The number of trees and splitting criteria were adjusted.
- **Decision Tree:** Hyperparameters such as tree depth, and minimum samples for splitting and leaf nodes were optimized.
- **Naive Bayes:** Evaluated with its standard parameters, though additional fine-tuning for smoothing parameters was considered.

3) Dimensionality Reduction and Visualization with PCA: To facilitate visualization and better understand the structure of the data, we applied Principal Component Analysis (PCA). PCA is a dimensionality reduction technique that projects high-dimensional data into fewer dimensions while preserving as much of the variance as possible. In this study, PCA was used solely for visualization, helping us to observe the distribution of different genotypes (RR, RX, XX) within the reduced feature space. This allowed us to visually identify any patterns or clusters in the data.

A PCA plot of the data distribution is shown in Figure where we can see how the genotypes (RR, RX, XX) are distributed across the first two principal components.

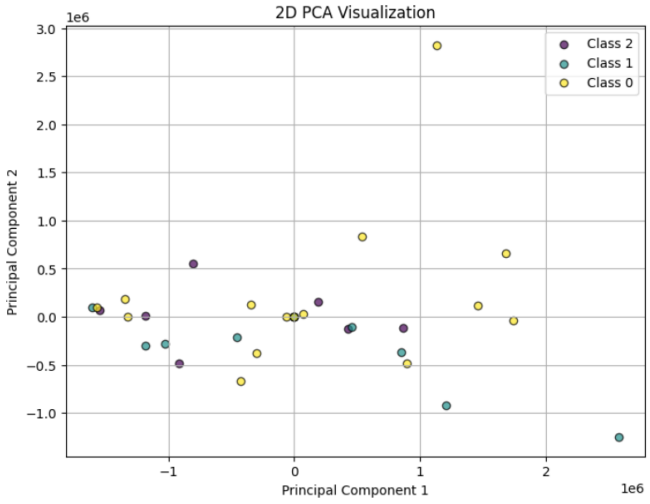

Fig. 7. PCA visualization

4) Training Model Comparison and Results: After training and tuning the models, we evaluated their performance. The training accuracies of the models were compared, and the results are summarized below.A comparison of the training accuracies is presented in Figure

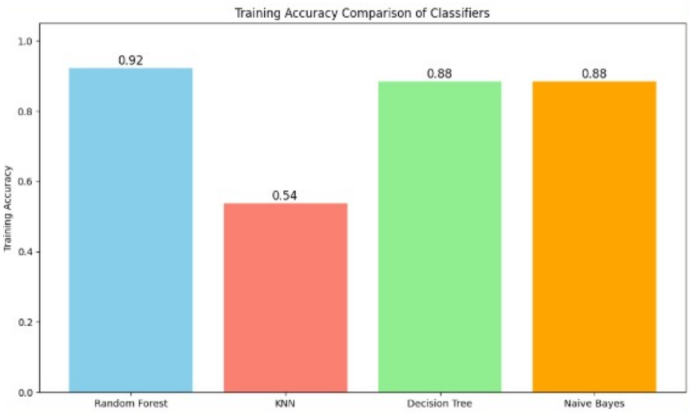

Fig. 8. Training accuracies

VIII. Conclusion

This research reinforces the understanding that athletic performance is not solely determined by external factors like training and nutrition but is also deeply influenced by genetic predisposition. By focusing on genes such as ACTN3 and ACE, the study highlights how specific genetic variants can contribute to differences in strength, power, and endurance among individuals. Through a bioinformatics-driven model, we demonstrated the feasibility of classifying genotypes based on performance traits, providing a foundation for future personalized approaches to training and talent identification in sports. These insights pave the way for a more integrated view of athletic development that includes both genetic and environmental factors.

IX. Figures and Tables

List of Figures

X. Conclusion

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