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GENETICS AND ATHLETIC PERFORMANCE

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(Review Article)

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Abstract
Sports Scientists and researchers in related disciplines unquestionably agreed on the fact that the level of physical development and process of adaptation to the exertions are due to the genetic makeup of individuals. Reasons such as lifestyle, environmental interactions and coming from different origins (ethnicity) by skin color are also facts that cannot be ignored in revealing the unique changes between people. The features encoded in DNA sequences or chains that cause changes between humans also determine the limits of physical performance. Therefore, the genetic characteristics of the Olympic athletes allow them to perform at a high level, more precisely to be slightly ahead of other competitors. The number of candidate genes associated with the potential for higher levels of physical exertion to occur is quite high. However, the number of genes that directly trigger athletic success among these candidate genes is also very limited. There are so many factors that affect athletic performance that even if one competitor is considered superior to another, the result is almost always doubtful. It is clear that ideal genes probably push an athlete to greatness, but that these ideal genes also do not guarantee an optimal result. The complexity of genetic and environmental influences on the physiological, motor and psychological characteristics also severely limits the scope of determining athletic abilities and generating a genetic profile of targeted success. Undoubtedly, athletes with a favorable genetic profile who interact with correct training practices are more likely to achieve higher performance levels. However, it is likely that the possible combinations of genetic and environmental factors that result in elite performance will remain enormous and often unpredictable.

Keywords: *Candidate Genes, Athletes, ACE, ACTN3*.

Introduction

Perfect software that takes place in genes and their sequences (DNA) are inconceivable formations that transform the physical and metabolic characters of the organism into a lifestyle in its mysterious adventure, which has been continuously structured since its time in the womb when life began to be encoded. Gene-triggered behaviors determine the vitality level, exercise adaptation, duration of effort, the level of calories expended, the likelihood of developing fatal diseases and above all, quality of life. The said changes (differences in sequences), provide increased physical effort for some in a shorter time while cause some to reach results in a longer time than expected. The ability of the physical exertion level to reach the expected target with increasing exertions occurs as a result of training exertions that continue for a minimum of three thousand (3000) hours for the extraordinarily talented, and for longer periods for other individuals who progress more slowly, which results from small but important changes in the DNA chains in question.

In the studies conducted to date on the role of genetic factors in the development of strength (1), power, muscular endurance and flexibility, the most comprehensive data on muscle fitness was obtained from family studies (2). In these studies, muscular endurance was evaluated by measuring the maximum number of sit-ups performed in 60 seconds and the number of push-ups completed without time limit; muscle power was evaluated by measuring grip strength, and body flexibility was evaluated by measuring the sit and reach test. In long-term studies conducted within the framework of physical fitness, determination of inheritance predictions as 37% for sit-up, 44% for a push-up, 48% for flexibility, and 37% for grip strength in the sit-up tests shows genetic factors are contradictory. Inheritance ability was observed as 41% in a sit-up, 52% in push-up capacity, 32% in grip strength effort and 48% in flexibility development under ideal conditions

(3). In family studies, muscular endurance and muscle strength development were evaluated with the sitting test and the knee extension test, which measures the maximum isometric contraction of the quadriceps muscles, respectively (4, 5). The significant familial similarity was observed in the performance of both muscle conformity, and the genetic transfer was explained as 21% for muscular endurance and 30% for muscle strength. It was observed that the direct effect of flexibility (the sit and reach test) gender differences and genetic factors account for 51% of women and 72% of men (6). In addition, in a study of 748 male twins, concentric strength measurements of the knee, trunk, and elbow varied between 63% and 87% minimum heritability, and the inheritance estimates detected in the corresponding isometric strength measurements also varied between 82% and 96%. Muscle strength or explosive power (speed-strength), in which the maximal strength is released in the shortest possible time usually measured by jumping tests (vertical jump or standing long jump). Most of the evidence regarding the heritability of muscle strength were obtained from studies conducted on twins, however, the data of studies on families are very limited. In one of the rare studies conducted on families (vertical jump tests), parent-child correlations were found to vary between 0.17 and 0.54 depending on the age groups of the children. In another study of twins (age 10) and their parents, a 65% endurance similarity was observed in vertical jump tests. Genetic prediction ranges of fitness components (biomotor abilities) responses to stimuli are 37-59% for muscular endurance, 32-60% for muscle strength (grip strength), 61-96% for isometric strength, 50-87% for concentric strength, muscle strength, 65% for (vertical jump) and 48-72% for flexibility (sit-reach test). The effect of genetic variations that should be taken into account within the framework of exercise and training science was observed to be approximately 40-60% for aerobic performance, 50-90% for anaerobic performance, 30-70% for muscular vigorous and 20-30% for cardiac functions, respectively.

Family studies related to physical fitness and genetics are very limited. One of the earliest studies found similar performances between fathers and sons when the performance of 24-year-old men compared to the performance of their fathers who were the same age as them 34 years ago. In another family study (559 men and women from 76 families), it was found that the maximal inheritance similarity regarding neuromuscular performance characteristics was 7% for hand stability, 16% for eye hand coordination, and 11% and 24% for the duration of movement and reaction, respectively (7). Physical performance development is affected by various morphological characteristics such as body structure (height and body weight) and the physical structure of the body (8). Family studies have shown that height varies between siblings around 0.30 to 0.50. Approximately 80% of the change in adult height is due to genetic factors. In an important study in a large population, it was stated that 81% of the change in height is due to genetic factors, 12% to common environmental factors and 7% to individual-specific environmental factors (9, 10). The hereditary relationship of body weight is less consistent than observations for height and shows a lower heritability (11).

Sports Scientists and researchers in related disciplines unquestionably agreed on the fact that the level of physical development and process of adaptation to the exertions are due to the genetic makeup of individuals. Reasons such as lifestyle, environmental interactions and coming from different origins (ethnicity) by skin color are also facts that cannot be ignored in revealing the unique changes between people (12). Lifestyle and environmental factors trigger the emergence of characters designed within genes. Climate is the only strong environmental indicator of physical activity, and other components of the physical environment (access to facilities, land structure, traffic) are poorly associated with physical activity (12). The lifestyle and environmental conditions that have changed in the great geography, where great migrations are supposed to have begun, made East Africans as distance runners and West Africans as unrivaled examples of short-term sprint disciplines. The basic reality that determines this condition is genetics (13, 14). Athletes of African origin living in the Caribbean of the Greater Antilles are ideal athletes created by the perfect combination of natural talent (genetic drift) and environmental factors. Their perfectly programmed anatomical structures and the speed gene (ACTN3) polymorphisms present in many are just a few of the differences behind their success. The environmental factors they are involved in and the coincidence of the traditional lifestyle with the DNA sequences caused them to focus on sprinting activities rather than other sports branches. Those who do not have a genetic predisposition or who do not comply with high speed and power parameters in their genes can never be an ideal sprinter. So why do the same traits not appear similarly in every individual? Pre-adolescence speed trait or ability is the main determinant of speed performance that can occur during and after puberty. Therefore, if a person does not have a current genetic background, he will never be a fast runner no matter how hard he works. Explosive power and sprinting ability depend on the composition of fast and slow-twitch muscle fibers and positive reflections or interactions of associated candidate genes. While muscle fibers that produce explosive power or strength increase acceleration more, oxygen-rich muscle fibers with high aerobic capacity or strength can also show high resistance in long-term efforts. You can develop fast-twitch muscle fibers within certain measures with low-intensity exertions (muscular and aerobic endurance) of 60% or less of the targeted heart rate, however, you cannot work your slow-twitch muscles with high-intensity training (exercise intensity of about 80-85% target heart rate). In other words, slow-twitch muscle fibers became a part of activity only when the exercise intensity is 60% or less and at the beginning of the exercise, that's why those who are slow in childhood can never be as fast as expected in the following processes.

Although there are different physical appearances and anatomical structures between individuals, general physical appearance, metabolic processes, and behaviors that differ or are triggered according to the variables or interactions (exercise and exertion type, exercise intensity and duration, etc.) created by the lifestyle, the environmental conditions and the individual depend on the properties of the DNA sequences. The characteristics in question reveal the difference between taking or not taking part in the podium, sitting on a bench or entering the game for top-level performing athletes. For example, East Asians (China, Republic of Korea, etc.) are more successful than their competitors in disciplines such as gymnastics, racing skate and diving in international championships by using their physical structure (flexible and short body limbs). Those who live in the east of Europe and have a muscular athletic body (mesomorph) show superior success in strength and speed-focused branches (weightlifting, wrestling, judo, shot put, discus and hammer throwing etc.) Blacks (Caribbean), whose origins are in Africa, have undeniably accepted their superiority in sprinting activities that require explosive power as a result of changes arising from environmental conditions and lifestyle.

Physical differences between ethnic groups are clear, this clearly shows that changes in body proportions generally evaluated by measuring different body segments (for example), such as arm length, leg length, and sitting height are under the control of genetics. For instance, blacks have longer extremities in throwback compared to whites, this has significant effects on athletic performance (such as blacks playing basketball in the NBA). It is likely that the structural proportions of the body are under the strong influences of genetics. One of the most common methods used to classify individuals according to their physique is the use of somatotype measurements (It is a taxonomy developed by William Herbert Sheldon in 1940 to classify human physics). Somatotype features measure the physique of the individual in three components: endomorphs characterized by the roundness and softness of the contours on the body, athletic mesomorphs characterized by a predominance of muscle, bone and connective tissues, and ectomorphs characterized by linearity and the dominance of surface area over body mass. In a study, 36%, 45% and 42% of endomorph, mesomorph and ectomorph variables of somatotypes have been associated with the genetic and cultural transmission, respectively (2,15). In another study, inheritance rates detected in 329 individuals aged 7-17 selected from African families was 40% for endomorph, 30% for mesomorph and 31% for ectomorph (16); in another study conducted with 103 families, general somatotypes were found to be 56% for endomorph, 68% for a mesomorph, and 56% (3) for ectomorph; in a larger sample group of more than 3000 people, the maximum inheritance characteristics of the general somatotype were explained as 55% for the endomorph, 52% for the mesomorph and 46% (17) for the ectomorph.

The debate on whether somatotypes are shaped by genetic characteristics or the lifestyle and environmental factors, are still ongoing. Another point of view put forward on the subject is that the organ or organelles that contribute the most to the formation during the leaf layer period when the development begins in the womb can affect the body shape of the individual; it may be ectomorph if the first contributing structure to development is the nervous system, or it may be mesomorph if it is the muscular system, or it may be endomorph if it is the digestive system (15). The clearest interpretation that can be expressed about the said formation is that no model can be 100% mesomorph, endomorph or ectomorph. Each individual maintains their life by acquiring certain proportions of characteristics from three different phenotypes. The metabolic differences between individuals make this situation easier to understand. It is unlikely that a mesomorphic or endomorphic athlete will be successful in long-term running; long-distance runners such as Kenyans take advantage of their classical ectomorphic structures and high ratios of slow-twitch muscle fibers, their light body and long extremities. In fact, it is obvious that factors such as environmental conditions, lifestyle and motivation trigger success, despite the performance differences created by all these variables in long-distance running. Because, in a perfect long-distance or marathon runner, about 23 different gene variants associated with endurance capacity must act in coordination. However, the probability of occurrence of this possibility in individuals is almost impossible (14, 19). That is, given the candidate genes associated with aerobic power or capacity, there is no perfect athlete who can achieve a high level of superiority in their origins. However, the least understood genetic variation to date among parameters determining athletic success, such as lifestyle, motivation, family and environment relations, nutrition, psychology, knowledge and experience of the trainer is actually the most important factor to consider when taken into account that training practices and methods are only one of the components that determine the physical performance achievement level. The features encoded in DNA sequences or chains that cause changes between humans also determine the limits of physical performance. Therefore, the genetic characteristics of the Olympic athletes allow them to perform at a high level, more precisely to be slightly ahead of other competitors.

Correct placement of DNA sequences as well as internal and external factors such as environmental interactions, lifestyle, motivation for the development of the physical performance level at the Olympic level facilitates taking part in the Olympic Games (18, 20, 21). Genetic findings related to athletic performance can be a guide for individuals who do sports for a healthy life in planning their exercises. Genetic features not only contribute to the physical development level and performance development but also can make significant contributions to personal interest, enjoyment, effort and motivation of individuals. Certain psychological behaviors or the propensity for motivation and tendency to enjoy favorable to sports-related training and competition are the factors that can prove the most use of genetic screening in talent identification programs (14, 22).

It is unthinkable to ignore the effects of genetics in the comparison of athletic performance and achievement level. If we indicate the said triggering effects in numbers, the heritability level would be 66% for aerobic strength (MaxVO2), 80% for height, 30% for endurance and 83% for strength, respectively. The number of candidate genes associated with the potential for higher levels of physical exertion to occur is quite high. However, the number of genes that directly trigger athletic success among these candidate genes is also very limited. The Angiotensin Converting Enzyme (ACE) gene and the Alpha-actinin-3 (ACTN3) gene are the two most important candidate genes emphasized in relation to athletic performance. The ACE gene regulates body fluids and is responsible for controlling blood pressure. In individuals with high levels of ACE enzymes, due to the increased vasoconstriction, sufficient oxygen delivery to the muscle cells cannot be provided, therefore long-term effort capacity is limited, on the other hand, activity performance requiring short-term explosive power is quite high. In people with low ACE enzyme levels, long-term effort capacity at a marginal level, adaptation to high altitude and performance efficiency occur as a result of the abundant oxygen reaching the tissues. The said gene both regulates blood pressure and affects skeletal muscle functions. In fact, ACE triggers the conversion of the vasoconstrictor agent angiotensin I to angiotensin II, not only as a stronger vasoconstrictor but also as a muscle growth factor involved in muscle hypertrophy due to overtaxing (23). Nitric Oxide initiates Nitric Oxide (NO) vasodilation (dilation of blood vessels) triggered by the Synthase Gene and provides adequate blood flow to tissues during training or physical exertion. High ACE activity reduces the release or production of NO (arginine amino acid is the precursor to nitric oxide). MaxVO2 (aerobic power or maximum oxygen utilization capacity) development and endurance performance are low due to the inability of tissues to get the oxygen they need because of excessive vasoconstriction in individuals with elevated ACE enzyme levels.

The ACE gene reflects two distinct characteristics: long-term endurance and muscle strength. Studies conducted on mountaineers and soldiers show that ACE increases oxygen and nutrients to muscle cells and improves physical performance (24). How can the said gene affect running performance? In order to understand the relationship, it is very important to understand the role of the Renin-Angiotensin System (RAS) and the Angiotensin Converting Enzyme (ACE) in this system (25).

RAS is a hormonal structure that controls blood pressure and body fluids. ACE gene has an important role in the control of plasma ACE levels. There are two key variations in the ACE gene. One of them is called the I allele (long or added) because it has extra-base pairs in its DNA, and the other is called the D allele (short or elided) because it has no base pair. There are three genotypes in this polymorphism: "DD", "II" and "ID" (Table 1.1). These differences show that ACE has an important role in affecting the production of Angiotensin II at the cellular level. While the highest ACE levels in serum (26) and tissue (27) were observed in individuals with the DD genotype, minimal ACE rates were identified in individuals with II genotype. As high ACE levels increase the production of Angiotensin II, the blood flow to the tissue decreases (Figure 1.1). Since ACE DD alleles cannot adequately supply the oxygen needed by tissues due to increased vasoconstriction, their maximum oxygen utilization capacity (maxVO2) is low. People with the ACE DD genotype show higher MaxVO2 levels and short-term aerobic endurance. In those with high ACE D allele frequency, high anaerobic performance, ideal development of muscle strength and mass are observed (28, 29). Those with the ACE DD genotype are associated with high MaxVO2 and short-term aerobic endurance, high performance (6 min) and generally higher ACE activity, therefore they theoretically exhibit sprint/power-oriented performance as they potentially trigger a high rate of type II muscle fibers (29, 30, 31). However, there are also studies in which no positive correlation has been found for DD alleles (each of the variants of a gene that detects a particular trait) to be successful in elite sprint/power disciplines (32, 33).

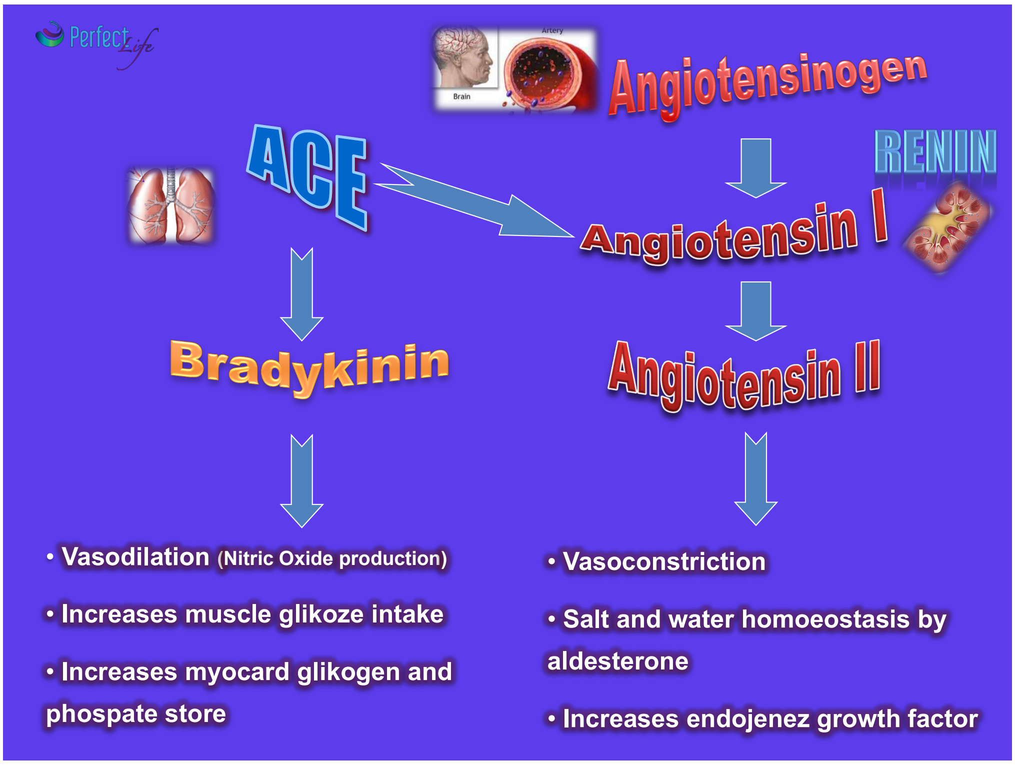
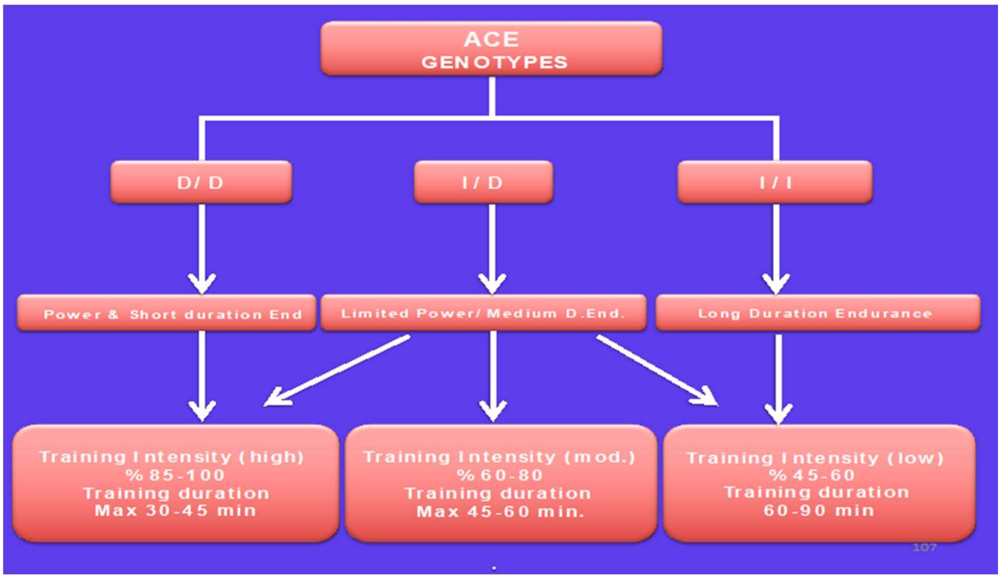


Figure 1.1: Renin Angiotensin System (RAS) and ACE enzyme activity.

Table 1.1: Classification of individuals with ACE genotype and their athletic performance characteristics.



The possible explanation for the metabolic effect on the speed performance of individuals with the ACE genotype has been linked to muscle fiber type differences. Zhang et al. (34) observed that the high percentage aerobic energy resource use of ACE II genotypes was associated with the percentage of muscle fibers (high percentage type I), on the other hand, they observed that the higher percentage of anaerobic performance in ACE DD genotypes was associated with type II muscle fibers. Although more studies are needed to fully explain the mechanism behind the potential relationship between the ACE genotype and human elite sprint performance, the fact that ACE variants can independently affect muscle fiber metabolism should also not be ignored.

ACE (I/D) polymorphism was associated with improvements in performance and exercise duration in various populations. It was observed that those with II genotypes are particularly associated with endurance-focused activities such as long-distance running whereas that those with the DD genotype were associated with a high level of athletic performance in team sports such as powerlifting, bodybuilding, weightlifting, sprinting, swimming, basketball and volleyball, which are related to performance-focused on strength and power. In addition, the ACE genotype was found to be associated with left ventricular mass changes in cardiac muscles in both health and disease conditions and in response to a stimulus to muscle strength development. ACE DD genotypes gave an exaggerated response to training stimuli, while the lowest cardiac growth response was observed in II genotypes. Similarly, ACE DD genotypes were associated with greater strength gain in skeletal muscles in response to exercise in both healthy individuals and chronic disease states. As with general performance, genetic polymorphisms associated with the ACE genotype can affect skeletal muscle strength and metabolic efficiency (35, 36). Cardiac structures and functions are critical limiting factors of endurance performance. In the light of data obtained from family studies, it is understood that genetic factors have a small but significant contribution of approximately 20-30% for cardiac size (37). In order to evaluate cardiac function associated with athletic performance, observable variables such as rest, and heartbeat, blood pressure, stroke volume during exercise, and the response of cardiac output (total amount of blood pumped in 1 minute) to exercise are generally used. In family studies on heredity, the significant familial similarity was found in resting heart rate, systolic and diastolic blood pressure values measured before exercise in sedentary patients; in this framework, the maximum heritability estimates were determined as 32% for resting heart rate, 54% for systolic blood pressure and 41% for diastolic blood pressure. Genetic predictions for basal metabolism (BMI) were 34%, 51% and 42%, respectively, and resting heart rate (RHR) heritability was 34% (39).

A significant relationship can also be predicted between the ACE genotype, which affects metabolic efficiency, and performance in hypoxic environments (high altitude). A significant increase in the I allele frequency was observed in professional British male climbers who climbed higher than 7000 meters without using additional oxygen, compared to control groups. Similar findings were found in 139 climbers aiming for an 8000-meter ascent (40). There are also confirming epidemiological evidence among indigenous peoples who speak the Quechua language and those who live above 3600 meters in South America and the Ladakh region of India. The researchers did not observe any correlation between height and responses to erythropoietin (EPO) stimulation among 63 athletes exposed to an altitude of 2200 meters of the ACE genotype (40, 41, 42, 43).

Another gene commonly prominent in athletic performance development is the alpha-actinin-3 (ACTN3) sprint gene. It is stated that alpha-actinins release high power and high-speed strength by activating fast-twitch fibers in activities (100, 200, 400m, shot put, javelin, hammer, Olympic lifts, body-building, powerlifting, team sports etc.) where speed and explosive power are dominant. Alpha-actinin-3 is associated with super strength and superior endurance performance on the grounds that it also provides an advantage in the development of high levels of muscle contraction and speed performance that can unleash explosive power in muscle fibers. ACTN3 gene provides production (encoding) of the actin-binding protein-alpha-actinin-3 and it forms the main component of the Z line, where actin thin filaments cross-link with fast-twitch muscle fiber cells. ACTN3 gene polymorphism is determined by the presence of X or R at the R577 position. The R allele is generally advantageous in strength and speed-focused sports, while those with the XX genotype are associated with low sprinting ability and muscle strength. Production of alpha-actinin-3 is limited in fast-twitch muscle fibers of XX genotypes. This means that the "C" base is replaced by the "T" base during the single change in the DNA coding of the ACTN3 gene locus of unknown reason, in the construction phase of the muscles. As a result of this unknown change, two different versions of the ACTN3 gene are formed and one of the versions carries the alpha actinin-3 protein to the fast-twitch muscle fibers. The "C" base version without alteration fully performs the ACTN3 gene function and produces quite a lot of alpha-actinin-3 protein (R allele). The alternative T base version (X allele) completes the protein production chain early (premature stop) and leads to the formation of the non-functional alpha-actinin-3 protein.

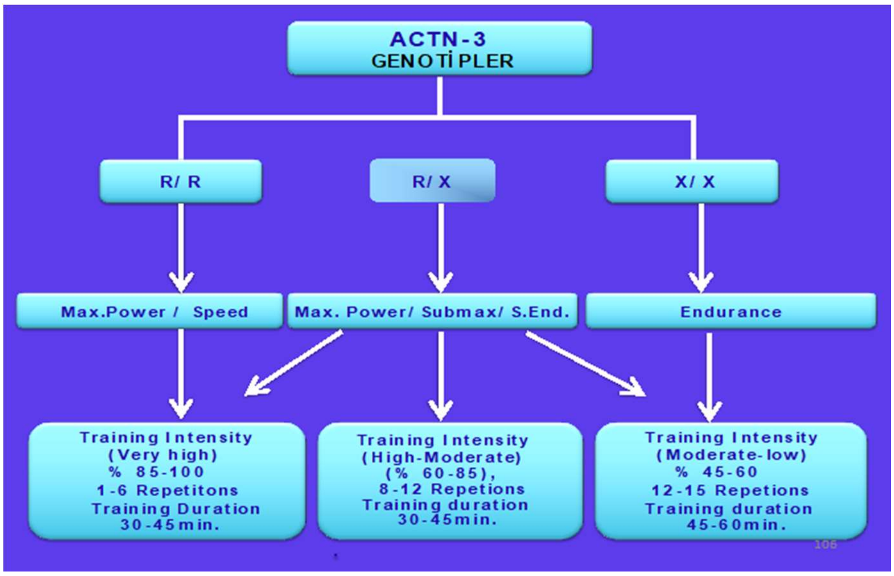
There are two gene regions in the cell nucleus. Since one comes from the mother and the other from the father, the genetic metamorphosis in individuals can be observed in 3 different combinations (Figure 1.2). Those with ACTN3 genotype classified in this scope:

* Homozygous normal (C/C) - RR (same character, one from mother, one from father), no change occurred, has high speed and power generation feature depending on muscle structure,

* Heterozygous carrier (C/T) - RX (different character), one copy of the ACTN3 gene has also been altered, a partial advantage is observed in both traits depending on the muscle structure.

* Homozygous mutant (T / T) - XX (same character, one from mother, one from father), both copies of the ACTN3 gene have changed, has high endurance performance characteristics depending on the muscle structure,

Table 1.2: Classification of those with the ACTN3 genotype and their athletic performance characteristics.



While those with the ACTN3 RR genotype provide a noticeable advantage in strength and sprint activities, those with the XX genotype play an important role in the development of endurance performance. In a study conducted in Australia, it was observed that the rate of ACTN3 577RR genotype was a quite high frequency while the 577XX genotype with α-actinin-3 deficiency was a lower frequency, in top-level sprint/power athletes (sprint, throwing and jumping branches), control group athletes and sedentary groups (44). Recent researches show that Olympic-level athletes interested in power sports need to have at least one copy of the "R" allele (alpha-actinin-3 protein production) to achieve successful performance (45). The top-level sprinting athletes (97%) have at least one copy of the R allele. At least one copy of the R allele is sufficient to produce the alpha-actinin-3 protein needed to increase power performance. These observations and studies show that the deficiency of α-actinin-3 limits the function of fast-twitch muscles for the explosive power needed for sprinting and power performance. Also, fast-twitch fibers and testosterone in low levels were observed in those with the ACTN3 577XX genotype (46).

In the homogeneous study of Papadimitru et al. (2016), the relationship between the ACTN3 R577X and ACE I/D variants of the Olympic athletes (Australian, Brazilian, Greek, Jamaican, Italian, Polish, Russian, Lithuanian, Spanish and US sprinters) performing elite athletics on a large scale and the personal best times of 100, 200 and 400 meters has been studied. As a result of the analysis of 555 individual best 100 m, 200 m and 400 m personal sprints of 346 elite sprinters selected from the countries mentioned in the study in question, 189 male and 66 female sprinters, all white (Australia, Greece, Poland, Lithuania and Russia) and a total of 91 male athletes of African origin (Brazilian, Jamaican, US, Italian and Spanish) participated in the study. In this study, neither ACE II and ACTN3 XX genotypes were found in any sprinter participating in international championships (Olympics, world and continental championships), nor were findings related to ACTN3 RR and ACE DD genotypes in any endurance discipline (47). Within the framework of the research in question, it was observed that male white sprinters with ACTN3 577RR or ACE DD genotype, on average, had the best 200 meters sprint time compared to those with the ACE II genotype. In the 2012 London Olympics qualifiers, it was determined that the sprinter with only one ACE II genotype had 200 m faster running time compared to twelve qualified sprinters with 577RR or 577RX genotype, whereas none of those with ACTN3 577XX genotype made it to the finals. It was also stated that sprinters with ACE DD genotype had the best 400 m sprint time compared to sprinters with ACE II genotype (46.94±1.19 s vs. 48.50±1.07 s, p=0.003). It was also determined that candidate sprint genes (ACTN3 577R and ACE DD) constitute 0.92% and 1.48% of the dominant sprint model time variables, respectively. The research in question revealed that despite the many candidate gene variants and sprint performance based on the environment, the percentage change in a sprint time of the ACE and ACTN3 genes is significant at the elite athletic performance level and that individual differences determine the difference between the world record and just running the final (47). Although the probability of being an elite sprinter/strength athlete is influenced by genetic factors, there are only a handful of genes associated with speed performance. Currently, the most promising candidate gene is ACTN3, which encodes the sarcomeric protein alpha-actinin-3 in skeletal muscle fibers. The alpha-actinin-3 protein is limited to fast, glycolytic type II muscle fibers responsible for the generation of 'explosive' power or strong muscle contractions. The worldwide estimated rate of rare homozygous single nucleotide polymorphism (577XX, rs1815739) in the ACTN3 gene is approximately 16-18%, and in such individuals, α-actinin-3 protein cannot be fully produced. ACTN3 RR genotypes have been associated with sprinting and athletic power performance in elite athletes.

Table 1.3: Training areas associated with energy systems and presumptive locations of ACE & ACTN3 genes.

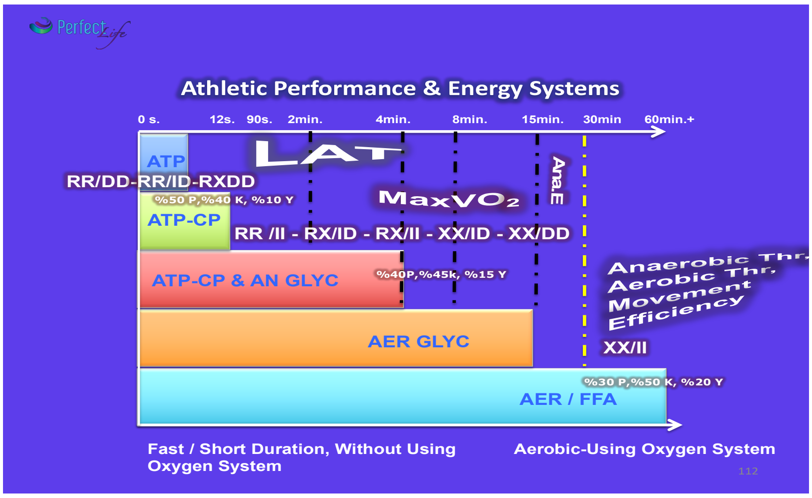
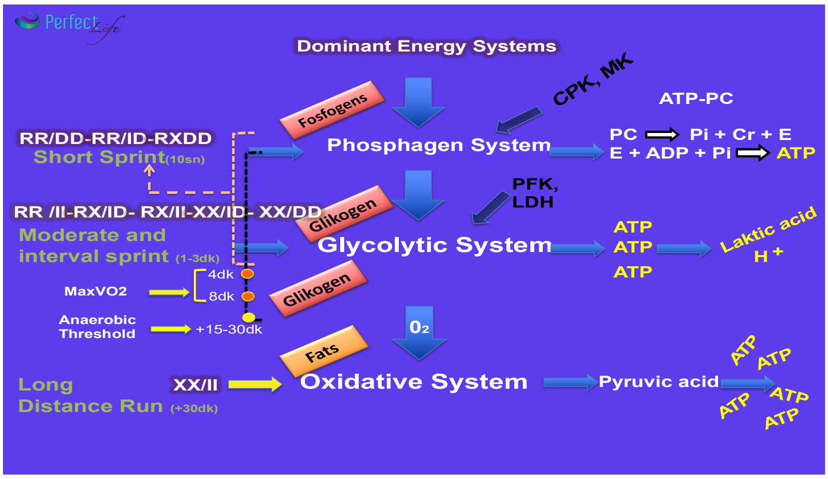
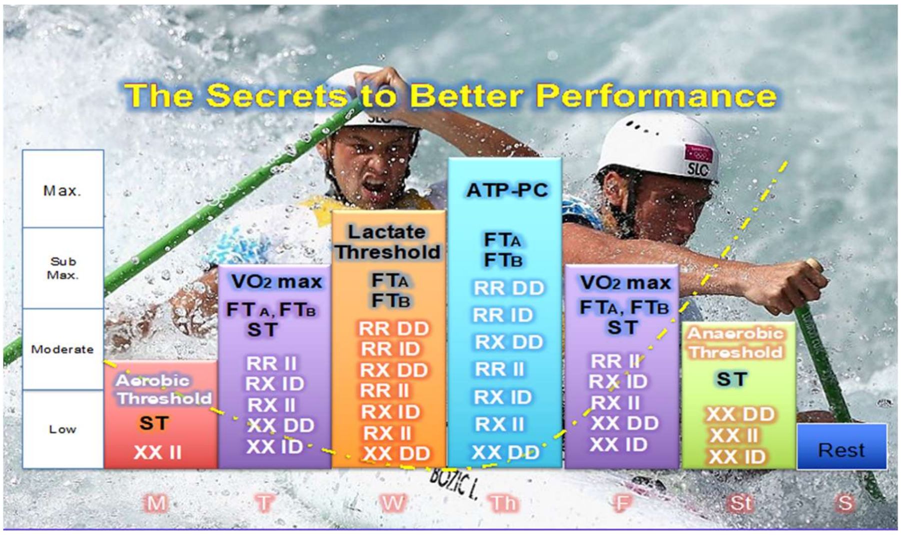


Table 1.4: ACE & ACTN3 genes and dominant energy systems (presumptive approach).



\Table 1.5: Weekly micro cycle associated with ACE & ACTN3 genes, exertion parameters, and muscle fiber types used (presumptive approach).



Considering these candidate gene interactions, their reflections on performance and different parameters, it is very complex and also open to interpretation. You can discover the right athlete with the right gene structure, however, reaching the goal is quite difficult and complicated when all variables such as individualized training, lifestyle, environmental factors and social relations are taken into consideration (Table 1.3-1.5) (48). To become a top-level athlete, years of training is an inevitable necessity, but it is not a guarantee of success. Some are luckier. Because they enter the races with their genetic advantages. For example, Bahamian athlete Donald Thomas won the high jump (2.35 cm.) world championship after just 8 months of training. It should not be forgotten that exercise repetitions do not provide the same improvement for every individual. Even if different individuals do the same exercises at the same time, their development over time will differ. Genetic differences can make some reach their goals in a very short time, while they can make some reach high performance after a very long time (49).

There are so many factors that affect athletic performance that even if one competitor is considered superior to another, the result is almost always doubtful. It is clear that ideal genes probably push an athlete to greatness, but that these ideal genes also do not guarantee an optimal result. While describing perfection in the process of athletic performance development, information derived from genetic testing can at best explain only part of the individual's probability of future success due to the remarkable complexity of athletic performance such as training, motivation, environment and the necessity of years of intense, sport-specific training. The complexity of genetic and environmental influences on the physiological, motor and psychological characteristics also severely limits the scope of determining athletic abilities and generating a genetic profile of targeted success.

Elite athletes set an example of the interactions of genomic and epigenomic features, training practices, changes in their organisms caused by nutrition, lifestyle and environmental factors. With genetic adaptation, the remodeling of the muscles based on usage patterns and their positive response to training stimuli is the best example of the ability to adapt. Researchers make an effort to reveal the adaptation of muscles to stimuli, how muscles work under different conditions (health and disease), and how skeletal muscle structures and properties change through evolution. Individuals with the same genotype respond more similarly to training stimuli than those with different genotypes. Studies to date indicate that there are different genetic components that affect performance-related phenotypes in response to untrained and regular exercise. The initial performance or level of success of the athletes constitutes an important part of the performance change that occurs as a result of the applied training. The fact that the responses to the training reveal individual differences is the most striking reflection of the influence of lifestyle and environmental factors.

Genetic contributions that reveal the ability to develop athletic performance are remarkably intertwined with unique gene-environment interactions unique to each athlete, including changes on mental, psychomotor, and sensory behavior. It is very important to carefully reflect the findings of research on the link between genetics and athletic performance to the world of sports (48). Studies to date have mostly focused on identifying genes underlying physiological traits that constitute an athletic performance, however, the fact that psychological characteristics such as attitude, motivation, strategic thinking and others also contribute to performance has been ignored. Identifying the role of psychological factors in performance is an ongoing challenge for sports science researchers, and it is not clear how necessary they are due to the complexity of these factors.

As a result of short-term and long-term studies on homogeneous groups in order to reveal the relationship between ACE gene and athletic performance, differences were found statistically between candidate gene variants within the framework of the results regarding the effects of exercise and candidate gene of ratios such as short term aerobic endurance (MaxVO2), muscular endurance, body fat percentage, body weight; however, it has been found that these differences vary depending on lifestyle and exercise, and that changes in metabolism caused by environmental factors and lifestyle over time appeared similarly in candidate gene variants, and that the ACE gene variant (DD), which was statistically advantageous at the beginning, was found to produce more or less similar performance results later (after 15 years) with other gene variants (ID and II) (50).

As a result, it has been observed that genetic characteristics are significantly affected by environmental interactions and factors resulting from lifestyle and that the difference in physical performance between individuals is directly related to exercise or lifestyle. When used in athletes performing at an elite level, candidate genes, which elicit athletic ability, are important clues that may reveal the reflection of the scope and intensity of training practices, exertion, resting and recovery processes, nutrition and injury risks to the field or podium. However, the suspicion that the tests or candidate genes are approaches or practices that are far from revealing possible predictions of athletic performance markers that may emerge in children and youth also pervades the minds. Most gene variants and gene products discovered to date have only presumptive effects or is in connection instability with a real variable. An approach using multiple genes that have been studied in a targeted pathway is just beginning to emerge. Although there is considerable interest in gene test panels that can identify athletic potential, the available information is quite limited at this point to justify the development and implementation of such a panel. So many foreign factors come together on the occasion of a sporting event that the result is almost always doubtful, even if one competitor is considered superior to another. It is obvious that ideal genes probably push an athlete to greatness, but that these good genes do not guarantee a result. While describing perfection in the process of athletic performance development, genetic information can at best explain only part of the individual's probability of future performance success due to the remarkable complexity of athletic performance such as training, motivation, environment and the necessity of years of intense, sport-specific training. The complexity of genetic and environmental influences on physiological, psychomotor and psychological traits indicates that the scope for successful genetic profiling will be extremely limited, or that they will be guided to general performance characteristics such as little use beyond typical talent selection approaches.

Conclusion

Although some argue that stability and dedicated effort of the many years and thousands of hours of training necessary to build elite athletic ability, the backgrounds of heritability studies and families of athletes are clear evidence that innate qualities give certain individuals an advantage for athletic endeavors. What matters is whether genetic screening techniques can identify the natural advantage or talent in question as part of talent identification programs. Although it is likely to determine the genes of certain physiological, motor, and psychological traits in the coming years, it is not yet clear whether the identification of these factors in childhood will contribute to successful athlete identification. Undoubtedly, athletes with a favorable genetic profile who interact with correct training practices are more likely to achieve higher performance levels. However, it is likely that the possible combinations of genetic and environmental factors that result in elite performance will remain enormous and often unpredictable. These parameters (genetics, environment and lifestyle) will limit the usefulness of genetic screening as part of a talent identification program. Although only 1 out of 10,000 people is doomed to top-level sports success, it seems highly optimistic that genetic screening will identify that person better than current talent identification strategies.

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