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Review

Genetic test for the personalization of sport training

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Abstract. Genetic variants may contribute to confer elite athlete status. However, this does not mean that a person with favourable genetic traits would become a champion because multiple genetic interactions and epigenetic contributions coupled with confounding environmental factors shape the overall phenotype. This opens up a new area in sports genetics with respect to commercial genetic testing. The analysis of genetic polymorphisms linked to sport performance would provide insights into the potential of becoming an elite endurance or power performer. This mini-review aims to highlight genetic interactions that are associated with performance phenotypes and their potentials to be used as markers for talent identification and trainability. (www.actabiomedica.it)

Key words: sports phenotype, endurance, power, genetic variants, polymorphisms

Introduction

To be a sportsman is a highly demanding job that not only requires determination, dedication, nutrition, supportive environment and intensive training but the 'intrinsic ability' coined by genetic traits. The finding that sport performance has a genetic background became a promising area of research in sports genetics since late 1990s when the first discoveries highlighting hereditary involvement in achieving elite sports status were published (1,2,3). Since then, several studies have been conducted to elucidate the gene-gene and gene-environment interactions that contribute to sport-related phenotypes contributing to elite performance status (4-8). In fact, sports performance is a complex multifactorial phenomenon governed by several intrinsic factors such as genetic polymorphism, psychomotor skills, physical fitness that are greatly influenced by extrinsic factors such as diet, training and health status (9-11).

Sport performance is difficult to define precisely. In fact, it greatly depends on aim and objectives of the sports. For instance an endurance performer such as weightlifter has different parameters of assessing performance than a sprint or power performer such as a runner. This implicates that each sport discipline has unique physiological, psychological, biochemical and anthropometric demands that result in shaping an overall performance phenotype encoded by heritable genetic traits (12,13,14). For instance, endurance performance is largely dependent on maximal oxygen uptake (VO_2 max), VO_2 at lactate threshold and efficiency of movements (15). It is necessary the coordinated action of cardiovascular system and muscular metabolism involving transportation of oxygen to and utilization of oxygen by the muscles (16). Besides that

enhanced aerobic endurance involves elevated mitochondrial gene expression and corresponding enzyme activity during aerobic respiration (17).

On the other hand power performance is dependent upon muscle structure, strength and the ability to generate force without being injured (18). Maximal power is a function of force and velocity of muscle contraction which in turn depends on the cross-sectional area and volume density of myofibrils of muscle fibre (19). Muscle power is the driving force behind sprinting, jumping and weightlifting (20).

Performance-enhancing gene polymorphisms

During the last two decades, several studies have provided compelling evidences that both endurance and power performances are influenced by genetic factors that collectively are called performance-enhancing gene polymorphisms (PEPs) (21). Surprisingly, PEPs are common in general population and more than 200 PEPs have been reported so far (21,22). However, only 20 out of 200 were specifically found in athletes and only 10 could be replicated in association studies (22). Since then approaches such as twin studies, familial aggregation studies, genome wide linkage and association analyses have revealed structural variants in genes having great influence on sport performance indicators such as aerobic endurance, muscular strength and power.

To understand the link between performance and PEPs we will consider the classic example of two of the most well-documented and adequately replicated PEPs, *ACE* I/ D and *ACTN3* R577X that are consistently linked with endurance and power performance phenotypes (23).

Angiotensin-conversion enzyme (ACE)

The pioneering study that revolutionized sports genetics is the finding of a polymorphism in *ACE* gene encoding angiotensin-conversion enzyme (ACE) in 1998 (23). ACE is an important enzyme of renin-angiotensin system which regulates blood pressure by controlling body fluids (24). Besides this, *ACE* is involved in bradykinin degradation, respiratory drive, regulation of inflammatory reactions to lung injury, erythropoiesis, tissue oxygenation, and the regulation of skeletal muscle efficiency (25).

ACE may exist in two polymorphic forms, I or D, depending on an intronic indel of 287 bps (26). *ACE* I has an intronic insertion of 287 bps which results in decreased serum and tissue ACE activity (27,28). *ACE* I/I genotype has been consistently linked to improved endurance performance and high exercise efficiency (28,29). On the other hand, the deleted form of the variant (D allele) is associated with higher circulating and tissue ACE activity (29) and enhanced power and strength performance in sprinting (30). In addition to this significantly higher frequency of *ACE* I allele was reported in elite Australian rowers as compared to normal control (31) while the I/I genotype was more frequently observed than the D/D genotype in elite British mountaineers. Besides this, all the top performers had and ACE I/I homozygous genotype (32). Similar results have been reported by Woods & Montgomery (33) and Thomson et al (34). The role of the *ACE* gene in endurance performance has been recently extensively reviewed (35,36). These systematic reviews revealed that with few exception, I allele is typically associated with endurance performance in elite distance runners, mountaineers, swimmers and rowers while the D allele is associated with elite power-oriented performance and training-related gain of strength (35, 36). Besides that, I allele is involved in the alteration of metabolic response by maximizing oxidation fuel for metabolism whereas the D allele in gaining strength and VO_2 max in response to training (36).

It is worth mentioning that although several studies have reported positive involvement of ACE I/D polymorphism in enhancing endurance and power performance some other studies have failed to report such association which could be due to inclusion of mixed sporting disciplines that results in phenotypic heterogeneity, sample size issues, and other confounding factors such as ethnicity and geography. For instance, none of the ACE I/D alleles\ were linked with the athletic performance in Kenyans depicting the involvement of ethnic and geographic factors (37). This suggests that, although the genotype is associated with elite performance phenotype, the effect of environment and other confounding factors determine the ultimate performance phenotype (38).

α-Actinin-3 (*ACTN3*)

Another classical example of PEPs is *ACTN3* gene that encodes a structural sarcomeric protein α-actin-3 found exclusively in fast type II muscle fibres used during explosive activities (28).

Association of *ACTN3* genotype with human elite athletic performance was first reported by Yang et al in 2003 (39). This was the first PEPs reported for genes regulating skeleto-muscle formation and function (38,39). They reported a significantly higher frequency of the functional 577R genotype in both male and female elite sprinters. Subsequent studies highlighted the association of RR genotype with elite power performance (40) and XX genotype with lower sprinting ability and muscle strength (41). Furthermore the power athletes were 50% less likely while endurance athletes were 1.88 % more likely to have XX genotype as opposed to RR genotype. Moreover the world class endurance performers had 3.7% more chances of having an XX genotype as compared to lower level athletes implicating the importance of ACTN3 at highest performance levels (42). *ACTN3* has been consistently associated with high performance in sprint and power athletes as compared to normal control population where it has no association with physical capabilities (43). Although the role of *ACTN3* in general population is speculative the frequency of homozygous XX allele differs between human population of different ethnic origins e.g. 16% of Africans and approximately 51% of some Eurasian populations have XX genotype suggesting ethnic factors in the inheritance pattern (44).

Genetic variants linked with injury risk

In addition to above mentioned performance indicators the underlying risks for getting injury during sports and training is another important aspect to consider during talent identification. Like other performance associated polymorphisms resistance to injuries and capability to recover is also conferred by genetic variants (45). Athletes generally suffer from concussion (mild traumatic brain injury) and tendinopathies.

Genes linked with concussion

Apolipoprotein E (*APOE*)

Several research groups are trying to find a link between the apolipoprotein E (*APOE*) e4 allele and concussion. APOE e4 has a strong association with Alzheimer's diseases (AD) (46), confers risk of severe brain injury (47) and particularly boxers having this allele are more likely to develop chronic injury (48) hence it is speculated to be a 'risk allele'. However in contrast to these reports, e4 allele was not observed to be associated with increased risk of concussion and poor outcomes after mild brain injury in college athletes and children, respectively (49,50). While other studies have reported effect of ethnicity , age and sex on expression of APOE e4 allele on development of poorer outcomes after traumatic brain injury (51,52). Besides this, three variants in the promoter region of APOE -219G>T , -419A>T, -427T>C have been studied in the context of head injury (51). -219G>T has been found to increase the risk of concussion and AD in athletes with TT genotype as compared to GG genotype (51). Besides that, -219T augments the expression of e4 while -419T reduces the expression and presence and absence of these two variants have been linked with association of e4 with concussion (52,53).

Microtubule associated protein tau

The microtubule-associate protein tau is another important protein encoded by the *MAPT* gene that has been extensively associated with many neurodegenerative disorders (54). Higher levels of tau protein have been reported in amateur boxers following head bows (55) and concussed hockey player in a study conducted among Swedish professional hockey players (n=47) with these levels declining after appropriate rest and rehabilitation (56). However there are scarce reports of tau protein association with concussion and only Terrell et al (2008) reported a weak association between tau Ser53Pro and increased concussion risk (51).

Genes linked with tendinopathies

Another important risk factor associated with performance is risk of having muscle injuries or ten-

dinopathies which have been linked with genetic variants in collagen-encoding genes such as *COL1A1* and *COL5A1*, connective tissue wound repair gene *MMP3* and the *TNC* gene encoding tenascin C. Exonic SNPs in *TNC* have been linked to risk for failure in healing and recovery (57,58). Presence of multiple risk alleles in an individual potentially increase the risk of injury and delayed recovery (59).

Effect of single vs multiple genetic polymorphisms on sport phenotype

Sport performance is based on complex interactions of interconnected genes and their variants which are responsible for regulating the key performance indicators and shaping the overall sports phenotype. In this context, the polygenic model of inheritance becomes more suitable for the explanation of sports performance (60). For example, the presence of more alleles associated with aerobic metabolism would result in better response to aerobic training (61) while having more alleles associated with endurance will increase likelihood of becoming a successful endurance performer (62). Besides that, some of the polymorphisms may fail to create an impact on performance alone but the presence of other polymorphisms may result in enhancing their impact on phenotype via genetic interactions. This means that a combination of polymorphisms might have a significant effect on overall sports phenotype than single polymorphisms and they need to be taken into account to predict sports performance and training regime (62, 63). William and Folland (63) proposed the total score genotyping (TGS) for helping the assessment of the balance between selected PEPs and has proven to be a very sensitive and reliable tool in differentiating the endurance and power athletes (64,65) as well as in distinguishing the elite athletes from general population on the basis of their genetic profile (66). However the sensitivity and sensibility of the TGS is dependent upon type and number of PEPs included in the calculations necessitating careful selection of only consistent polymorphisms associated with a particular sports type for TGS calculation (67). The application of TGS however is limited by the fact that it gives same weight to all polymorphisms used (68).

Rare genetic variants

Unravelling the physiological mechanism by which genetic variants effect performance becomes essential in linking these variants to sports phenotype (69). In addition to this, rare genetic variants might result in sports excellence (70). For example, truncating mutations in myostatin gene (*MSTN*) result in enhancing sprinting (71). Similarly, a rare erythropoietin receptor gene (*EPOR)* variant has a significant effect on haematocrit and VO_2 max and an Olympic gold medallist in country skiing was found to be carrier of this variant (72). Identifying more rare genetic variants may help in prediction of multitalented and gifted athletes with exceptional performances. Nevertheless, this area is still poorly understood (72).

Our current knowledge related to PEPs is still scarce and more research in this area is required to fully understand the genetic interactions resulting in high level sports performance. Identification of new polymorphisms with significant consistent association with sports phenotype and replication of the association of existing PEPs across various ethnic groups and different environmental conditions would help to predict the sport performance of potential athletes thus helping in talent identification.

Genetic variants for talent identification

The overall peak performance of an individual depends upon the intrinsic ability to perform well and the trainability. However numerous articles published in the past two decades highlight the association of fitness and sport performance with autosomal, X-linked and mitochondrial genes and their polymorphic variants (73,74). Hence, attaining elite athlete status cannot be solely attributed to practice and hard work but also to the right genetic background (75). Genetic variations have been reported to influence every single aspect of elite performance such as trainability (76), post exercise recovery (77-80), risk of injury (81), skill acquisition (82), post exercise fatigue (83), psychological traits (84) and athletic development (85). Thus proving their potential in identification of elite athletes status.

Genetic variants related to endurance performance

Recent advances in sports genetic have led to the identifications of genetic variants with potential influence on key performance indicators in both endurance and power sports (Table 1). The major genetic variants linked with endurance athlete status are those which influence aerobic endurance capacity, muscular strength,

Table 1. Genetic polymorphisms associated with sport performance

Gene	Full name	Associated phenotypes	Polymorphism ID	References
ACE	Angiotensin I converting enzyme	I allele, endurance performance; D allele, power performance	rs4646994 (Alu I/D)	28,29,31,146
ACTN3	α-actinin-3	577Ter (T) allele, endurance performance; Arg577 (C) allele, power performance	rs1815739 C>T	28,39,91,147
ADRB2	β-2adrenoreceptor	16Arg (A) and Gln27 (C) alleles, endurance performance	rs1042713 G>A; rs1042714 C>G	92,93,94,146
BDKRB2	Bradykinin receptor B2	T allele, endurance performance	rs1799722 C>T	93,146
COL5A1	Collagen, type V, α1	CC genotype, protection from exercise-associated muscle cramps during an ultra-marathon; T allele, endurance performance	rs12722 C>T	95,96,146,148
CRP	C-reactive protein, pentraxin-related	A allele, endurance performance	rs1205 A>G	97,98,146
GABPB1	GA binding protein transcription factor, β subunit 1 (nuclear respiratory factor 2	G allele, endurance performance	rs7181866 A>G	99,146
PPARA	Peroxisome proliferator-activated receptor α	G allele, endurance performance; C allele, power performance	rs4253778 G>C	100,101,146
PPARGC1A	Peroxisome proliferator-activated receptor γ coactivator 1 α	G allele, endurance performance	rs8192678 G>A	102,103,146
VEGFA	Vascular endothelial growth factor A	C allele, endurance performance	rs2010963 G>C	104,105,146
ADRA2A	α-2A-adrenergic receptor	Central role in the regulation of systemic sympathetic activity and hence cardiovascular responses such as heart rate and blood pressure	*Dra* I identifies a restriction fragment length polymorphism in the 3_-untranslated region (6.7-/6.3-kb polymorphism	92
AMPD1	Adenosine monophosphate deaminase 1	GG homozygotes, elite power athlete status, quicker acceleration and sprint times	rs17602729 G>A	77,149
EPAS1	Endothelial PAS domain protein 1	AA genotype in rs1867785, underrepresented in sprint/power athletes; TT genotype in rs11689011, underrepresented in sprint/power athletes	rs1867785; rs11689011	124

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Table 1 ***(continued)*****.** Genetic polymorphisms associated with sport performance

Gene	Full name	Associated phenotypes	Polymorphism ID	References
NFATC4	Nuclear factor of activated T cell calcineurin-dependent 4	G allele, elite endurance athlete status	rs2229309 G>C	125
NOS3	Nitric oxide synthase 3	GG genotype, slower than the other genotypes	rs1799983 T>A>G	92,126
AGT	Angiotensinogen	235Thr (C) allele, power performance	rs699 T>C	106,107,146
IL6	Interleukin-6	G allele, power performance	rs1800795 C>G	108,109,146
TRHR	Thyrotropin- releasing hormone receptor	C allele, muscle mass	rs16892496 A>C	110,146
VDR	Vitamin D receptor	A allele, power performance	rs1544410 A>G	111,112,146
PPARGC1B	Peroxisome proliferator-activated receptor γ coactivator 1 α	C allele, power athlete status	rs10060424 T>A,C	114
PPARG	Peroxisome proliferator-activated receptor γ	G allele, short-term and very intense exertion with anaerobic energy production	rs1801282 C>G	115
HIF1A	Hypoxia-inducible factor 1α	T allele, higher frequency in weightlifters and power-orientated athletes	rs11549465 C>T	115,117,118
PTPRK	Protein tyrosine phosphatase receptor type K	C allele, sprint test performance	rs55743914 C>T	120
TERT	Telomerase reverse transcriptase	G allele, sprinters	rs33954691 G>A	120
RDH13	Retinol dehydrogenase 13	G allele, increased proportion of fast-twitch muscle fibres	rs4806637 A>G	120
CBLN2	Cerebellin 2 precursor	G allele, sprinters	rs8093502 C>T	120
CPNE5	Copine V	G allele, sprinters	rs3213537 C>T	120
CNTN4	contactin 4	A allele, overrepresented in football players	rs62247016 A>T	120
LINC00305**, *****LINC01924***	Long intergenic non-protein coding RNA 305, 1924	Functional role in development of atherosclerosis by inducing production of inflammatory cytokines in monocytes, by regulating apoptosis via miR-136	rs2850711 A>T	150
AGTR1	Angiotensin II receptor type 1	C allele, essential hypertension. A allele, downregulated by the miR-155	rs5186 A>C	151
MIR499A	MicroRNA 499a	GG genotype, myocardial infarction and ischemic stroke. The rs3746444 polymorphism disturbs regulation of blood pressure and anti-apoptotic effect in cardiomyocytes	rs3746444 A>G	152

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Table 1 ***(continued)*****.** Genetic polymorphisms associated with sport performance

Gene	Full name	Associated phenotypes	Polymorphism ID	References
MIR4513	MicroRNA 4513	Blood pressure, total lipids, total cholesterol, low-density lipoprotein cholesterol, blood glucose. TT genotype, coronary artery disease. T allele, decrease in Mir-4513	rs2168518 C>T	152
MIR149	MicroRNA 149	Coronary artery disease	rs2292832 T>C	152
MIR27A	MicroRNA 27a	C allele, increases in the expression of the miR with negative effect on adipogenesis. CC genotype, protective role against T2DM. G allele, increased risk of early cardiovascular autonomic neuropathy	rs895819 T>A,C,G	152
CREB1	CAMP responsive element binding protein 1	A allele, smaller reduction in heart rate during a submaximal exercise test following training; greater exercise-induced temperature increase	rs2253206 A>G,T	148
CPT2	Carnitine palmitoyltransferase 2	Minor alleles, CPT2 deficiency	rs1799821 G>A; rs1799822 A>G	149
PYGM	Muscle associated glycogen phosphorylase	Truncating variant, exercise intolerance, cramps and contractures during exercise and stressful situations	rs116987552 G>A	149
CNTF	Ciliary neurotrophic factor	GG genotype, athlete phenotype	rs1800169 G>A	147
ACVR1B	Activin A receptor type 1B	Dynamic knee flexion and extension, isometric strength	rs11612312 T>C; rs2854464 A>C,G	153
NGF	Nerve growth factor	CC genotype, more anxious females; TT genotype, more anxious males, less anxious females	rs6330 C>T	154
BDNF	Brain-derived neurotrophic factor	CC genotype, quicker sprinters than A allele carriers	rs6265 G>A	154
NGFR	Nerve growth factor receptor	Vagal autonomic dysregulation	rs2072446 C>T	154
MSTN	Myostatin	Peak power during muscle contractions	rs1805086 A>G	154,155
SCN9A	Sodium voltage-gated channel alpha subunit 9	AA genotype, increased perception of pain	rs1805086 A>G	154
COMT	Catechol-O-methyltransferase	A allele, higher dopamine levels; lower pain threshold; enhanced vulnerability to stress. G allele, lower dopamine levels; higher pain threshold; better stress resiliency	s4680 G>A	154

biomechanical efficiency, mental endurance and physical characters such as weight and height (86,87). Not only these genetic variants are heritable but differ between ethnicities and their effect are modified by environmental factors such as training and nutrition (88).

A recent review by Ahmetov et al has highlighted 93 endurance associated DNA variants (89) while a systematic research by William et al has identified 97 DNA variants associated with VO_2max/peak trainability (90). The key genetic variants involved in endurance performance and trainability are located in the following genes: *ACE, ACTN3, ADRB2, BDKRB2, COL5A1, CRP, GABPB1*, *PPARA*, *VEGFA, ADRA2A, AMPD1, EPAS1, NFATC4, NOS3, TFAM*. The functions and associated phenotypes of these genes are listed in Table 1.

Genetic variants related to power performance

On the other hand the current literature review revealed 69 genetic markers associated with power athlete status. Most genetic markers associated with power athlete status are linked with skeletal muscle structure and function, blood pressure control, modulation of oxygen uptake, inflammatory and repair reactions during and after exercise, regulators of energy metabolism and cellular homeostasis, factors that control gene expression and cellular signalling pathways (Table 1). The most important of these are *AGT*, *ACE, ACTN3, HIF1A*, *PPARA*, *PPARGC1A, PPARGC1B, PPARG*, *PTPRK, SEMA4A, TERT, RDH13,CBLN2, MORC4, CPNE5, CNTN4, TRHR, VDR, IL6*.

In addition to the genes listed in Table 1 there are some SNPs near *MORC4* that were reported to be linked with enhancing the expression of *RNF128* in nerves while the *C allele increases the expression of CLDN2* in thyroid tissue. These are important with respect to gene expression in skeletal muscles, nerves, blood and thyroid tissue and in skeletal muscle fibre composition and fast twitch muscle fibres (122,123). Another SNP rs12688220 near *MORC4*, was found to be associated with sprint performance, elite sprint athlete status, and increased proportion of fast twitch muscle fibres. However the mechanism through which this locus affect the sprint phenotype is poorly understood (120).

Performance prediction and talent identification based on genetic profiling

Although the association of most genetic variants with sports performance has weak scientific background, their presence in an individual either alone or in combination predisposes towards an increased chance of success in power or endurance performance (127). Nevertheless, it should be highlighted here that each individual polymorphism has only limited contribution to an elite athlete status and if considered alone may result in inadequate predicting of potential elite athlete phenotype (11, 127).Consequently genetic tests based on one or few genetic markers lack scientific backing for prescription of personalised exercise and sports training. Therefore considering a polygenic profile of various polymorphic variants encoding diversified products involved in wide variety of cellular processes and pathways becomes crucial for accurate talent identification (128). Besides that, the identification of large numbers of SNPs affecting a given trait and then combining them into a TGS model for that trait, would probably improve the predictive precision of genetic evidence (11).

Another important consideration in selecting the genetic markers for performance prediction is that rare genetic variants have a more powerful impact on sports phenotype as compared to common variants. One of the rare variants that conferred the winning performance to Finnish cross country skiing Champion Eero Mantyranta is the *EPOR* that resulted in an increased red blood cell production corresponding to elevated oxygen carrying capacity and aerobic endurance (72, 129). Another rare variant in lamin/AC (*LMNA*) gene was reported in one of the best Canadian sprint hurdler Priscilla Lopes-Schilep (130).

Although using rare genetic variants as markers for elite performance predictions sounds interesting and promising their low frequency make them hard to identify (11). Moreover to associate these variants to sports phenotypes would require studies with very large samples of unrelated individuals (11, 72). And finally there are ethical concerns as some of these variants might also predispose to disease states (129).

Commercial genetic testing

A study conducted by William et al in 2016 reports on the commercial direct to consumer (DTC) genetic testing. They surveyed 39 commercial testing companies and collected information regarding genetic variants tested by them. Their results indicated that only 18 companies had provided details of genetic variants they test. *ACTN3* was found to be the most frequently analysed variant with 88.8% of the 18 companies using it for commercial testing followed by *ACE* (61.1%), *PPARGC1A* (50%), *ADRB2* (44.4%), *COL5A1*, *VDR* (38.9%), *COL1A1*, *VEGF* (33.3%), *AGT*, *AMPD1*, *NOS3* (27.7%), *MMP3*, *PPARD*, *TRHR*, *CRP* (22.2%). Total number of genetic variants tested by these companies was 54 and only a few companies (7) provided a polygenic profile ranging between 14-27 genetic variants. 11 companies with exception of 2 companies which were providing test for single gene variant rest were conducting tests for 2-9 genetic variants (127).

Genetic performance tests pros and cons

Genetic plays a critical role in development of sports phenotype and exercise response. However to get positive benefits, training regimes and healthy lifestyle habits are of utmost importance. In other words, genetic coupled with a fitness and training regime can lead to development of an elite performance phenotype. Consequently, one of the most interesting application of sports genetic is development of tests for predicting performance and devise training regime. Furthermore, the potential for genetic testing to predict injury predisposition, may help in ensuring health and safety of athletes during sports training.

An excellent example of this is one of the Australian Rugby team which claimed that it has utilized genetic testing to develop training programs for its team members to gain a competitive edge over other teams. The team got tested 18 of its 24 players for 11 exercise-related genes (140). Subsequently their training programs were redesigned according to their genetic profile. In addition to this some professional sports teams are using the genetic test results for direct training recommendations (127).

However it is important to consider that studies identifying these gene linkages with sports performance have been conducted at population level and therefore they indicated the effect of these genetic variants on a study population while the effect of a particular genetic variant may differ considerably when seen in perspective of a single individual. Moreover, neither currently nor in future there is a chance of having a single gene variant that can conclusively provide sufficient information for an overall sports performance. Therefore genetic profiling to identify many genetic variants can help predict a world class talent and that can be useful if proper diet, nutrition and training regimes and positive environment are provided to develop the desired phenotype. It is worth mentioning here that, although the interest in commercial genetic testing is increasing, there is scarcity of evidence supporting notion of exercise prescription and talent identification. Consequently, it is far too realistic to claim the prediction of next generation of sports champions (60-64). Similarly, recommendation of target specific training protocols for power and endurance performance based on genotype or polygenic profile have insufficient evidence to guarantee their authenticity at the present (141). However some of the commercial genetic testing companies prescribe training regimes based on algorithmic approaches described in peer-reviewed research (142). Even though these tests might provide an insight into individual responses to training and exercise based on the genetic profiles these lack scientific backing unless improved methodologies with much larger sample sizes are used (141, 142).

There should be a standardized procedure of categorizing individuals as endurance or power in order to remove potential bias in replicating the studies (143).

Ethical concerns related to genetic testing for sports

Genetic testing in sports can raise several ethical concerns related to basic human rights of safety, privacy and secrecy of information. Besides that, the consequence of genetic tests specifically in children that aspire to become athletes can have several negative impacts such as depression and psychological problems in case the sports related genotype is not identi-

fied. Furthermore, most coaches, parents and athletes themselves do not have enough scientific background to understands the limitations and implications of results and this raises the questions that who should actually be allowed to ask for a test? (127)

In addition to that, genetic testing of athletes has a potential to be misused by commercial sports companies with preferences being given to some athletes over other thus violating basic human rights. Therefore the research in human sports and exercise genetics is also subjected to rigorous ethical screening by the ethical review committees as per the Helsinki declaration (World Medical Association, 2008). This ethical review process minimises the ethical problems arising from genetic research and their future applications (127,143-145).

Conclusion

Traditionally, sports talent identification is based on physical and physiological characteristics and performance in a specific sports discipline. However, inclusion of genetic tests in talent hunt would revolutionize the field of sport. Genetic tests to elucidate the inherent capabilities of youth with respect to sport performance will not only help them in selecting the right sports career but also the exercise and training regime that would complement their genetic background. Early detection of potential traits of practical utility will help in devising training plans during growth and development, thus enhancing the capabilities and skills for attainment of peak performance. Current evidence suggests that a favourable genetic profile, when combined with the appropriate training, is advantageous, if not critical for the achievement of elite athletic status. However, though few genes have now been repeatedly associated with elite athletic performance, these associations are not strong enough to be predictive and the use of genetic testing of these variants in talent selection is premature. Nevertheless, further molecular level research is required to strengthen our understanding of sports genetics, however this is possible only through a shift in the approach of policy makers followed by substantial funding that would lead to achieving excellence in sports.

Conflict of interest: Each author declares that he or she has no commercial associations (e.g. consultancies, stock ownership, equity interest, patent/licensing arrangement etc.) that might pose a conflict of interest in connection with the submitted article

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