Athletic performance and risk of injury: can genes explain all?

Ross Tucker*, PhD; *Malcolm Collins*†, PhD

UCT/MRC Research Unit for Exercise Science and Sports Medicine
**Department of Human Biology - Faculty of Health Sciences - University of Cape Town - Cape Town*
†South African Medical Research Council - Cape Town - SOUTH AFRICA

Sporting success is the result of the combination of innumerable genetic and environmental factors, and there is no single path to becoming a champion athlete. Susceptibility to injuries is also a multifactorial phenotype and is a less acknowledged contributor in determining elite athletic ability. The relative importance of deliberate practice, other environmental factors, and genetic factors in molding champions is a constant area of debate. We review two models, the "Practice Sufficiency" and "Genetic Ceiling" models that explain expert performance development and injury risk. We conclude that although the deliberate training and other environmental factors are critical for achieving elite performance, the "Practice Sufficiency Model" does not adequately explain performance. The "Genetic Ceiling Model," on the other hand, acknowledges both nurture and nature and is a more accurate theory.

Keywords: elite athlete; nature; nurture; musculoskeletal; polymorphism; training
Address for correspondence:
Prof Malcolm Collins, UCT/MRC Research Unit for Exercise Science and Sports Medicine, PO Box 115, Newlands, 7725, South Africa
(e-mail: malcolm.collins@uct.ac.za)
Dialogues Cardiovasc Med. 2012;17:31-39

Sporting success is the result of the combination of innumerable factors, and there is no single path to becoming a champion athlete *(Figure 1, page 32).* The factors predicting sporting success have been widely studied, and include proposed genetic predisposition and innate physiological advantages for sporting performance[1,2] exposure to the right coaching and competition environments, the age at which the young athlete begins deliberate practice,[3] and the number of hours of deliberate practice accumulated.[4,5] Throughout the process of developing a young athlete, developmental, psychological, and motivational factors are crucial,[6] and require the correct training volumes and methods at different stages of development. In addition, young athletes must avoid injury and burnout as a result of training,[7] and only when the optimal combination of all these factors exists will a champion athlete be produced *(Figure 1).*

While few would dispute the complexity of sporting success, there has recently been renewed debate over two of the above factors—the relative importance of genetic factors (nature) and the theory of accumulated hours of deliberate practice (part of nurture). Broadly, two models exist to explain these factors in expert performance development.

In the first, which we have termed the "Genetic Ceiling Model," performance is ultimately determined by the presence (or absence) of specific genetic sequence variants.

These genetic variants would predispose an individual to success in certain sports. Conversely, the absence of these variants would limit an individual's sporting ability. This model holds that innate (genetic) factors are responsible for determining the level of performance reached in a given task, effectively establishing a ceiling beyond which an athlete cannot improve, regardless of training or practice.[2] In fact, many biochemical, physiological, and other intrinsic characteristics of an individual are partly determined by their genetic makeup *(Figure 1).*[8-11]

The alternative theory, developed and proposed by Ericsson et al,[12-14] which we shall term the "Practice Sufficiency Model," proposes that expert performance is the result of accumulating many hours of deliberate practice. This theory, recently popularized by books such as Malcolm Gladwell's *Outliers,* Daniel Coyle's *Talent Code,* and Matthew Syed's *Bounce: How Champions are Made,* holds that deliberate practice is sufficient to attain expert performance levels in any sport. By extension, it predicts that interindividual differences will disappear with practice, so that every individual can at-

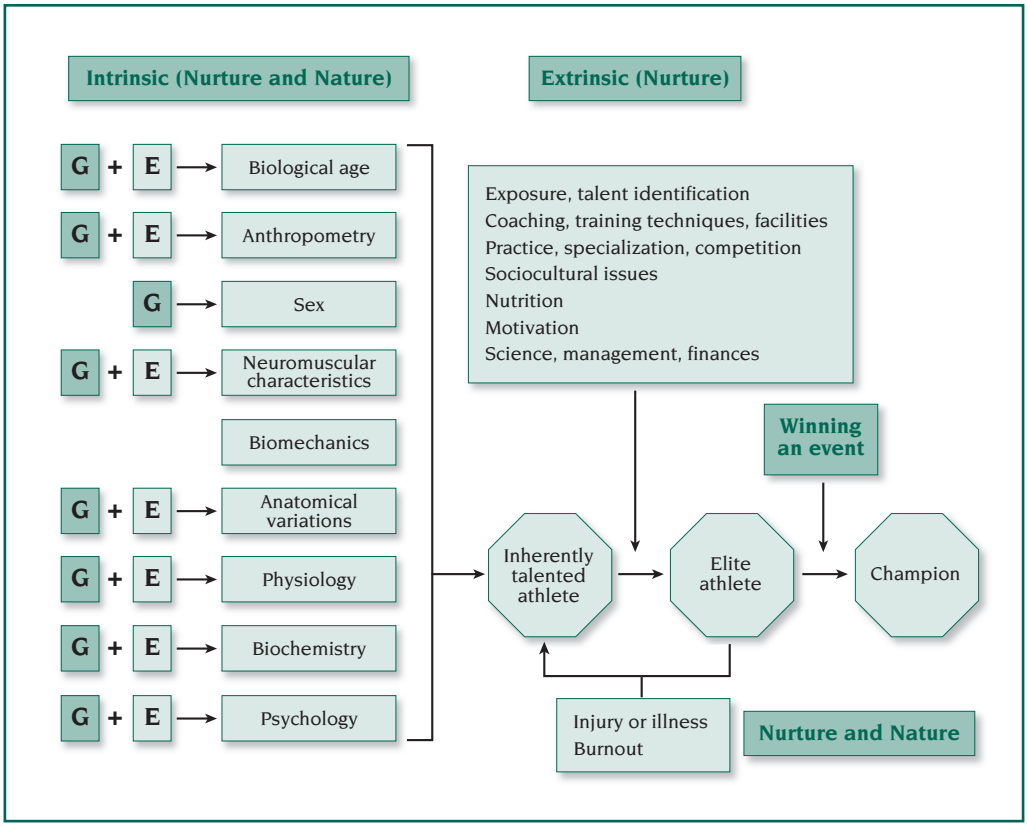

Figure 1. A diagram illustrating the complex relationship between intrinsic and extrinsic factors that determine elite athleticism. *This is not a complete list of all the factors, but rather illustrates the complexity of the phenotype. Many of the individual intrinsic risk factors are also in their own right multifactorial phenotypes determined by, to a lesser or greater extent, both genetic (nature, G) and environmental (nurture, E) factors.*

tain performance mastery given sufficient training time. It is worth emphasizing from the outset that any model that adopts an exclusive or extremist approach to explaining sporting performance is likely to be incorrect, given the above-mentioned complexity in sporting performance. That is, neither extreme argument (that genetics or deliberate practice are solely responsible for sporting performance) is likely to be accurate. However, in order to better understand the relative importance of these two views, each must be evaluated based on its singular prediction that either genes or deliberate practice are sufficient for elite sporting performance.

The aim of the present review is to critically evaluate each of the above models in turn, with the purpose of characterizing the relative importance of an athlete's genetic profile as crucial to sporting success.

THE GENETIC CEILING MODEL

The "Genetic Ceiling Model" traces its origins back to Sir Francis Galton (1822 to 1911) who, having identified that body size and height had some heritable component, wrote the following in 1869:

> Now, if this be the case with stature, then it will be true too as regards every other physical feature—as circumference of head, size of brain, weight of grey matter, number of brain fibers…and thence, a step on which no physiologist will hesitate, as regards mental capacity.[2]

Having extended his heritability model to mental capacity, Galton proposed that improvements in performance could in fact be achieved with training, and that they may occur rapidly at first, but would reach a ceiling for each individual such that maximal performance capacity was ultimately determined by innate, heritable factors. Galton's model is thus not one that discounts the importance of training, but rather one that says that performance improvements with training become increasingly smaller until an upper limit of performance is reached (as described by Ericsson[12]), irrespective of training. We have thus termed this the "Genetic Ceiling Model," since it holds that performance (and response to training) is ultimately determined by one's genetic profile.

It is worth nothing that at the time of his writing, heritability was very poorly understood—it preceded the discovery of DNA by almost 100 years, for example. Also of interest is that Galton did not apply this model to sporting performance. Indeed, he could not, since in 1869, organized sport was in its infancy, the domain of the upper class male society only, and no research of any kind existed to support arguments

around sporting performance. However, his theory of a ceiling determined by genetics became established and widely applied, and as argued by Ericsson,[12] is consistent with many contemporary theories for skill acquisition.

THE PRACTICE SUFFICIENCY MODEL

The opposing view, which we term the "Practice Sufficiency Model," argues that innate or genetic factors are not required to explain the development of elite performance. This model, developed by Ericsson,[12,15] holds that performance is the result of the accumulation of sufficiently large volumes of deliberate practice. He writes, for example, that:

> distinctive characteristics of exceptional performers are the result of adaptations to extended and intense practice activities that selectively activate dormant genes that are contained within all healthy individuals' DNA.[12]

In this explanation, deliberate practice (training) is sufficient for any individual to attain expert performance in any sport, and the only constraint on the development and achievement of expert performance is the individual's engagement in deliberate practice and the quality of the available training resources.[12]

Evidence for the "practice sufficiency model"

To date, we are unaware of any studies that have systematically examined whether training does indeed reduce interindividual differences to zero, or that practice is sufficient to achieve elite level performance in sport. This is unsurprising, however, given the complexity of evaluating skill levels in many sports, and the impossibility of accounting for numerous confounding variables that may have influenced skill acquisition from birth to adulthood. However, there are studies on the development of expert performance in activities and sports where skill is more easily quantified, such as violin playing, chess, and darts. Perhaps the most famous such study is that of Ericsson et al on violin players.[15] This study found that the very best players had accumulated over 10 000 hours of deliberate, solitary training time by the age of 20,[13,15] giving rise to the now popularized "10 000 hour rule" for performance.

This study, foundational to the "Practice Sufficiency Model," is worth more detailed analysis. One of the key observations is that the study did not report statistical measures of variance or ranges of training hours within each ability group. This may be problematic, since it is possible that there was significant overlap in the actual training hours between the groups.

This is crucial, because inherent in the "Practice Sufficiency Model" are the following three requirements: (i) variance in performance must be explained entirely by accumulated deliberate practice time; (ii) exceptional performance cannot occur in the absence of high volumes of accumulated training; and (iii) individuals who accumulate high volumes of deliberate practice must always achieve expert or elite levels in that activity. Indeed, Ericsson et al[12] have written that:

> the development of expert performance will be primarily constrained by individuals' engagement in deliberate practice and the quality of the available training resources.

This implies that failure to reach the "best expert" group must be associated with reduced training time and/or access to resources. If this is not the case, then other factors must constrain performance ability, or enhance it in those cases where individuals reach performance mastery despite performing less accumulated training.

This variance was however evident in a study on the impact of deliberate practice on chess performance,[16] in which 104 players, ranging from those without an international rating to grandmasters (the highest level of chess performance) were evaluated. In agreement with the main finding of the violin study,[15] players who reached the master level had accumulated an average of 11 053±5538 hours of total practice.[16] The coefficient of variation (CV) of 50% suggests how variable this training time was. Specifically, it was found that a range greater than 20 000 hours existed, with the fastest player to attain master level doing so with only 3016 hours, compared with another player who did so with 23 608 hours. This is an eightfold difference. The authors further report that some players had not reached master level despite more than 25 000 hours of practice. Clearly, this study suggests that high training volumes are essential, but not sufficient, for the attainment of high performance levels.

The same appears to be true of darts performance.[17] Eighteen professional and 18 amateur dart players completed questionnaires to ascertain, among many factors, hours of deliberate practice. The average number of accumulated hours of deliberate practice after 15 years of playing was 12 839±7780 for professional men, compared with 3270±2916 hours for amateur men. Again, the CV was high, 61% and 89% for professionals and amateurs, respectively. Since ranges were not reported, it is unclear whether there was overlap between the two groups, though given the large standard deviations, this would seem likely, in a repeat

of the finding with chess. It is not surprising that large differences in practice time exist when comparing amateurs and professionals, or best expert performers and least accomplished performers. This is analogous to the comparison of a physiological determinant of performance such as VO_{2max} between elite athletes and recreational athletes or sedentary individuals. Between these groups, large differences in VO_{2max} exist. However, within each group, VO_{2max} becomes a poor predictor of performance, because numerous other factors, such as efficiency or economy, metabolic capacity, and possible neural factors play a role.[18]

Similarly, finding large differences in training volume between professional and amateur darts players and best expert and least accomplished violinists does not necessarily indicate a sufficient role for training time in performance.

In fact, this type of difference may even be explainable as a result of innate differences, which manifest very early on, creating a behavioral bias for further practice. That is, it may be that children who show early promise in an activity as a result of innate factors are encouraged and supported to train more. Indeed, Ericsson[13] describes a phenomenon observed by Bloom where young children are introduced to an activity in a playful manner, but "as soon as they show promise compared to peers in the neighborhood, their parents help them seek out a teacher and initiate regular deliberate practice." The key question is what factors determine the early display of promise in an activity? It may be that when observing an elite group who has already obtained expert status in an activity, one is actually studying the effects of deliberate practice within a group that is already selected from the general population as a result of innate factors. Clearly, practice is important for elite performance, but practice alone cannot be sufficient for performance in any of the previously described studies.[15-17]

Sporting examples

So far, we have focused exclusively on activities where performances are solely determined by motor skill, cognitive ability, and repetition. These activities are not "physiologically constrained" or determined in the way that sports such as football, running, or weight lifting may be. In these sports, it is much more difficult to objectively evaluate performance. It is equally difficult to characterize training as being deliberate or simple play, particularly retrospectively. However, data do exist regarding training time and years of participation in sport before attainment of an elite level. These data have revealed that elite sports people rarely complete 10 000 hours of deliberate training, but that they often participate for approximately 13 years before attaining Olympic or equivalent level performance.[19,20] Wrestlers accumulated only 6000 hours of training in 10 years,[19] while international level footballers accumulated 4000 hours.[20]

It is important to note that elite performance at the adult level is a combination of both ability and physical development. As a result, very few teenagers reach senior elite levels in sports that favor a peak after the age of 20. The 13-year period is thus not necessarily a period of deliberate training, but may be a "waiting period" to allow physical development and maturity to peak to enable elite adult competition. Indeed, there is evidence that some sports people succeed within a few years of introduction, provided they commence as adults. In one study, 69% of elite performers had been playing their sport for less than 4 years.[21] However, they had all been exposed to a wide range of other sports before selecting their specialization. This indicates that talent transfer is possible, and has driven the talent ID programs of Olympic programs in Australia and the United Kingdom, where champion athletes have been discovered in skeleton, rowing, and netball, where one player made the national team with 6 years and only 600 hours of deliberate practice![22,23]

Conclusion

The studies on deliberate practice have clearly established that accumulated practice time is a key differentiator between elite and sub-elite performers. There is no question that in order to achieve sporting success or to master a skill, high volumes of training are required. However, these studies have not established that innate differences, which may have been present from very early on in childhood, are not themselves responsible for influencing behavioral differences that ultimately result in disparities in accumulated training time. It seems clear that 10 000 hours of training are not required by all athletes, and that some will succeed with substantially less training. Equally, some athletes will fail despite much more deliberate training, though these athletes are rarely documented, an inherent bias leading to an incomplete picture of success and failure in elite sporting performance. The "Practice Sufficiency Model" thus cannot completely explain either success or failure in sporting activities, or even in activities such as chess, violin-playing, and darts, where the risk of injury and the importance of physiology may be less important.

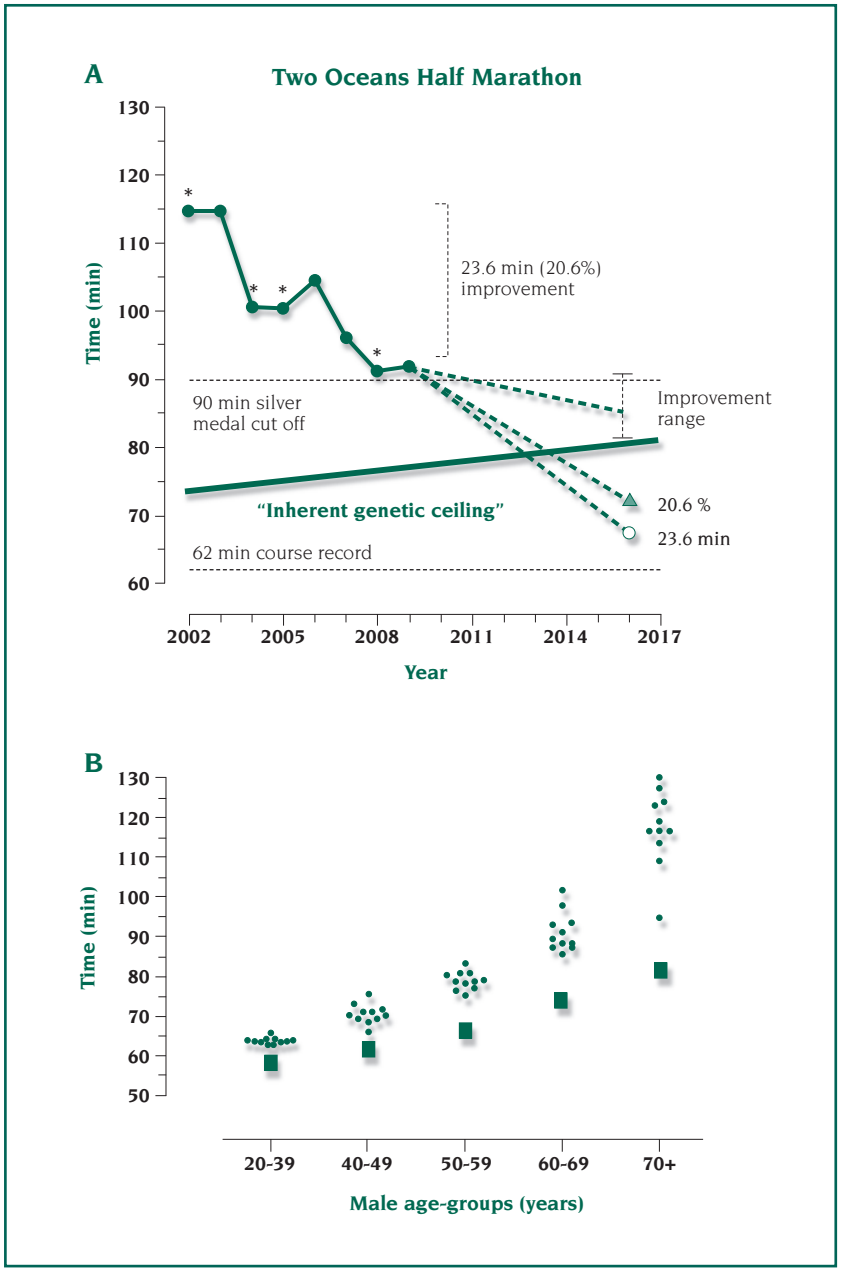

Figure 2. Half-marathon performance of a male recreational athlete over 7 consecutive years.

(A) *Case study examining the 21.1-km road race (Two Oceans Half Marathon, Cape Town, South Africa) performance of a male recreational athlete over 7 consecutive years from 2002 (36 years old) until 2009. The solid circles and solid lines plot his actual performance during the 8 consecutive races. Personal best times for the half-marathon are indicated with an asterisks. The horizontal dotted lines indicate the 90-minute silver medal cutoff time limit and the 62.9-min course record. The bold solid line represents a hypothetical personal inherent performance "genetic ceiling" for the athlete. If only nurture determined performance then this athlete would be able to improve by an additional 23.6 min (clear circle) or 20.6% (clear triangle). Alternatively, in spite of continued nurture this athlete will eventually reach an "inherent ceiling" preventing any further improvement. His range for potential improvement in performance is the difference between his currently ability and his personal "inherent ceiling" (Improvement Range).*

(B). *The male age-group winning times during the Two Oceans Half Marathon from inception of the race in 2001 until 2011 (solid circles) and current age-group half-marathon world records. Based on data from references 24 and 25.*

NATURE AND NURTURE: A PRACTICAL EXAMPLE

Performance is far more complex than being singly constrained by engagement in deliberate practice. As a result, the "Practice Sufficiency Model" is itself insufficient to explain performance, and so we turn to genetic factors to describe how some of the variances in performance may be accounted for.

It should be noted that elite athletes might not be the ideal population to use to illustrate the genetic contribution in performance, since to reach this level they have already been selected out of the general population and genetic profiles may not therefore play a large role at this level. Thus, within this relatively narrow band of performers, there may be little genetic heterogeneity, and nurture may then become the most important determining factor between first and second place at the Olympics and World championships.

The following case study examines the half-marathon performance of a male recreational athlete over 7 consecutive years. In 2002, at 36 years old and after about 16 months of training, he participated in his first 21.1-km road race. During the following years he specifically trained for and, with the exception of the 2006 event, raced in the same event, running three personal best times. Of note, there was a 23.6-min (20.6%) improvement in his performance during the 8 races *(Figure 2A)*.[24,25] If there were no interindividual variation in the inherent limit to athletic ability, then it would be possible for him to continue improving merely by implementing the correct training, nutrition, and motivation strategies (which had been sufficient up to this point). A further 23.6-min improvement would mean that he

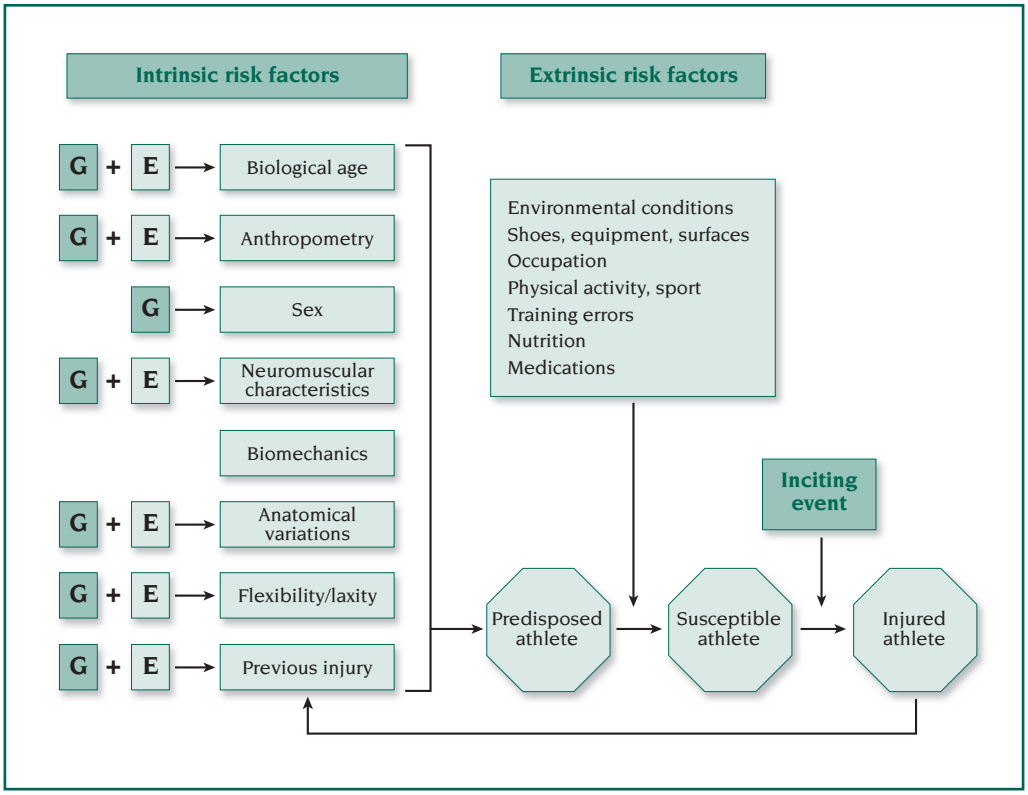

Figure 3. A diagram illustrating the complex relationship between the "traditional" intrinsic and extrinsic risk factors, as well as the role of the inciting event in the etiology of sporting injuries.
Many of the individual intrinsic risk factors are also in their own right multifactorial phenotypes determined by, to a lesser or greater extent, both genetic (nature, G) and environmental (nurture, E) factors. For acute injuries the inciting event will be the macrotraumatic event that cause the injury, while the inciting event for a chronic injury will be the point in time when the volume of accumulated micotraumatic damage to the tissue becomes symptomatic.

After reference 33:
Meeuwisse WH. Clin J Sport Med. *1994;4:166-170.*
© 1994, Raven Press, Ltd.

could complete the race in 67.4 min, 4.5-min slower than the current course record. Since this athlete would be in his early 50s in the next 8-year period, this improvement will bring him within 0.7 min of the 50-55 male half-marathon world record, which one assumes is close to the inherent ability for male athletes *(Figure 2B).*[24,25] The logical conclusion of this example is that if only nurture determined performance then this athlete would be able to at least set an age-group half-marathon world record with the correct coaching.

Alternatively, although there was, at least in this athlete, a large scope for improving performance due to training and motivation (nurture), it is more likely that in spite of continued nurture this athlete will eventually reach an "inherent ceiling" preventing any further improvement. His range for potential improvement in performance is the difference between his current ability and his personal "inherent ceiling" and not between his current ability and the world record.

GENETIC CONTRIBUTION TO SUPERIOR ATHLETIC PERFORMANCE

It is generally accepted by the scientific community that both environmental and genetic factors determine an individual's athletic ability.[11,26] The role of genetics in elite athletic performance has been extensively reviewed within the scientific literature.[27] We will therefore only highlight some key milestones in the development of this area of research.

The first attempts to identify genetic markers in phenotypes believed to be associated with athletic performance investigated red blood cell and HLA antigens and as well as isoforms of skeletal muscle enzymes in Olympic athletes during the 1970s.[28] These initial studies were followed by an era of genetic epidemiological studies designed to determine the heritability of various physiological traits believed to contribute to performance.[11,29] Large ranges of heritability estimates have been reported in these studies. The method of linkage analysis studies, which follow the inheritance pattern of a specific phenotype in families, has also been successfully used to identify genes that contribute to performance-related phenotypes in a few studies.[11]

Since 1998, genetic association studies is the predominant method being used to identify the many genetic variants located throughout the human genome that may be associated with performance and performance-related phenotypes.[27] Recently, Bouchard et al.[30] identified 21 single nucleotide polymorphisms, which accounted for 49%

of the variance in VO_{2max} trainability following a 20-week standardized training program in 473 white sedentary adults using a genome-wide association study (GWAS).

GENETIC CONTRIBUTION TO INJURIES

Athletes are at increased risk of acute and chronic musculoskeletal tissue injuries as a result of training and/or competition.[31] Predisposition to muscle, tendon, ligament, and/or other injuries can negatively impact an athlete's ability to train optimally and perform during competition, ultimately ending their competitive careers or preventing them from reaching their full potential. Multiple intrinsic and extrinsic risk factors are implicated in the etiology of these injuries.[32] Like elite athleticism, acute and chronic musculoskeletal tissue injuries are multifactorial phenotypes, determined by a poorly understood interaction of multiple factors *(Figure 3).*[33] Typical injury risk models include multiple intrinsic risk factors *(Figure 3),*[32,33] which individually are also in their own right multifactorial phenotypes determined by, to a lesser or greater extent, both genetic (nature) and environmental (nurture) factors.[8-11] Many are polygenic traits, with each individual gene having a small effect on the phenotype, but added together having a significant contribution.[9] In addition, these factors do not necessarily determine risk independently of each other. For example, both sex and age are also common intrinsic factors for flexibility.[34]

Gene	Polymorphism	Location	Injury	References
Collagens and glycoproteins				
COL1A1	rs1800012	Intron 1, functional Sp1-binding site polymorphism, G/T	Cruciate ligament ruptures Shoulder dislocations Achilles tendon ruptures (?)	32
COL5A1	rs12722	Exon 66 (3'-UTR), C/T	Achilles tendinopathy Female ACL rupture	32
	rs240736	Exon 66 (3'-UTR), C/A or (3'-UTR Haplotype)	Achilles tendinopathy	
COL12A1	rs12722	Exon 29, C/T, T1738I	Achilles tendon ruptures (?)	32
	rs970547	Exon 65, A/G, S3058G	Female ACL rupture Achilles tendon ruptures (?)	
TNC	-	Intron 17, GT dinucleotide repeat polymorphism	Achilles tendinopathy Achilles tendon ruptures (?)	32
ECM enzymes				
MMP3	rs679620 rs591058 rs650108 (rs679620 + rs591058 + rs650108)	Exon 2, G/A, E45K Intron 4, T/C Intron 8, G/A *MMP3* Gene haplotype	Achilles tendinopathy	32
MMP10 *MMP1, MMP3, MMP12*	rs486055 + rs1799750 + rs679620 + rs2276109	*MMP* Gene cluster haplotype	ACL rupture	35
Signaling molecules				
GDF5	rs143383	Promoter, T/C functional	Achilles tendinopathy Achilles tendon ruptures (?)	36
IL-1β IL-1RN IL-6	rs1143627, rs16944 rs2234663 rs1800795	IL signaling pathway	Achilles tendinopathy	37

Table I. Sequence variants (polymorphisms) within genes encoding for collagens, glycoproteins, extracellular matrix (ECM) enzymes and signaling molecules associated with soft tissue injuries.

Abbreviations: ?, suggested association bases on observations in small sample sizes. ACL, anterior cruciate ligament ; IL, interleukin ; MMP, matrix metalloproteinase ; TNC, tenascin C.

Recently, specific genetic sequence variants have also been identified as intrinsic risk factors for some injuries *(Table I, page 37).* These genes encode: (i) structural components of connective tissue (collagens and glycoproteins); (ii) extracellular matrix (ECM) proteinases (MMPs); and (iii) cytokines and growth factors. Interestingly, mutations within *COL5A1* and *COL1A1* cause Ehlers-Danlos syndrome and osteogenesis imperfecta, respectively.[32,35-37] In addition, common polymorphisms within these genes are associated with milder musculoskeletal soft tissue injuries *(Table I).*

In support of this, common polymorphisms within genes are associated with interindividual variation in stature. The tall or short stature syndromes are caused by mutations within the same genes.[9]

None of the genetic risk factors or any of the other intrinsic risk factors cause injuries. They merely modulate or contribute to the risk for these injuries. Predisposed athletes need to be exposed to appropriate extrinsic factors and an inciting event before they are injured *(Figure 2).*[24,25] For acute injuries, the inciting event will be the macrotraumatic event that causes the injury, while the inciting event for a chronic injury will be the point in time when the volume of accumulated micotraumatic damage to the tissue becomes symptomatic.

CONCLUSION

The 0.1% difference in the sequence of human DNA results in visible and measurable interindividual differences (ie, biological variation). Athletes are therefore not identical. There are differences in structures and functions of biological systems, and the response of these systems to training and loading are therefore not identical. In addition, the response of injured tissue to healing and treatment modalities is not identical.

Similarly, there is also a large interindividual ability in athletic performance. An individual athlete's limits (ceiling) in performance, adaptation to training, injury, healing, and response to treatment modalities is predominately determined genetically. Deliberate training and other environmental factors (nurture) play a critical role in enabling an athlete to reach and maintain their performance potential. Unfortunately, both nature and nurture are also involved in the etiology of injuries, which could eventually prevent athletes from reaching or maintaining their true performance potential.

In summary, we have investigated two models that explain expert performance development and injury risk. Although deliberate training and other environmental factors are important in reaching elite performance, the "Practice Sufficiency Model" cannot adequately explain performance. The "Genetic Ceiling Model," on the other hand, acknowledges both nurture and nature as well as the impact that nature (genetics) can have on the athlete's ability to train through injury (nurture), and is a probably more complete theory.

REFERENCES

1. Myburgh KH.
What makes an endurance athlete world-class? Not simply a physiological conundrum.
Comp Biochem Physiol A Mol Integr Physiol. 2003:136:171-190.

2. Galton F.
Hereditary Genius: An Inquiry into Its Laws and Consequences (Originally published in 1869). London, UK: Julian Friedman Publishers. 1869/1979.

3. Moesch K, Elbe AM, Hauge ML, Wikman JM.
Late specialization: the key to success in centimeters, grams, or seconds (cgs) sports.
Scand J Med Sci Sports. 2011;21: e282-e290.

4. Cumming J, Hall C, Starkes JL.
Deliberate imagery practice: the reliability of using a retrospective recall methodology.
Res Q Exerc Sport. 2005:76:306-314.

5. Baker J, Côté J, Deakin J.
Cognitive characteristics of expert, middle of the peak, and back of the pack ultra-endurance triathletes.
Sport Exerc. 2005:6:551-558.

6. Baker J, Côté J, Deakin J.
Patterns of early involvement in expert and nonexpert masters triathletes.
Res Q Exerc Sport. 2006:77:401-407.

7. Côté J, Baker J, Abernethy B.
Practice and play in the development of sport expertise. In: Eklund RC, Tenenbaum G, eds.
Handbook of Sport Psychology. New Jersey, NJ: John Wiley & Sons; 2007:184-202.

8. Wheeler HE, Kim SK.
Genetics and genomics of human ageing.
Phil Trans R Soc B. 2011:366:43-50.

9. Lettre G.
Genetic regulation of adult stature.
Curr Opin Pediatr. 2009:21:515-522.

10. Maes HH, Beunen GP, Vlietinck RF, et al.
Inheritance of physical fitness in 10-yr-old twins and their parents.
Med Sci Sports Exerc. 1996:28:1479-1491.

11. Macarthur DG, North KN.
Genes and human elite athletic performance.
Hum Genet. 2005:116:331-339.

12. Ericsson KA, Nandagopal K, Roring RW.
Toward a science of exceptional achievement: attaining superior performance through deliberate practice.
Ann N Y Acad Sci. 2009:1172:199-217.

13. Ericsson KA.
The influence of experience and deliberate practice on the development of superior expert performance. In: Ericsson KA, Charness N, Feltovich P, Hoffman RR, eds.
Cambridge Handbook of Expertise and Expert Performance. Cambridge University Press. Cambridge, UK 2006:685-706.

14. Ericsson KA, Krampe RT, Tesch-Romer C.
The role of deliberate practice in the acquisition of expert performance.
Psychol Rev. 1993:100:363-406.

15. Ericsson KA, Krampe RT, Heizmann S.
Can we create gifted people?
Ciba Found Symp. 1993:178:222-231.

16. Campitelli G, Gobet F.
The role of practice in chess: a longitudinal study.
Learn Individ Differ. 2007;17.

17. Dufft LJ, Baluch B, Ericsson KA.
Dart performance as a function of facets of practice amongst professional and amateur men and women players.
Int J Sport Psychol. 2004:35:232-245.

18. Lucía A, Hoyos J, Pérez M, Santalla A, Chicharro JL.
Inverse relationship between VO_{2max} and economy/efficiency in world-class cyclists.
Med Sci Sports Exerc. 2002:34:2079-2084.

19. Hodges NJ, Starkes JL.
Wrestling with the nature of expertise: a sport specific test of Ericsson, Krampe and Tesch-Römer's (1993) theory of "deliberate practice".
Int J Sport Psychol. 1996:27:400-424.

20. Helsen WF, Starkes JL, Hodges NJ.
Team sports and the theory of deliberate practice.
J Sport Exerc Psychol. 1998:20:12-34.

21. Oldenziel K, Gulbin JP, Gagne F.
How do elite athletes develop? A look through the rear-view mirror.
Australian Sports Commission, Canberra. 2007. http://cev.org.br/biblioteca/factors-affecting-the-rate-of-athlete-evelopment-from-novice-to-senior-elite-how-applicable-is-the-10-year-rule.

22. Burgess DJ, Naughton GA.
Talent development in adolescent team sports: a review.
Int J Sports Physiol Perform. 2010:5: 103-116.

23. Baker J, Côté J, Abernethy B.
Sport-specific practice and the development of expert decision- making in team ball sports.
J Appl Sport Pschychol. 2003:15:12-25.

24. *www.twooceansmarathon.org.za.* (accessed during April 2012).

25. *www.mastersathletics.net.* (accessed during April 2012).

26. Brutsaert TD, Parra EJ.
Nature versus nurture in determining athletic ability.
Med Sport Sci. 2009:54:11-27.

27. Ahmetov II, Rogozkin VA.
Genes, athlete status and training—an overview.
Med Sport Sci. 2009:54:43-71.

28. Bouchard C, Dionne FT, Simoneau JA, Boulay MR.
Genetics of aerobic and anaerobic performances.
Exerc Sport Sci Rev. 1992:20:27-58.

29. Peeters MW, Thomis MAI, Beunen GP, Malina RM.
Genetics and sports: an overview of the pre-molecular biology era.
Med Sport Sci. 2009:54:28-42.

30. Bouchard C, Sarzynski MA, Rice TK, et al.
Genomic predictors of maximal oxygen uptake response to standardized exercise training programs.
J Appl Physiol. 2010;110:1160-1170.

31. Ljungqvist A, Schwellnus MP, Bachl N, et al.
International Olympic Committee consensus statement: molecular basis of connective tissue and muscle injuries in sport.
Clin Sports Med. 2008:27:231-239.

32. Collins M, Raleigh SM.
Genetic risk factors for musculoskeletal soft tissue injuries.
Med Sport Sci. 2009:54:136-149.

33. Meeuwisse WH.
Assessing causation in sport injury: a multifactorial model.
Clin J Sport Med. 1994;4:166-170.

34. Brown JC, Miller C-J, Schwellnus MP, Collins M.
Range of motion measurements diverge with increasing age for COL5A1 genotypes.
Scand J Med Sci Sports. 2011;21: e266-e272.

35. Posthumus M, Collins M, van der Merwe L, et al.
Matrix metalloproteinase genes on chromosome 11q22 and the risk of anterior cruciate ligament (ACL) rupture.
Scand J Med Sci Sports. 2011 Mar 16: doi: 10.1111/j.1600-0838.2010.01270.x.

36. Posthumus M, Collins M, Cook J, et al.
Components of the transforming growth factor-beta family and the pathogenesis of human Achilles tendon pathology—a genetic association study.
Rheumatology. 2010:49:2090-2097.

37. September AV, Nell EM, O'Connell K, et al.
A pathway-based approach investigating the genes encoding interleukin-1β, interleukin-6 and the interleukin-1 receptor antagonist provides new insight into the genetic susceptibility of Achilles tendinopathy.
Br J Sports Med. 2011;45:1040-1047.