

The Sports Gene by **David Epstein** (338 pages, 2013)

A Summary

Introduction – In Search of Sports Genes

Imagine the genome (all 23,000 or so regions of DNA that contain genes) as a 23,000-page recipe book that resides at the centre of every human cell and provides direct instructions – or genes - for building proteins. Some of the 23,000 pages have instructions for an array of functions, and if one page is moved, altered or torn out, then some of the other 22,999 pages may suddenly contain new instructions. The instructional pages, that is, interact with one another.

In the years following the sequencing of the human genome in 2003, sports scientists tried to pick single genes that they guessed would influence athleticism. However, it was found that that single genes usually have effects so tiny as to be undetectable in small studies. Further most of the genes for easily measurable traits such as height have eluded detection.

Slowly but surely scientists have moved beyond single gene studies to explore the combination of genetic endowments that impacts athleticism. Further they have also started to explore how the interplay of biological endowments (nature) and rigorous training (nurture) impacts athleticism.

The broad truth is that nature and nurture are so interlaced in any realm of athletic performance that the answer is usually both. But we cant stop here. We need to ask – 1) How specifically might nature and nurture be at work here 2) How much does each contribute. This book attempts to answer these questions through an exploration of genetic research and what is known about the innate gifts and abilities of elite athletes.

1 – Beat by an Underhand Girl

Top Baseball batters including Albert Pujols, Barry Bonds were unable to put bat to Jenny Finch’s pitches (she was a softball pitcher).

The reaction times of elite athletes are the same as that of ordinary people (1/5ths of a sec). But yet they are able to handle 100mph baseball pitches and 130mph tennis serves. How? The answer came thanks to a series of studies including Adrian de Groot’s 1940s chess studies, Janet Starkes’ occlusion experiments in 1970s, , Herbert Simon & William Chase’s chunking theories of chess expertise & Bruce Abernathy’s cricket research. They found the following

- Chess masters and elite athletes chunk information on the board or the field. Rather than grappling with a large number of individual pieces, experts unconsciously group information into a smaller

number of meaningful chunks based on patterns they have seen before.

- Elite athletes extract critical information and chunk this information into familiar patterns from the arrangement of players or subtle changes in an opponent's body movements, in order to make unconscious predictions about what will happen next. This is how batsmen prepare for an inswinger or how top tennis players discern from the miniscule pre-serve shifts of an opponent's torso whether a shot was going to their backhand or forehand, whereas average players have to wait to see the motion of the racket, costing invaluable response time.

This explains why Pujols couldn't put bat to Jenny Finch's pitches – he had no mental database of her body movements or even the spin of the softball to predict what might be coming.

When Abernathy studied the eye movement of elite and novice badminton players, he saw the novices were already looking at the correct area of the opponent's body. They just did not have the cognitive database to extract information from.

As an individual practices a skill, the mental processes involved in executing the skill move it from the frontal lobes (higher consciousness area) to the more primitive areas (basal ganglia) that control automated processes. In sports, brain automation is hyper-specific to the practiced skill. Even runners who are cycling see activity in the frontal lobe, even though both are endurance activities.

Thinking is the sign of an amateur in sports! The more automatic the better. One way to avoid the pressure of choking (Sian Beilock) is to sing – thus preoccupying the higher consciousness areas of the brain.

Chunking and automation travel together on the march towards expertise. It is only by recognizing body cues and patterns with the rapidity of an unconscious process that an Albert Pujols can determine whether to swing at a ball that has just left the pitcher's hand.

The result of expertise study, from de Groot to Abernathy, can be summarized in a single phrase "It's software, not hardware". Perceptual sports skills that separate masters from novices are learned, and are not innate.

This has helped spawn one of the most popular theories of gathering expertise, including in sports – the ‘10,000-hour rule’ or as its key proponent K Anders Ericsson calls it, the ‘deliberate practice framework’.

2 – A Tale of Two High Jumpers

The 10,000 in the 10,000-hour rule, named by Malcolm Gladwell in his book ***Outliers***, refers to the average numbers of hours of deliberate practice required to attain mastery of a subject or sport. For a 2007 study done for chess masters, it was found that 11,000 hours were required on average to become an ELO master. However the variance was high – one player only needed 3,000 hrs whereas another required 23,000 hrs. The explanation suggested for such a high variance was that individual differences in talent have a huge effect - a kind of butterfly effect of expertise. The scientists said “We assume it takes about 10secs to learn a chunk, and it takes about 300,000 chunks to become a Grandmaster. If one person learns a chunk in about 9s and another in 11s, then these small differences are going to be amplified.

The chapter then elaborates the story of two high jumpers – Stefan Holm, who practiced and practiced his way (*Holm thinks he has taken more high jumps than any other human being*) to an Olympic gold in '04, and Donald Thomas, a newbie who leapt his way to a surprise victory in the Osaka Athletics World Championships in '07.

Holm was only 5'11" – so along with his relentless practice, he also weight-trained, concentrating on his left leg, to the point where he could squat 2x his bodyweight. He also focused on a faster run-up, hitting a speed of 19mph. To accommodate that he had to create a longer run-up.

Holm's training routine – about 12 sessions a week – was split into a morning routine starting around 10am of 2 hours of weights, box jumps, hurdles, and a late afternoon session of at least 30 jumps at full-competition height, making sure that he trained until he crossed whatever height he had set for the session.

Holm's standing vertical jump was only 28", but his fast approach allowed him to slam down on his achilles tendon, which acted like a rebounding spring to propel him over the bar. His Achilles tendon had stiffened so much from jumping, that a force of 1.8 tons was needed to stretch it 1cm (4x the normal stiffness).

Donald Thomas had barely 8m of training when he won a gold at the Osaka World Championships in '07 beating Stefan Holm. Later on, scientists identified one of the key reasons for his dramatic success – a 10.5" uncharacteristically long Achilles tendon. The longer and stiffer the tendon,

the more elastic energy it can store, and when stretched rocket its owner into air. This is called the stretch-shortening cycle – basically the stretching and subsequent snapping back of of the spring-like tendon. The more power stored in the spring, the more power you generate when it is stretched.

Tendon length is a function of the distance between calf muscle and heel bone, which is connected by the Achilles tendon. Tendon stiffness can be increased by training, but there is also growing evidence that stiffness is partly influenced by an individual's version of genes involved in making collagen, a protein in the body that helps build ligaments and bones.

A longer and stiffer Achilles tendon is only one of the factors which explains how Holm and Thomas arrived at the pinnacle of their sport in such diverse fashion. Interestingly Thomas has not improved 1cm since he entered the professional circuit, whereas Holm improved constantly through his career.

Sports science has found a huge variation – as much as 10x even – in the number of hours needed to attain an elite / international level. In fact even the 10,000-hr number is being questioned. The average sport-specific hours required to reach international levels in Basketball, Field Hockey and Wrestling as estimated by their respective bodies are 4000, 4000 and 6000 hours respectively.

The chapter also details an investigation by Edward Thorndike which discovered a kind of Mathew Effect (“the rich get richer and the poor get poorer”) in training. Increased practice enhance’s everyone’s results but the difference between the haves and have-nots gets magnified. Similar research by Philip Ackerman has revealed that in simple tasks, increased practice brings people together, but in complex tasks, it pulls people apart. The individual differences go up in tasks involving complex motor skills thanks to practice and not down.

By the strictest 10,000-hr thinking, accumulated practice should explain most of the skill variance (variance is the dispersion from the mean. If there are 2 people and one finishes a mile in 4mins and the other in 5mins, then the mean is 4.30mins and the variance is 1min). However sport science says that the amount of variance accounted for by practice is between low and moderate (a study by K Anders Ericsson on darts put it at 28%). The data clearly supports a view of skill acquisition as innate hardware and learned software.

3 – Major League Vision and the Greatest Child Athlete Sample Ever

Researchers have found that Major League Baseball (MLB) players have on average, visual acuity of 20/13. In fact most of the hitters even touch 20/11.

(A person with 20/13 vision can stand at a distance of 20 ft and can tell the difference between 0 and c that a person with 20/20 vision can only detect if they stood 13ft away). 20/8 is around the theoretical limit of human visual acuity – this is really rare – an Indian survey of 9,411 revealed just one eye with 20/10 vision. The degree of visual acuity is predetermined by the density of particular photoreceptor cells or cones in the macula, an oval-shaped spot in the centre of the eye. Cones vary between 100,000 cones / mm² to 324,000 / mm², and this is genetically predetermined.

While MLB hitters might not have any faster reaction time (Ch 1), they do have superior vision that can help them pick up the anticipatory cues they need earlier, making slow reaction speed less important. Baseball players have to know before the final 200 milliseconds of a pitch as to where to swing, so the earlier they pick up the anticipatory cues (such as position of the seam) the better.

“It is not the bat speed, it is the visual skills. That little bit is the difference between ordinary and extraordinary” – Al Goldis, an MLB talent scout

A research project done with women possessing normal visual acuity but varying depth perception, revealed that those with good depth perception improved during softball training, while those with poor depth perception did not improve at all. Better hardware sped the download of sport-specific software.

Interestingly, a 2009 Emory study suggested that children with poor depth perception start self-selecting themselves out of Little League baseball and softball by 10 yrs.

While physical hardware alone, like depth perception or visual acuity, is as useless as a laptop with an OS but no programs, innate traits have value in determining who will have a better computer once the sport-specific s/w is downloaded.

Wolfgang Schnieder’s studies on 8-12 yr old tennis players (the ‘Greatest Child Athlete Sample Ever’ referred to in the title) revealed that children’s tennis-specific skills acquired through practice, such as the accuracy with which a player could return a ball back to a specific target, accounts for about 60-70% of the variance in their eventual tennis ranking. He also found that tests of general athleticism – such as in a 30m sprint or start-and-stop agility drills – influenced which children would pick up the tennis-specific skills most rapidly. Thus the kids who were better all-round athletes (hardware) were better at acquiring tennis specific skills (software).

The Groningen Talent Studies on Dutch soccer youth players revealed that some of the traits that help predict future pros are behavioural – the future pros not only tend to practice more, but they also take responsibility for practicing better, such as asking the trainer for feedback. The Groningen studies confirmed the conclusion reached with the tennis players – small variations in physical traits at age 12 delineates the haves from the have-nots. A gap of 0.2s in shuttle sprints is a good predictor of who will make pro and who will fall behind.

“It is important to be fast. You need a minimum speed. If you are really slow, then you cannot catch up, and speed is really hard to train...”

The chapter talks about how the Australian Institute of Sports selected candidates for the Winter Olympics sport of Skeleton on the basis of general non sport-specific athleticism. They had found that the beginning sprint accounts for about half of the variance in total sports time. So they looked for women who could sprint and fit snugly on a tiny sled. The initiative succeeded beyond their wildest imagination.

Australia’s success with talent transfer attests to the fact that a nation succeeds in a sport not only by having many athletes who practice prodigiously at sport-specific skills, but also by getting the best all-round athletes into the right sports in the first place.

Even at the most basic levels, it is always a hardware and software story. The hardware is useless without software and vice versa. Sports skill acquisition does not happen without both specific genes and a specific environment, and often the genes and the environment must coincide at a specific time.

Interesting factoids – the chance of reaching the international masters level in chess was drastically reduced if the player did not start serious chess by 12 years. It didn’t matter how early they started, as long as it was before 12. Perhaps 12 is an approximate age by which certain chunks must be learned and certain neuronal connections reinforced lest the opportunity be lost.

We are born overflowing with neurons, and the ones we don’t use early are pruned away, and those that we do are strengthened and inter-connected. The brain becomes less broadly flexible but more narrowly efficient.

This is one reason why advocates of the strict deliberate practice approach suggest training should begin as early as possible. But there is a growing body of scientific evidence which suggests that early specialization, barring in certain sports such as gymnastics, is not only not required to make it to the highest level, but should perhaps be actively avoided.

In sprinting, early heavy and specific training often leads to the athlete getting stuck in a certain top speed and running rhythm – the dreaded speed plateau – ingrained in him due to his early training.

A 2011 Danish study of athletes in cgs sports (sports measured in cms, gms or secs) found that both elites and near-elites had both sampled a number of sports in childhood but could be distinguished by a certain quality indicative of specialization. The near-elites practiced more than the elites by age 15. It was only after age 15 that the elites accelerated their practice pace and by age 18 had surpassed their near-elite peers in training hours.

Often practice-only narratives appeal to our innate sense of fairness – that the right environment and effort can make anything possible. But narratives that shun the contributions of innate talent can have negative side effects in exercise science. Without genes the picture of sports expertise is woefully incomplete. As renowned exercise researcher Janet Starkes asks “if only accumulated hours of practice matter, then why do we separate men and women in athletics?”

4 – Why Men Have Nipples

The chapter starts with the tragic case of Spanish female athlete Maria Jose Martinez-Patino, whose career stalled after she was diagnosed as a man, and then proceeds to use that as a case study to explore physical differences in the sexes and how they influence athleticism.

In running, from 100m to the ultramarathon, the top 10 men are on average about 11% faster than the top 10 women. Larger gaps occur in throwing and explosion events (javelin gap = 30%, long jump gap = 19%), while the smallest gap (6%) occurs in distance swimming races.

Why does this gap exist in the first place?

Sexual selection (those accumulated DNA changes that spread or die out as a result of the competition for and the choosing of mates) is the source of most human sex differences, and is vital to the understanding of human athleticism.

Among the key physical differences between the sexes. Men are / possess

- heavier and taller
- longer arms and legs relative to their height
- biggest hearts and lungs, thus able to absorb and process more oxygen

- twice as likely to be left-handed (high physical combat societies have more numbers of lefties – this arose due to natural selection as lefties have an advantage in combat)
- less fat
- denser bones, and a heavier skeleton that can support more muscle
- more oxygen-carrying red blood cells
- narrower hips which makes running more efficient and decreases the chances of ACL tears (epidemic in female athletes) while running and jumping
- 80% more muscle mass in upper body and 50% more in lower body

The differences in size and strength between men and women is analogous to those in gorillas, the most sexually dimorphic of our close relatives (male gorillas are 2x the size of female gorillas). The reason for this similarity arises from a general principle – the sex that has the higher potential reproductive rate gets bigger (gorillas vs seahorses). The male of the species in primates that have intense male-male competition (for females) grows bigger thanks to sexual selection that favours bigger and better fighters.

A pattern that holds across species with high male-male competition is that physical abilities essential to combat are bolstered, exclusively in males, via puberty. Up until the age of 10, both boys and girls have similar bodies with most athletic traits nearly indistinguishable.

While girls mature early and quickly, boys go through a puberty that is both late and long, giving more time for growth, during which their athleticism explodes. They develop stronger arms and wider shoulders, and can throw much further (by 18, 3x that of girls). Interestingly, the boys also develop facial features that make it easier to withstand blows.

The testosterone surge of male puberty also stimulates the production of more RBC, such that men are able to use more oxygen than women, and it also makes men less susceptible to pain than women. In some cases, women fare worse after puberty, as increased oestrogen leads to fat accumulation on hips, leading to a plateau or decline in the vertical jump.

Female athletes have traits that are more typical of men, such as low body fat and narrower hips. In certain cases these are the result of an XY gene (male gene). Sometimes the XY gene is paired with androgen insensitivity (which makes it impossible for the body to absorb the increased testosterone produced by the body), as in the case of female athlete Maria Jose Martinez-Patino.

The increased height of XY women, may result from an extended growth period, because their bodies don't heed hormonal stop messages, or from genes on the Y chromosome influencing height (men who have an extra Y chromosome – XYY – tend to be very tall).

Much of sexual differentiation comes down to a single gene on the Y chromosome, the SRY or the Sex Determining Region Y Gene, which triggers testis development. The DNA differences between men and women are extremely small, limited to the single chromosome that is X in women and Y in men. Human biology is set such that the same two parents can produce masculine sons and feminine daughters even though they are passing on the same genes. The SRY gene is a DNA skeleton key that selectively activates the genes that make the man. In so far as there is an athleticism gene, this is it.

An interesting factoid about women athletes is that the elites have testosterone levels that have consistently remained more than twice as high as that of the non-elites. A typical woman makes less than 75 nanograms of testosterone per decilitre of blood. For men the range is between 240-1,200.

Why are women athletic at all? For much the same reason that men are athletic.

The question is analogous to why men have nipples. They have nipples because women have them. Nipples are essential for reproductive success in women, but are not so harmful in men that there has been significant natural selection pressure to get rid of them. Instead of great numbers of genes changing, nature has left us with a system whereby men and women have almost entirely the same genes, and hormones can selectively activate genes to different effects. The small genetic difference in the SRY gene can induce a cascade of biological consequences that lead to huge disparities in the fields of play – not just in physical proportions or traits, but even in trainability! There are actually small DNA differences on Y chromosome that ultimately affect even trainability.

5 – The Talent of Trainability

In 2011, a seminal and long-running research initiative called **HERITAGE** (**H**ealth, **R**isk Factors, **E**xercise **T**raining and **G**enetics) reported a breakthrough in exercise genetics when they identified 21 gene variants – slightly different versions of genes between people – which accounted for about 50% of an individual's potential for aerobic improvement. This still leaves about half of aerobic trainability due to other factors, but the 21 gene markers had the power to delineate the high and low responders. HERITAGE subjects who had 19 of the favourable versions of the genes improved their

VO₂ max upon receiving aerobic training to nearly 3x that of subjects with similar training, who had fewer than 10 of the favourable versions.

Improvement in VO₂ max (or aerobic capacity) – the amount of oxygen that a person's body can use when he is going all out – was used as the key measure of aerobic trainability. VO₂ max is determined by how much blood the heart pumps, how much oxygen the lungs impart to that blood and how efficient the muscles are at snatching and using the oxygen from the blood as it hurtles past. The more oxygen one can use, the better one's endurance.

Endurance exercise also in turn boosts VO₂ max – more blood is produced, and it flows through new capillaries that sprout like roots into muscle. The heart and lungs strengthen, and energy-generating mitochondria proliferate in the cells.

The HERITAGE researches also found that the impact of endurance training led to a wide variation in VO₂ max improvements. The amount of improvement had nothing to do with how aerobically fit the person was to start with. In some cases, the poor got relatively poorer (people who started with low aerobic capacity and improved little); in others the oxygen rich got richer (people who started with higher aerobic capacity and improved rapidly); with all manners of variation between – exercisers with a high baseline aerobic capacity and little improvement and others with a meager starting aerobic capacity whose bodies transformed drastically.

It is still unknown whether the predictor genes that Bouchard and crew have identified are the important genes themselves or whether they are simply markers for broader networks of genes. Gene expression data suggests that hundreds of genes are involved in each person's response to exercise – some like RUNX1 gene are likely involved in changes in muscle tissue or in the formation of new blood vessels. Still others are found among genes that have helped organisms adapt to life in the oxygen-rich atmosphere of Earth starting three billions of years ago.

It is thus clear that some people appear to be trainability bombs, waiting to go off as training begins, their bodies's endurance power improving steadily. They are gifted with high trainability. Yet others as low responders may find that their predetermined genetic soup may not spell 'runner'. Yet there is a bright side. If doctors know how a patient responds to exercise, they can determine whether an exercise plan can usher in a desired health benefit such as lower BP or to resort to medication, thus impacting outcomes earlier.

Interestingly, just as for aerobic training, low and high responders have been identified using experiments based on explosive training as well. This shows

that there is no one-size-fits-all training plan. If you suspect that you aren't responding as well to a particular training stimulus, then rather than giving up, you could try something different.

The chapter proceed to look at rare cases of high baseline aerobic capability (high VO_2max) even without training, sometimes in people leading sedentary existences. These are individuals with blood volumes that could have been mistaken for that of endurance athletes (VO_2max scores 50% higher than the average untrained persons). This is thanks to increased diastolic filling – meaning the part of the heartbeat when the heart muscle relaxes to allow blood back in. “When you fill the right side of the heart with more blood, it then pumps more blood into the left side, and the left side pumps it into the body. The return of blood to the heart is enhanced because of the extra blood volume.

The chapter also looks at some real-life cases of people with naturally occurring high baseline capability (“naturally fit” individuals) such as middle-distance runners and Olympic medalists Jim Ryun (also a high responder) and Meb Keflizhigi, triathlete Chrissy Wellington and 1976 Olympics' 400-800m gold medalist Alberto Juantorena, dwelling at length on their rise to success.

6 – Superbaby, Bully Whippets, and the Trainability of Muscle

Muscles are pieces of meat made of millions of tightly packed threads, or fibers, each a few millimeters long and so thin as to be barely visible on the end of a needle. Along each fiber are a number of command centres, or myonuclei, that control muscle function in the area. Each command centre presides over its fiber fiefdom.

Outside of the fibers hover satellite cells. These are stem cells that wait quietly, until muscle is damaged – as happens when one lifts weights – and then they swoop in to patch and build the muscle, bigger and better.

For the most part, as we gain strength we do not gain new muscle fibers but simply enlarge the ones we already have. As a fiber grows, each myonuclei command centre governs a larger area, until the point when the fiber gets big enough that the command centre needs backup. Satellite cells then form new command centres so that the muscle can continue to grow.

Just like the HERITAGE study, there have been studies showing diverse responses in muscle fiber growth to workouts. The subjects whose muscle fibers grew the most had the highest satellite cells in their muscles, waiting to be activated and build muscle. Their default body settings were better

primed to profit from weightlifting. One reason steroids help in rapid muscle gain is because they help prompt the body to make more satellite cells available for muscle growth.

3 genes show higher expression levels in the bodies of extreme responders to weight training – all influencing muscle function and growth

- 1) IGF – IEa : IGF stands for Insulin-like Growth Factor
- 2) MGF
- 3) Myogenin

Another cause of differing individual response to strength training lies in the different allotment of muscle fibers. These come in 2 major types –

A – Slow twitch or Type 1

B – Fast-twitch or Type 2

Fast-twitch fibers contract at least twice as quickly as slow-twitch fibers for explosive movements – the contraction speed of muscles has been shown to be a limiting factor of sprinting speed in humans – but they tire out quickly. Fast-twitch fibers also grow twice as much as slow twitch fibers when exposed to weight training. So the more fast-twitch fibers in a muscle, the greater its growth potential.

Slow-twitch fibers require abundant oxygen and thus are surrounded by blood vessels, which make them appear dark. Most people have muscles comprising slightly more than half slow-twitch. But the fiber type of athletes fit their sports. Sprinters are 75%+ fast-twitch, and marathon-runners are at the opposite end. The more the distance specialized in, the greater the percentage of slow-twitch muscles. This begs the question of whether athletes get their unique muscle fiber combinations via training or whether they gravitate to and succeed in their sports because of how they're built. Evidence suggests the latter.

Interesting factoid: Aerobic training can make fast-twitch fibers more enduring and strength training can make slow-twitch fibers stronger, but they don't completely flip; only if one's spinal cord is severed, in which case all fibers revert to fast-twitch.

Interesting factoid 2 : The guys with a lot of fast-twitch fibers that can contract their muscles very fast have much more risk of a hamstring injury than those with a lot more slow-twitch fibers. The former get injured far more often. Armed with this finding, coaches are beginning to customize their training plans to fit the body type of the individual. For some athletes, less training is the right medicine.

The Institute of Sports Medicine, Copenhagen, is using muscle biopsies to identify proportions of fast and slow-twitch fibers of an athlete's body and then direct him or her into the appropriate sport (kayaker with 90% slow-twitch fibers moved from 500m race to long-distance competitions).

The term superbaby in the chapter's title refers to a baby born in Berlin in late 90s who had mutations on both his myostatin genes (on chromosome 2), and was born packed with muscle. Myostatin genes act as a check on muscle growth, primarily because muscle is costly – it requires calories, and specifically protein to sustain it and having massive muscles can be a big problem for organisms including humans that don't have steady access to the protein necessary to feed the organs; though this is a diminishing concern now.

Bully whippets refer to racing dogs (whippets) that are bred to be born with a myostatin mutations on one of their genes. These turn out to be ace racing dogs. However if they are born with two copies of the mutation, they are born heavily muscled and buff, and are too bulky to sprint. These are typically put down.

7 – The Big Bang of Body Types

In 1925, an average elite volleyball player and discus thrower were the same size, as were a world-class high jumper and shot-putter.

As winner-take-all market emerged in sport and a tiny elite at the apex of their sport taking home disproportionate rewards - in 1975 sportsmen made 5x the median salary while today they make between 40-100x - intense competition emerged in each sport, and over time this led to self-selection of traits required for success in each sport - high jumpers became taller and leaner, shot-putters became taller and heavier, female gymnasts and male divers became smaller and leaner. Thus the singular, perfect athletic body faded in favour of more rare and specialized bodies that fit like finches' beak into their athletic niches.

When Australian sports scientists Kevin Norton and Tim Olds plotted on a height-weight graph the average physiques of elite athletes in two dozen sports in 1925 and for 1995, they discovered a distinct pattern. In 1925, the top athletes from every sport clustered around that average physique, but by 1995 they had blasted off in every direction. Norton and Olds called it the Big Bang of Body Types, given it was similar to how galaxies were speeding away from each other.

The world of pro sports has become a laboratory experiment for extreme self-sorting, or artificial selection, as Norton and Olds called it, as opposed to natural selection.

Big Bang data in hand, Norton & Olds devised a measure that they called the Bivariate Overlap Zone (BOZ). It gives the probability that a person randomly selected from the public has a physique (height and weight) that could possibly fit into a given sport at the elite level. As the Big Bang has proceeded, the genes required for any given athletic niche has become rare, and the BOZ for most sports has decreased profoundly.

The BOZ for professional soccer players is 28% , 23% with elite sprinters and 9.5% with Rugby stewards, and just 0.5% with supermodels. The Big Bang of body types goes down to the body part as well. Croatian Water Polo players' arm length increase more than an inch, 5 times more than that of the Croatian general population. As performance requirements become stricter, only the athletes with the necessary physical structure consistently make the grade at the elite level. The shorter-armed athletes (like junior league baseball players with worse than 20/13 vision) are weeded out.

Body types have more nuanced effects as well. Going back to Croatian Water Polo players, we find that not only do they have longer arms, but they have longer lower arms as well, giving them a more efficient throwing whip. In an other case, taller (5' 8") marathoner Paula Radcliffe typically fails during summer marathons (2004, 2008 Olympics) because her smaller skin area (relative to her volume) means her body cannot dissipate heat as efficiently as shorter runners. Heat dissipation is critical for endurance running as the Central Nervous System forces a shutdown or stop of effort as body's core temperature passes 104° F. Hence Paula Radcliffe's best runs have come only on cooler autumn days including her World Record run.

Interesting factoid: Among the 6 DNA variants that influence heft, the most critical is the FTO gene. However at present our studies are still incomplete and more genes are likely to be found soon. The present 6 do not account for a significant fraction of bulk. Lifestyle has a bigger impact. It is also interesting to note that the more the proportion of fast-twitch fibers a person has, the lower his capacity to burn fat – one possible reason sprint and power athletes tend to be bulkier than endurance runners, even after their training stops.

It is also interesting to note that like muscle, bone responds to exercise as well. The serving arms of tennis players have forearm bones a quarter of an inch longer than the forearm bones of the other arm. This is true of nonathletes as well. We tend to have more bone in the arm we write with,

simply because we use it more, so the bone becomes stronger and capable of supporting more muscle.

The skeleton you are bequeathed with will determine whether you will be able to make the weight required for a particular sport. Each kg of bone supports 5 kgs of muscle. If you are at 5:1 then eating more will lead to putting on fat, not muscle. This can be of help to shot-putters as they don't need to move very far, so even adding extra fat might be worthwhile, since the athlete needs to pack on bulk to become relatively more muscle than the object being thrown. This is also true of offensive linemen in American Football and Sumo wrestlers. However in Javelin since the athlete needs to both run fast and throw hard, he should not add weight beyond the 5:1 ratio.

Today the expanding universe of body types is slowing down. Much of the self-sorting, or artificial selection, is finished. Population progression is slowing globally, and so we are going to see slowing growth in both body size and body shapes, and records as well.

8 – The Vitruvian NBA Player

A mere 5% of American men are 6'3" or taller, whereas the average height of an NBA player is 6'7". The probability that an American man between ages of 20 and 40 rises exponentially with every 2" height increase (Between 6'0" and 6'2" the probability of his being in NBA is 5 in a million; between 6'2" to 6'4" that increases to 20 in a million, between 6'10" to 7'0" it rises to 32,000 in a million or 3.2%; For those Americans standing 7'0" and above, the probability of his being in NBA rises to 17%). "What it means is that basically anyone who can play basketball and is 7ft tall is part of the league. " – Tim Olds

Interestingly the number of 7-footers grew from 0 to 5% of the NBA till the early 80s. Post '83, when a winner-take-all market emerged thanks to the collective bargaining agreement between league and players, and the entry of Nike, the number of 7-footers jumped to 11% where it has stayed since.

Unlike the men's basketball league, the WNBA does not have players disproportionately taller than the normal population. The average WNBA height is 5'11" – 6'0", which is only about 10% taller than the average woman's height (men 15%). One reason is that the WNBA is still not as lucrative a sport as the NBA is, and many taller women gravitate to sports such as tennis where they can make far more money. The top 3 women's tennis players have a height of 5'11" 2/3 which is equal to the average WNBA height.

While inhabitants of the industrialized world grew at an average of 1 cm a decade in the 20th century, thanks to more protein in the diet and lack of growth-stunting childhood infections, and perhaps due to tall and short people mixing genes with tall genes dominating short ones, NBA players have been growing at 4 times the rate, and the tallest of NBA players at 10x the rate.

There are short players such as Muggsy Bogues (5'3"), Nate Robinson (below 5'8") who have played successfully in the NBA. However they could all jump spectacularly (all NBA drafts have been able to touch the rim). And more critically, their arm spans enabled better reach. Nate Robinson with a near 5'8" height had an arm span of 6'1". The average arm-span to height ratio of an NBA player is 1.063 (a ratio of 1.05 and above is one of the diagnostic tests for Marfan Syndrome, the disorder of the body's connective tissues which results in elongated limbs). Unlike the traditional vitruvian man, whose arm span = height and hence fitted into the circle, the vitruvian NBA player would fit into an ellipse and rectangle not a circle and square.

Stat-savvy NBA managers such as MIT-educated Daryl Morey of Houston Rockets have been cleverly drafting superficially undersized players with superior wingspan. The bottom line is that when an NBA player doesn't have the height required to fit into his slot in the athletics-body universe, he nearly always has the arm-span to make up for it.

Studies have shown the heritability of height at about 80%, and balance 20% of the height differences between people can be attributed to environment. In non-industrial societies, heritability is lower as people are prevented from reaching their genetic height potential by nutritional deficiencies.

There are large number of genes which contribute to height differences. Thus far we are not even halfway through identifying genes which contribute to height differences. However the genetically programmed nature of height is obvious from studies on identical twins. An interesting factoid is that when children are exposed to brief period of famine, their growth grounds to a halt. When food becomes plentiful, their bodies catch up with their genetically determined height potential. The undernourished child slows down and waits for better times. Similarly female gymnasts delay their growth spurt with furious training, but that doesn't delay their ultimate adult height.

The height that inhabitants of urban societies gained over the 20th century has come principally from increased leg length, and not torso length (We see this in Japan as well). In developing countries with severe nutritional disparities, leg length is the difference in height between the comfortable and the afflicted.

One body type difference that has attracted the interest of sports scientists is the greater leg length of African Americans (2.4” longer than that of a European). Not only do legs make a greater proportion of the body in an individual of recent African origin (Allen’s rule as you will read below), they also tend to have narrower pelvic breadth (Bergmann’s rule after 19th-century biologist Carl Bergmann), giving them a more linear build. Both these traits – narrower pelvis and longer legs – aid in running better.

An analogy can be drawn with the advantages enjoyed by short-limbed Asians and eastern Europeans, who have had a long history of success in weightlifting and gymnastics. Another interesting body type difference is the centre of mass – black adults have a higher centre of mass (approximately the belly button) that is about 3% higher than white adults. This translates into a 1.5% running speed advantage for adults with a higher belly button, and a 1.5% swimming speed advantage for athletes with a lower belly button (white athletes).

In 1877, American journalist, Joel Asaph Allen, published a paper which noted how the extremities (limbs) of animals get longer and thinner as one travels closer to the equator (African elephants’ ears etc). The underlying basis for this is heat dissipation (as you get to sweat over a greater surface area). It is therefore not so much ethnicity (black vs white) as much as geography (Africans living away from the equator, do not necessarily have especially long limbs).

9 – We Are All Black (Sort Of)

Humans split from our common ancestor with chimpanzees five million or so years ago. For millions of years, DNA changes had accumulated – both randomly and by natural selection – in the genomes of our ancestors inside Africa. But with only 90,000 years for unique changes to occur outside of Africa, there simply hasn’t been as much action in many stretches of the genome. Thus the basis for the finding that African population has more variations – different spellings of the same gene or area of the genome – in their DNA than populations from East Asia or Europe. In fact there is more genetic variation among Africans from a single native population than among people from different continents outside of Africa.

The implication for sports: Kenneth Kidd, a scientist, says that for any skill that has a genetic component, the most and least athletically gifted individuals in the world might be of African origin. There are of course also average differences between populations, which is why Masaais are tall and Pygmys are short. Thus Kidd is suggesting that certain Africans or those of

African origin, do have a genetic advantage in sports at the upper end of athleticism. But they don't have an average genetic advantage (this makes it palatable to other scientists and the press).

Before scientists go about identifying whether Africans or Americans have specific genes conferring advantages in certain sports, they should first identify specific genes that have advantages for sports performance, and then examine whether they occur more frequently in some populations than the rest. For example, about 90% of the variation in the shape of human skulls occurs within every ethnic group – only 10% separates ethnicities – with Africans indeed showing the greatest variation. But the exact opposite is true for skin colour. About 90% of the difference is between groups.

The chapter speaks about the ACTN3 gene, which enables the production of a particular structural protein called alpha-actinin-3 in the muscles. Structural proteins are critical for the body. They make fingernails, hair, skin, tendons and muscle. Humans tend to be diseased or die when the genes that code for them are not functioning. One finding is that there are no top-level sprinters who have stop codons (a single letter switch in the DNA that prevents alpha-actinin-3 from being produced in their muscles) on their ACTN3 genes.

ACTN3 is clearly a gene for speed. Why this is so is not clear? When mice are bred for no alpha-actinin-3, their muscle fibers have very little active glycogen phosphorylase, the enzyme that mobilizes sugar for explosive actions like sprinting, and their fast-twitch muscles take on the properties of slow-twitch endurance fibers. Japanese and American women who are deficient in alpha-actinin-3 have smaller fast-twitch muscles and less muscle mass over all.

Why would the body allow stop codon mutations on ACTN3 genes? Possibly to make fast-twitch fibers metabolically efficient, like slow-twitch muscles – a boon in frigid, food-scarce northern latitudes. An other theory, given the approximate timing of when the stop mutation on ACTN3 began appearing (15-30,000 years ago), is to link it to the transition to agriculture, when humans would have had less need to sprint in war or hunting, but would need to be metabolically efficient and to work at a steady rate for long hours.

10 – The Warrior-Slave Theory of Jamaican Sprinting

The Warrior-Slave Theory is a popular lay theory that emerges from the finding that most of the top sprinters hail from the district of Trelawny, which is where most of the fiercest slaves (originally enslaved from the fiercest warrior tribes of Africa) escaped and congregated to. In this remote

area, walled in by sheer vertiginous cliffs, they formed a fierce tribe, called Maroons, who after several wars with the British were given freedom.

Thus the Warrior-Slave theory explains Jamaica's sprint success in a Darwinian fashion, stating that strong people were taken from Africa; that the strongest survived the voyage to Americas, the strongest of those strong fed the Maroon society that cloistered itself in the most remote region in Jamaica, and that the Olympic sprinters of today come from that isolated, warrior genetic stock.

However data doesn't support the above theory – there are top sprinters from other regions of Jamaica as well, and secondly it is hard to confirm that all of the top Jamaican sprinters are pure-bred Maroons. There is also no way genetically to distinguish a Maroon from other Jamaicans today. Essentially research has shown that neither Maroons nor Jamaicans overall constitute any sort of isolated, monolithic genetic unit. Rather, as we should expect from a mixed group of West Africans, Jamaicans are highly genetically diverse.

It is more likely that Jamaica's success in sprint comes from the administrators' ability to divert the most promising talent into track. In US, similar talent like Bolt (6'5" and sprinter) goes into American football as wide receivers. There is way too much money in US Football. In Jamaica there is no such alternate lucrative sport to tempt a Bolt or Powell. There are worries now that the increasing popularity of Basketball could siphon off track talent.

11 – Malaria and Muscle Fibers

In 2006, Jamaican scientist Errol Morrison and writer Patrick Cooper, proposed that rampant malaria along the west coast of Africa, from where slaves were taken, led to specific genetic and metabolic alterations beneficial for sprint and power sports. The hypothesis: malaria in western Africa led to proliferation of characteristics like a high prevalence of the sickle-cell gene mutation (and other gene mutations) that cause low haemoglobin for protection from malaria. These mutations which reduce an individual's ability to make energy aerobically, led to a shift to more fast-twitch muscle fibers, which are less dependent upon oxygen for energy production.

The first part of Cooper's (and Morrison's) hypothesis that sickle-cell trait and low haemoglobin are evolutionary adaptations to malaria are now proven. It is the coda of the Cooper and Morrison hypothesis that fast-twitch fibers moved in as haemoglobin moved out that that is considered speculative. There is evidence of low haemoglobin prompting switch to fast-twitch fibers in a study done on rodents, who were put on iron-deficient

diets, and on rats put on periodic blood draws. But no one has done a study on humans and mice have a greater ability to swap muscle fiber than humans do. So the hypothesis remains as yet unproven.

12 – Can Every Kalenjin Run?

The chapter starts by describing the KenSAP (Kenyan Scholar Athlete Project), an initiative that aims to get top Kenyan students from the hotbed of endurance running, the Western Rift Valley into premier U.S. colleges. KenSAP selects these students on the basis of their scholastic performance as well as a 1500m race.

The Western Rift Valley is inhabited by the Kalenjins, a 4.9m people who represent about 12% of Kenya's population but over 75% of the top runners. One explanation (by John Manners who founded the KenSAP initiative) of their prowess is founded on their cattle-raiding skills – from the practice of stealthily walking into the land of neighbouring tribes, rounding up cattle, and escorting them back into Kalenjin land as quickly as possible. Distances of 100miles were not uncommon.

Given that successful cattle raiders had to be strong runners to hustle captive herds to safety, and the best cattle raiders accumulated more wives and children (typically a *muren*, or warrior, who brought back a large number of cattle from a raid was hailed as a courageous and athletic warrior, and could use his cattle and prestige to acquire wives), then cattle raiding could serve as a mechanism of reproductive advantage that favoured men with superior distance running genes.

A 1998 study by University of Copenhagen's Muscle Research Centre comparing Kenyan runners to their Danish counterparts, yielded the following findings – not only were the Kenyan athletes legs longer – on average Kalenjin boys were 2" shorter than their Danish peers, but had legs that were about $\frac{3}{4}$ " of an inch longer (and Achilles tendons longer by 2.7" and therefore able to store more energy). But the most unique findings was not the length of the legs but their girth. The volume and average thickness of the lower legs of the Kalenjin boys was 15-17% less than in the Danish boys. This finding is substantial because the leg is akin to a pendulum, and the greater the weight (distal weight) at the end of the pendulum the more the energy required to swing it. This is also the logic behind lighter shoes.

A pound less on the legs leads to energy savings at 8% per kilometer, and thus better Running Economy (measure of how much oxygen a runner utilizes to run at a given pace). Elite distance runners thus have both high

VO₂ max and good running economy (imagine a car having a rare mix of a big engine and good fuel economy).

It isn't as if thin legs are confined only to the Kalenjin. But the Kalenjin do in fact have a particularly linear build, with narrow hips and long thin limbs. This body type is referred to by Anthropologists as a Nilotic type – this type evolved in low latitude environments that are both hot and dry, because the long thin proportions are better for cooling. The opposite of this is the short, stocky type, referred to as the Eskimo type, though this term is not used because Eskimo is considered derogatory in some countries.

Another explanation for the success of Kenyan (Kalenjin) runners lies in the fact that countries such as U.S., Britain grew wealthy, fat and were increasingly interested in other sports, thus less likely to train seriously in distance running. Between 1983 and 1998 the number of U.S. men who ran under 2.20 in the marathon declined from 267 to 35! Meanwhile Kenya exploded from a single sub-2:20 man in 1980 to 541 by 2006, thanks to declining travel restrictions and disappearance of a notion that marathon training causes male infertility.

13 – The World's Greatest Accidental (Altitudinous) Talent Sieve

Interestingly research by Yannis Pitsiladis reveals that most of these international-level runners in Kenya are from the Kalenjin tribe, most often from poor, rural areas and very likely had to run to school growing up. There are of course conspicuous exceptions to the 'run to school' rule, such as Paul Tergat & Wilson Kipketer who lived next door to the school. But the poverty rule is key – typically an Olympic medalist's kids never take to distance running – because they have grown up comfortably thanks to the increased earnings, and the hunger that drove their parents' training no longer exists. Pitsiladis also believes that there is another essential component to Kenyan running success. They are lucky to live at a height between 6 - 9,000 ft above sea level. This seems to be a rough sweet spot for training, an altitude high enough for physiological changes to happen and where red blood cell production increases, but not so high like the Himalayas that the air is too thin for hard training.

Western athletes who have consistently trained at altitude have seen adaptations in their body leading to higher red blood cells and a higher VO₂ or improved running economy. Preferable to moving to a higher altitude for training is being born there.

Being born (and growing up) at that altitude confers an important physiological advantage over sea-level natives – larger lungs which have

larger surface areas that permit more oxygen to pass from the lungs into blood. This is not genetic, as it is seen amongst American children who do not have altitude ancestry but are born and grow up high in the Rockies. Once childhood is gone, so too is the chance for this adaptation. It is not genetic, but neither is it alterable after adolescence.

Thus it is the combination of impoverished backgrounds, birth and childhood at a high altitude filled with running, and having disproportionately thin legs that comprehensively explains Kenyan running success.

14 – Sled Dogs, Ultrarunners and Couch Potato Genes

The chapter looks at Alaskan Huskies bred to run in Iditarod Trail Sled Dog Race, and triathlete / ultrarunner Pam Reed to come up with the theory that there are people (and animals) who are biologically predisposed to get an outsized sense of reward or pleasure from being constantly in motion? All 16 studies conducted thus far have found a large contribution of heredity to the amount of voluntary physical activity that people undertake.

The genetic basis for running addiction has been linked to genes that control dopamine, a neurochemical in the brain that conveys messages between cells. It is clear that the dopamine system responds to physical activity. This is one reason that exercise can be used as part of treatment for depression and as a method to slow the progression of Parkinson's disease, an illness that involves the destruction of brain cells that make dopamine. And there is evidence that the reverse is true as well, that physical activity levels respond to the dopamine system.

Particular variants of dopamine receptor genes – which control dopamine production – have been associated with higher physical activity and lower body-mass index. One of those variants, the 7R version of the DRD4 gene, increases an individual's risk for attention deficit hyper-activity disorder or ADHD. Ritalin drives dopamine up in hyperactive children, and their activity decreases. While this may be a good thing for kids who have difficulty sitting still in school, it may have unintended consequences, as sports scientist Dr Tim Lightfoot says “These may kids that have a very strong desire to be active, and maybe we are blunting it with medications”.

Interestingly, the 7R version of the DRD4 gene is more common in populations that have migrated long distances, as well as those that are nomadic, compared with settled populations. Scientists have therefore proposed that hyperactivity and impulsivity may have had advantages in the

ancestral state of man in nature, leading to the preservation of genes that increase ADHD risk.

As Dr Lightfoot says “there are biological (genetic) mechanisms that actually influence people to be active or not; you can have a predisposition to be a couch potato.”

15 – The Heartbreak Gene

This chapter looks at specific genetic mutations and how they impact the lives (and sometimes deaths) of sportsmen.

The first is a number of possible mutations that result in hypertrophic cardiomyopathy (HCM), a genetic disease that causes the walls of the left ventricle to thicken, such that it does not relax completely between beats and can impede blood flow into the heart itself. About 1 in every 500 people have HCM, though many will never exhibit serious symptoms. HCM is the most common cause of sudden death in young athletes.

In people with HCM, the muscle cells of the left ventricle, are not stacked neatly like bricks in a wall as they should be, but are all askew as if they had been dumped in a pile. When the electrical signal that cues the heart to flex travels across the cells, it is liable to bounce erratically. Intense athletic activity can trigger this short-circuit, which is especially dangerous during competition, when an athlete straining his body will not respond to the early signs of danger. People with HCM can be at increased risk of dropping dead because they exercise!

Till the early 90s, it was thought that HCM came from one of 7 different mutations on a single gene, MYH7 gene that codes for a protein found in heart muscle. However by now, we have discovered that there are as many as 1,452 different mutations across 18 different genes that can cause HCM. Most of the mutations are in genes that code for proteins found in heart muscle, and around 70% of those with HCM have a mutation on just one of two specific genes. The most common cause of HCM is a DNA spelling error known as “missense mutation”. A missense mutation occurs when a single letter is swapped in the DNA code, but in such an important place that it changes the amino acid that goes into making the resulting protein.

A definitive determination of HCM is important in athletes, because the most conspicuous sign of HCM is an enlarged heart, which is common in athletes. It takes a true HCM expert, of whom there are few left in the world to determine whether the enlargement is merely a training adaptation or a sign of HCM. This is why genetic testing is critical to determining HCM.

Typically once you are identified with HCM via genetic testing, then you are required to abstain from rigorous exercise, as the increase in adrenalin may spark a deadly heart rhythm. Often a defibrillator is planted in the chest – this is a matchbox-sized device waiting for an abnormal heart rhythm. It then fires an electrical shock to jolt the heart back to a normal pattern.

Another genetic trait that has a considerable health impact is getting a particular version of the ApoE gene, or Apolipoprotein E genes. It comes in three variants ApoE2, ApoE3 and ApoE4. Studies of Alzheimer's patients indicate getting an ApoE4 variant substantially increases an individual's risk of getting the disease. Everyone has two copies of the ApoE gene, one from each parent. A single ApoE4 copy increases the risk of Alzheimer's threefold. Two copies increase the risk eightfold. Around half of Alzheimer's patients have an ApoE4 gene and those who do, develop it at a younger age.

People who have an ApoE4 variant take longer to clear their brains of a protein called Amyloid, which floods in when the brain is injured. They tend to have longer comas, more bleeding and bruising in the brain, less success with rehabilitation and are more likely to suffer permanent damage or die. Sportsmen with ApoE4 variants take greater time to recover and are also at greater risk of suffering dementia later in life.

The chapter then details how mutations in genes such as COL1A1 and COL5A1 that code for the proteins that make up collagen fibrils, the basic building blocks of tendons, ligaments and skin. Collagen is sometimes referred to as the body's glue, holding connective tissues in proper form. People with a certain mutation in COL1A1 gene have brittle bone disease and suffer fractures easily, and a particular mutation in COL5A1 causes Ehlers-Danlos syndrome or extreme hyperflexibility. Ehlers-Danlos is rare but much more common variations in collagen genes influence both flexibility and an individual's risk of injuries to the connective tissues, like Achilles tendon rupture etc.

An extremely rare mutation in the SCN9A gene blocks pain signals that travel from the nerve cells to the brain, thus leading to congenital insensitivity to pain (typically such people do not live long as the lack of feedback leads to injuries and infections leading to an early death).

The gene that has been most studied for its involvement in pain modulation is the COMT gene, which is involved in the metabolism of neurotransmitters in the brain, including dopamine. Two common versions of COMT are known as 'Val' or 'Met' based on whether a specific part of the gene's DNA sequence codes for the amino acid valine or methionine. The Met version is less

effective at clearing dopamine, leaving higher levels in the front cortex. Subjects with 2 Met versions tend to do better at cognitive & memory tasks but are more prone to anxiety and more sensitive to pain (worriers). Anxiety or catastrophizing is a strong predictor of an individual's pain sensitivity. Conversely, Val/Val carriers do slightly worse on cognitive tests that require rapid mental flexibility, but may be more resilient to stress and pain (warriors). They also get a better boost from Ritalin, which increases dopamine in the frontal cortex.

In the U.S. 16% of people are Met/Met, 48% are Met/Val and 36% are Val/Val, making the case that both warriors and worriers are needed in every society, so there is widespread preservation of both forms of the gene.

Despite all of the above, there is also stress-induced analgesia – the brain's ability to block pain in high-pressure situations / conditions of acute stress. A system to block pain in extreme situations evolved in the genes of all humans, and sports settings tap into it.

Pain can be modified by a game situation or by the emotions of the athlete, but the genetic blueprint for pain in the body is encoded in the brain, whether or not that body even exists in its entirety (phantom limbs and pain). In fact while pain is innate it needs to be learned (Ronald Melzack's experiments with dogs that had been isolated from the outside world), and if we miss the window for acquiring the software, the hardware (genes) are of little use.

16 – The Gold Medal Mutation

This chapter details a mutation that confers a decisive advantage for participants in endurance sports. It illustrates it through the real-life story of Finn Eero Mantyranta, a 3-time gold medalist in the Winter Olympics in the 60s.

Eero Mantyranta had a mutation on the EPOR gene – erythropoietin receptor gene. This particular gene tells the body how to build the EPO receptor, a molecule that sits atop bone marrow cells awaiting the EPO hormone. If the EOP receptor is a keyhole, it is one made specifically to accept only the key that is the EPO hormone. Once the key is in the lock, the receptor signals the bone marrow cell to start the process of creating a red blood cells that contains haemoglobin.

The mutation on Eero's EPOR gene – a stop codon on position 6,002 of the total 7,138 bases that make up EPOR – meant that RNA (Ribonucleic acid) or the molecule that reads DNA code so that it can be translated into action was

told in this case that the instructions are finished, i.e., There is nothing more left here – move on. Thus the Mantyranta mutation caused the receptor simply to stop being built with over 15% of its construction unfinished.

The unfinished portion happens to be a segment of the receptor in the interior of the bone marrow cell, acting as a brake to halt haemoglobin production. In Eero's cells, missing this brake, the production of red blood cells runs amok.

Fortunately the overproduction of red blood cells did not lead to ill health. Instead by having haemoglobin levels of 20-23 gms of haemoglobin per decileter of blood (average is 14-17gms), he was able to get an undue advantage in his sport – Cross-country skiing. His increased haemoglobin levels (65% higher than the average man) did create suspicions of blood doping (taking a synthetic version of EPO) throughout his career till they were laid to rest by the finding of the EPOR mutation in the early 90s.

17 – The Perfect Athlete

Instances in which a single gene (or mutation) has a dramatic effect, as in Mantyranta's case are exceedingly rare, and finding athleticism genes is extraordinarily complex and difficult. But a present inability to pinpoint most sports genes doesn't mean they don't exist, and slowly scientists will find more of them.

One of the concerns held by Yannis Pitsiladis, the sports scientist, is that discovering genes that influence athletic performance will detract from the hard work undertaken by athletes; especially if those genes tend to be concentrated in one ethnic group or region than another. But we already know that some ethnic groups have genes that equip themselves superiorly or inferiorly for particular athletic endeavours (long thin legs for endurance running). Despite this, the opposite extreme - ignoring gifts as if they didn't exist – is much more common in the sports sphere.

Acknowledging the existence of talent and of genes that influence athletic potential in now way detracts from the work that it takes for that talent to be transformed into achievement. Athletes are essentially always distinguished by both their training environments and their genes.

In some cases, as with baseball hitters, a skill that seems based on superhuman reflexes is actually the result of a learned mental database. In others as we saw with the HERITAGE study, genes mediate the ability to respond rapidly to endurance exercise, and thus the improvements that come from hard training. In all likelihood, we overascribe our skills and traits to

either innate talent or training, depending on what fits our personal narratives.

As the study of genetics matures, we will increasingly find genetic inputs – some trivial, some critical – behind many sporting achievements. But we are unlikely to receive complete answers from genetics alone, and not merely because environment and training are critical factors. It is increasingly clear that many traits (as we saw with height) are influenced by the interplay of large numbers of DNA variations, requiring thousands of subjects (not always easy to get such a large number of elite athletes for testing) to get at the genetic roots of such traits. The rare single-gene mutations – like EPOR or Myostatin – that have outsized athletic impacts, have proven the exception.

Trying to engineer the perfect athlete doesn't seem possible in the foreseeable future. A genetically perfect athlete would have to luck into the right versions of the genes for her sport. A study done for endurance athletes by scientists Alan Williams and Jonathan Folland, found that the probability of ending up with two correct versions of the 23 gene variants that have thus far been most strongly linked to endurance talent was less than 1 in a quadrillion! Given the paltry 7 billion people on our planet, chances are that nobody has the ideal endurance profile for more than 16 of the 23 genes.

Just as there is no one-size-fits-all medicine to success in modern sports, science will continue to show that there is no one-size-fits-all training program. If one sport or training program isn't working for you, it may not be the training. It may be *you* in the deepest sense. Successful training plans may be as varied as the individuals who would undertake them.

As put by JM Tanner, hurdler and growth expert, “Everyone has a different genotype. Therefore, for optimal development...everyone should have a different environment.”

Happy training.