WHO HAS THE SPEED GENE, AND WHO DOESN'T?

HOW MUCH OF PERFORMANCE IS GENETIC?

HOW DID EARLY HUMANS BECOME ATHLETES?

AND CAN THE PERFECT ATHLETE BE GENETICALLY ENGINEERED?

SPORTS GENES

BY DAVID EPSTEIN

ILLUSTRATIONS BY BRYAN CHRISTIE
Remember the guy or girl in high school who just had it? He was all-everything at quarterback and high jump; she led the pitching rotation and was also the starting point guard. Naturals. Or were they? Did Ken Griffey Jr. inherit good genes, or did he become a superstar because he grew up in a clubhouse? Or both? For the price of a family outing at the ballpark, some companies will tell you if you have a certain sports gene. I have the sprinter gene, for instance, and you probably have it too.

So are there really such things as sports genes? If there are, do only elite athletes have them, or do they separate themselves from the rest of us more by their work ethic? And the work ethic—is there a gene for that?

There's only one place to look.

THE GENETIC PLAYBOOK

At the center of our every cell lies the twisting ladder of the double helix. A mere four molecules—adenine, cytosine, guanine and thymine—pair off three billion times to make up our DNA, the instruction manual for our bodies. Twenty-five thousand sections of each DNA ladder are especially important. They are genes, and it is they that actually tell the body how to build itself.

In April 2003 an international consortium of scientists announced the completion of the Human Genome Project. After 13 years of work (and 200,000 years of modern man), the project had mapped all 25,000 regions of our every gene. Twenty-lion times to make up our DNA, the human genome; all 25,000 regions of our genetic material were published in all of us, from All-Pro NFL running backs or Olympic swimmers who have been examined in 2004 by biologists Dennis Bramble of the University of Utah and Daniel Lieberman of Harvard. Their conclusion contradicted the common assumption that East African dominance of distance runners is a by-product of their superior endurance. They argued that those who could jog in the punishing equatorial heat could beat the hyenas to a carcass. They could survive another day, perhaps long enough to have children.

The major changes that took hold in the body over the next half-million years were examined in 2004 by biologists Dennis Bramble of the University of Utah and Daniel Lieberman of Harvard. Their conclusion contradicted the common assumption that human running was simply a by-product of walking. Nearly every one of the major anatomical changes can be traced back to our ancestors, the professors argued, to make him the hot-weather endurance running champion of the savanna.

There is, for instance, the rubbery neck ligament that acts like a shock absorber for the head during running; the gill of sweat glands to help keep the body cool while running; the lack of body fur for the same reason; shoulders that move, unlike in apes, independently from the neck so that the arms can swing while the head remains still; long legs and narrow waists; larger surface areas in hip, knees and ankle joints, again for improved shock absorption; short toes, which are better for pushing off than for grasping tree branches; an arched foot, which acts as a spring; and big butt muscles to keep us upright. “Have you ever looked at an ape? They have no buns,” Bramble says. “We think running is one of the most transforming events in human history.”

No longer content merely to scavenge, our ancestors, despite having no greater weapons than sticks and stones, became deadly hunters. They overwhelmed their perspirationally challenged quarry with a methodical chase that lasted until the

Their forest home had begun to give way to hot, dry savannas, with few trees and with grass short enough to give sight lines that stretched until the earth curved away. Our forebears saw for the first time the hordes of wildebeest and antelope that filled the plain.

Gradually these ancient, mostly vegetarian primates dropped from the trees and went looking for steak. Initially they might have used vultures as their guides, racing hyenas to scavenge the leftover brains and bone marrow of dead antelopes. For the first time in history a two-legged mammal had reason to run long. Those who could jog in the punishing equatorial heat could beat the hyenas to a carcass. They could survive another day, perhaps long enough to have children.

Their journey to the center of our genetic material is the story of how we evolved into something different.
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And so do billions of people worldwide, but Wisconsin - La Crosse and co-author of the paper was startled by his preliminary findings. In words, is understand what makes the runner better an individual’s aerobic capacity than his opponent. In fact, it seems that all an ACTN3 test can do consistently is tell someone he’s not going to make the Olympic 4 x 100 relay team. But even that sweeping conclusion leaves room for exceptions, such as the Spanish long jumper who also has them yet sailed more than 27 feet and twice made the Olympics.

“It was a wild ride,” says Carl Foster, director of the Human Performance Laboratory at Wisconsin–La Crosse and co-author of several ACTN3 studies. “If you want to know if your kid is going to be fast, take him to the playground and have him race the other kids.”

The case of the curiously alpha-actinin-3-deficient long jumper means that running and jumping are influenced by a tangled skein of genes, not to mention training. So far, around two dozen genes have made strong scientific cases for inclusion in the equation of explosiveness, as have about two dozen for endurance. What Pitsiladis is trying to do, in his words, is understand the other gene that makes the perfect athlete. He and colleagues have identified thus far, is genetically perfect. He and colleagues have found that a particular variant of the ApoE gene is associated with increased risk of the disease. Each person has two copies of the ApoE gene. (Each one is either ApoE2, ApoE3 or ApoE4.) People with one copy of ApoE4 have a three- to fourfold increased risk of Alzheimer’s, while people with two ApoE4 copies have eight times the risk. Alzheimer’s patients with ApoE4 variants also tend to show signs of dementia at an earlier age than Alzheimer’s patients who do not have the variant.

The more ApoE has been studied, though, the more it has been associated not only with Alzheimer’s but also with the ability of the brain to heal following head injury. People with ApoE4 variants who hit their heads in car accidents, for example, are more likely to have permanent damage or to die than those who have other variants. And a series of small studies suggests that athletes with ApoE4 variants who get hit in the head are more likely to recover slower and to suffer greater dementia later in life. It is not entirely clear how ApoE affects brain recovery, but the gene is involved in the inflammatory response of the brain after injury. ApoE4 appears to appear and tell the brain’s cleargen of a particular protein called amyloid, which flies with ApoE4 and thus blocks the way.

A 1997 study of 30 boxers found that those who had taken a blow to the head and had an ApoE4 copy scored significantly worse on tests of brain impairment than similar fighters who did not have an ApoE4 copy. The ApoE4 variant is present in less than 50% of the general population, but it was present in all three of the boxers in the study who were severely impaired. A 2000 study of 32 active pro football players concluded that three factors caused some players to score lower than others on tests of brain function: 1) age, 2) having been hit in the head a lot and 3) possessing an ApoE4 variant.

Last year, during the NFL’s concussion controversy, doctors from Boston University made news with research on dozens of cases of brain damage in deceased football players and boxers. What escaped the news was the genetic data the researchers had for nine of the athletes. Five of them, or 56%, had at least one ApoE4 variant, more than twice the proportion found in the general population.

Two years after the study of the 53 football players, Barry Jordan, one of its authors and until a year ago the chief medical officer of the New York State Athletic Commission was praised by researchers and coaches as a pioneer in screening for all boxers in the state but then backed off. Doctors agree that more work is needed to understand how ApoE4 affects brain recovery before a genetic test should become common practice. Jordan and James P. Kelly, a neurologist on the Colorado State Boxing Commission, cited two other arguments against offering an ApoE test to athletes: first, teams and insurance companies might unfairly discriminate against an athlete with a certain gene; second, to tell someone he has an ApoE4 variant is to tell him about his risk of developing Alzheimer’s later in life, information he might not want to know.

“With ApoE4, some would argue that knowledge is not power,” Kelly says.

THE UNCOMMON THREAD

IN A FEW CASES SINGLE GENETIC MUTATION HAS BEEN DIRECTLY LINKED TO HEIGHTENED ATHLETICISM

Stephen M. Roth, an associate professor of kinesiology at Maryland, is among a group of scientists who track all the published work on potential genetic changes and compiled a list known as the Human Gene Map for Performance. Roth notes that even seemingly straightforward athletic qualities, such as speed and strength, are highly complex, influenced as they are by multiple genetic and environmental factors. “With any single gene that seems to have a positive effect on performance, the effect is very subtle,” Roth says. There are, however, a few documented cases in which a single rare genetic mutation has hugely increased an individual’s athleticism.

-Euro Martinez is a former cross-country skier who won three gold, two silver and two bronze medals for Finland in three winter Olympics in the 1960s. But suspicions of blood doping cast a pall over his achievements, because he had 25% more oxygen-carrying red blood cells than his competitors despite his claim that his training was no different from theirs. Three decades later, in 1993, Finnish scientists published the results of a study that examined five generations of his family. The study found that a rare mutation on the EPON gene runs in the Martinez family, causing an exocrine response to erythropoietin, or EPO, a hormone that spurs red-cell production. In essence, Martinez and 13 other relatives who were studied have naturally what some athletes gain only by cheating with EPO injections. Not surprisingly, the family has a number of accomplished endurance athletes.

-RFU player Ron Duguay, famous in the 1980s for playing without a helmet, and even at age 50, when he still had a 92 mph fastball, is an ApoE4 carrier.

-In 2008 Pitsiladis tested Colin Jackson, a British former world-record holder in the 110-meter hurdles, for ACTN3. “He had the ‘wrong’ copies of the explosive-ness gene, or the Spanish long jumper who added, “the best genetic test right now is a stopwatch. Take him to the playground and have him race the other kids.”

With ApoE4, some would argue that knowledge is not power.”

KELLY SAYS.
Part of the concern over the insurance issue was allayed last year when the federal Genetic Information Nondiscrimination Act took effect, barring employers and insurance companies from discriminating on the basis of genetic information. And research at Boston University has found that people who volunteer for an ApoE screen do not feel undue dread if they find out they have the deleterious variant. In fact, they usually embrace lifestyle habits, such as exercise, that doctors tell them might decrease their Alzheimer’s risk. “This is a very controversial area,” says Robert C. Green, a BU neurologist who helped conduct the work.

“The world of genetics for decades has suggested that there’s no reason to give people genetic-risk information unless there’s something proven you can do about it.”

For athletes facing head trauma, perhaps there are some relatively painless actions they could take. “If this gene is how you’re describing it,” says Duguay, whose test showed that he has one ApoE4 copy, “and I knew I had it when I was playing, I would’ve seriously considered wearing a helmet.”

Glen Johnson, a 41-year-old boxer with a 50-13-2 record, including wins over Roy Jones Jr. and Antonio Tarver, says he was considering retiring after his November loss to Chad Dawson. The older Johnson gets, the more he wants every bit of information that can tell him about what his life might be like after he quits fighting. Johnson—who understands that it isn’t a particular gene variant, but rather getting hit in the head—that is the key factor in brain injury—has already put his relatives and friends on alert to tell him if they notice any differences in his speech or memory. “I’d have to get a better understanding of [ApoE4], and

You had the talent to be a sports star, but you lacked the discipline. You couldn’t bring yourself to go to bed early or pick up a barbell instead of an Oreo. On the bright side, your kid gets to start from square one: He or she inherits your talent but not the repercussions of your lackluster approach.

Or maybe not. Research in the growing science of epigenetics—which, among other things, looks at how genes can be switched on and off—suggests that your actions could have genetics-related consequences for your child. Better think twice before pounding down that next cookie.

At the turn of the 19th century, French naturalist Jean-Baptiste Lamarck proposed a theory of evolution that said animals quickly adapt to their environment and pass the adaptations along to their progeny. For example, giraffes acquired long necks because their ancestors stretched to reach leaves high on trees. But Lamarckian evolution was eclipsed in 1859 when Charles Darwin published his theory of evolution by natural selection, which says traits are acquired over millions of years as random genetic changes that happen to be beneficial are passed on. Today, however, the idea at the heart of Lamarck’s theory—that our choices can affect our children’s genes—is making a comeback.

Your behavior will not change the sequence of your child’s DNA, but it might change the action of certain molecules—called epigenetic marks—that attach to DNA and signal genes to turn on and off. Consider a study, published in 2008, of adults in the Netherlands whose mothers had suffered through a German-imposed food embargo in the winter of 1944–45. Six decades later, those whose mothers were in the early stages of pregnancy during the Dutch Hunger Winter had fewer “turn off” signaling methyl molecules attached to their IGF2 genes than did their siblings. The IGF2 gene is a key component in growth and development, and the study may help explain why children of mothers who experience famine during pregnancy have a higher risk of developing obesity, schizophrenia and diabetes later in life.

Studies now under way should further illuminate the epigenetic link to sports prowess. In one study published last year, rats that exercised regularly and rats that didn’t were twice thrown into beakers of water. On the first go-round, all the rats struggled vigorously to swim and attempt to escape the beaker. A day later, when they were again put into the water, the regularly exercised rats displayed better stress coping mechanisms: Instead of clawing at a glass wall they could not climb, these rats, having learned from the previous day’s experience, conserved energy by floating. When their brains were examined afterward, researchers found that the exercised rats had altered epigenetic marks that in turn affected gene expression in a part of the brain that helps form memories. “Exercise has a great impact on the brain, not just the muscles,” says Johannes Reul, one of the researchers and a neuroscientist at the University of Bristol, England.

Perhaps one day a similar epigenetic explanation of memory will help us understand why great cornerbacks don’t get beat the same way twice.

—D.E.
I’d take a lot of other things and tests into consideration when I think about fighting again,” Johnson says, “but I’d never hide from extra information.”

**FINDING THE PERFECT ATHLETE**

I

PITSILADIS IS to pinpoint the athletically perfect genetic specimen, he or she must first exist. Just how many of these folks might have stepped right off Mount Olympus is a question that kept Alun Williams awake two years ago. Williams, a geneticist at Manchester Metropolitan University in England, and a colleague pored over the scientific literature and chose the 23 genetic variants that have been most strongly associated with talent in endurance sports. The scientists gathered information about the variants’ prevalence—some are found in more than 80% of people and others in fewer than 5%—and made statistical projections of how many “perfect” endurance athletes (people with two “correct” variants of each of the 23 genes) stride the earth.

Williams figured that perfection would be rare. After all, a Lance Armstrong comes around only once in a lifetime. But Williams was shocked when he ran the algorithm on his computer and saw that the odds of any person having all the right gene variants for endurance were less than one in a quadrillion. That’s one followed by 15 zeroes. Think of it this way: If you pony up for 20 tickets each week, you’d have a better chance of winning the Mega Millions twice in a row than of hitting this genetic jackpot.

The bottom line is that even Lance isn’t a perfect specimen. Based on only the 23 chosen genes, there’s almost certainly no genetically perfect athlete alive. In fact, given that a measly 6.8 billion people live on our planet, chances are that nobody has the ideal endurance profile for more than 16 of the 23 genes. An individual is also unlikely to have only a few of them. Essentially everybody falls in the muddled middle, differing by only a handful of genes. It’s as if we’ve all played genetic roulette over and over, moving our chips around, winning sometimes and losing sometimes and gravitating toward mediocrity. “We’re all relatively similar because we’re relying on chance,” Williams says.

But if anyone is the beneficiary of a long genetic winning streak, it should be a world-record holder, shouldn’t it? Pitsiladis selected 24 gene variants most often associated with sprinting or endurance prowess and looked for them in the genomes of four men who have held the world record in the 100-meter dash and five who have held the world record in the marathon. What he saw was that based on those genes, the world-beaters are not genetic outliers at all. Pitsiladis analyzed the DNA of some of his graduate students for comparison and found that “a student of mine has a better rating for sprinting than the likes of an Asafa Powell or Usain Bolt.” (Pitsiladis is legally prohibited from identifying specific athletes with their genetic material, so he used Powell and Bolt as rhetorical examples.)

That rather startling result leaves two broad possibilities: First, there is a tremendous amount of work left to be done to find all the remaining genes that contribute to athletic success; second, something other than genetics is at work. Both may well be true, but only time and more research will rule on the former, while Pitsiladis has compiled considerable data on the latter.

Some of the most intriguing work comes from his study of the demographics of elite East African distance runners. When Pitsiladis analyzed Kenyan runners, he found that three quarters of all elite international competitors were from a single tribe, the Kalenjin, who make up a mere 10% of Kenya’s population. At first blush that would seem to indicate a genetic advantage in the Kalenjin, but Pitsiladis also found that they were likely to be living and training at altitude in the Rift Valley. When Pitsiladis compared 400 elite Kenyan athletes with a group of randomly selected Kenyans, he found that as children, the athletes were more likely to have lived at least several miles from school, and much more likely to have had to run there and back. Eighty-one percent of the elite Kenyan runners he studied had to rely on their feet to get to and from school, compared with only 22% of the control group. One 10-year-old boy whom Pitsiladis tested last year was already such an experienced runner that he clipped off six-minute miles when Pitsiladis tested him on a dirt track.

Pitsiladis and his colleagues found a similar pattern in Ethiopia. What was not shared between the Ethiopians and the Kenyans, however, was a particularly large proportion of their genes.

The maternal line of DNA has been found to be more influential in endurance than the paternal, so Pitsiladis’s group analyzed that genetic material and found that Ethiopian and Kenyan athletes “could not be more different genetically,” Pitsiladis says. The Ethiopian athletes, for example, were much more likely to have blocks of gene variants common in Europe and Asia than were the Kenyan athletes. Pitsiladis’s conclusion is that whatever specific genes are necessary for endurance, they aren’t exclusive to either Ethiopians or Kenyans.

His work suggests that some sports phenomena that seem on the surface to be entirely based in genetics might not be. (Or at least not in the way we’re used to thinking about genetics. A newer science called epigenetics is unraveling how environment and behavior, such as exercise, can actually turn particular genes on and off in patterns that might be passed on through generations.)

Similarly counterintuitive conclusions about the interplay between nature and nurture have come from outside sports: African-Americans are more prone than white Americans to hypertension, but the trend is not found among black people in some of the countries from which black Americans came, such as Jamaica and Nigeria. That points to the U.S., not to genes associated with blackness, as a culprit.

This is not to say that all ethnicities are the same. Nigerians are known for sprint-
LOVAZA is a prescription medication, called a lipid-regulating drug, for adults.

It is used for people who need treatment to lower high levels of blood fats.

Before you start taking it, and each time you get a refill, there may be

A special guideline to remember is that your body needs but cannot produce itself.

Telling your doctor about all of your medical conditions that your body needs but cannot produce itself.

Especially tell your doctor if you take medicines:

• To reduce clotting—known as anticoagulants or blood thinners.
• One of the tests to check liver function (ALP).
• To check blood counts and platelet counts.
• To check serum creatinine and blood urea nitrogen.

Your doctor or pharmacist will give you the latest information about the specific dose of each drug you need.

It may change:

• The amount of liquid that you drink.

What is LOVAZA?

LOVAZA is used along with a low-fat and

The HERITAGE Family Study is con-

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the HERITAGE Family Study is con-

It may change:

• Your body needs but cannot produce itself.

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A special guideline to remember is that your body needs but cannot produce itself.

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The Chinese basketball federation) is

If not, say, Floyd Mayweather Jr.—

The HERITAGE Family Study is con-

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The most common side effects with LOVAZA are:

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