



CONDITIONS

INJURIES	03
BODY COMPOSITION	38
NUTRITION	74
TRAINING	119
RECOVERY	180
ANALYTICS	211
SUPPLEMENTATION	260
PERFORMANCE	314

NOTES

The following results are based on current studies and scientific evidence; however, they are not to be taken as a diagnosis of any genetic disease. Added to this, results point only to tendencies, not in any way stating that a person with a higher risk of injury, for instance, will surely suffer from one, nor that it will never happen to a person with a lower risk.

BROWSING YOUR RESULTS



Attention Risky area



Average
Within population's standard results

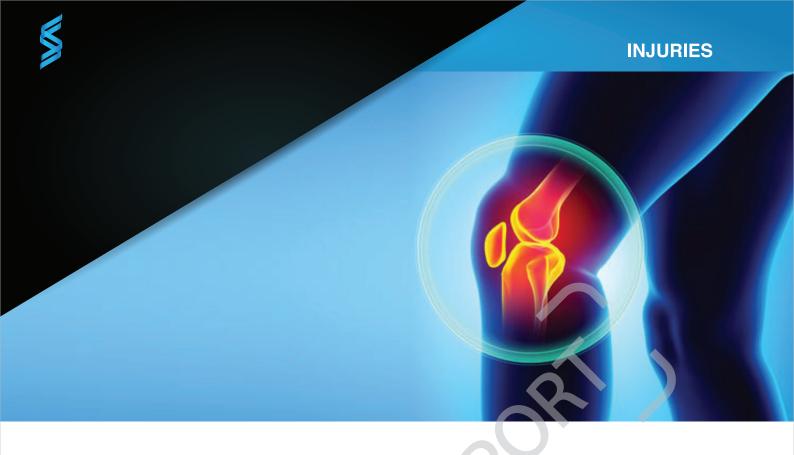


Above average
Positive Highlight

In case you have any questions concerning your results, please contact us through our website or e-mail:

www.gensportspro.com · info@gensportspro.com

> ANTERIOR CRUCIATE LIGAMENT TEAR	03
> MENISCUS INJURIES	06
> ANKLE SPRAIN	09
> ACHILLES TENDINOPATHY	12
> EPICONDYLITIS (TENNIS ELBOW)	15
> CARPAL TUNNEL SYNDROME	18
> SHOULDER DISLOCATION	21
> LUMBAR DISK DEGENERATION	24
> PERIODONTAL HEALTH	<mark>27</mark>
> STRESS FRACTURES	30
> MUSCLE CRAMPS	33



ANTERIOR CRUCIATE LIGAMENT TEAR



MY GENETICS

Your genetic composition, in comparison with the general population, is associated with being 1.5 TIMES MORE LIKELY to suffer a TEAR OF THE ANTERIOR CRUCIATE LIGAMENT in the knee.

WHY ANALYSE A TORN ANTERIOR CRUCIATE LIGAMENT?

The anterior cruciate ligament (ACL) is essential in sports practice and a tear in this ligament (snapping sensation) occurs during physical activity. Its function is to stabilise the knee when it is turning.

Knowledge of the athlete's individual tendency to suffer from this type of injury may be of interest due to its serious nature (highly disabling) and the risk certain exercises in training present: specific strength training phase; contact with the floor during a jumping routine (plyometric training); some cross-training (skiing, etc.).



WHAT DOES OPTIMAL LIGAMENT RESISTANCE MEAN IN PRACTICE FOR THE ATHLETE?

- > Ability to better withstand traction without the ligaments losing their functional nature.
- > Provides the joint with stability, allowing it to bend and extend.
- **> Reduction in the effect** that mechanical stress, derived from exercise, has on the ligaments and, therefore, a reduction in the risk of suffering from an injury related to this area.

+INFO FOOTBALL

To know more, consult the following chapters in your Informative Manual:

- > Torn anterior cruciate ligament.
- > Non-contact injuries.
- > Injury prevention. Recommendations.

COL5A1 - C:C

GENE FUNCTION

> COL5A1: COLLAGEN TYPE V ALPHA 1 CHAIN

18%

This gene encodes an alpha chain for one of the low abundance fibrillar collagens. Fibrillar collagen molecules are trimers that can be composed of one or more types of alpha chains. Type V collagen is found in tissues containing type I collagen and appears to regulate the assembly of heterotypic fibers composed of both type I and type V collagen. This gene product is closely related to type XI collagen and it is possible that the collagen chains of types V and XI constitute a single collagen type with tissue-specific chain combinations. The encoded procollagen protein occurs commonly as the heterotrimer pro-alpha1(V)-pro-alpha1(V)-pro-alpha2(V). Mutations in this gene are associated with Ehlers-Danlos syndrome, types I and II. Alternative splicing of this gene results in multiple transcript variants. [provided by RefSeq, May 2013]. NCBI.

COL1A1 - G:T

GENE FUNCTION

> COL1A: COLLAGEN TYPE I ALPHA 1 CHAIN

29%

This gene encodes the pro-alpha1 chains of type I collagen whose triple helix comprises two alpha1 chains and one alpha2 chain. Type I is a fibril-forming collagen found in most connective tissues and is abundant in bone, cornea, dermis and tendon. Mutations in this gene are associated with osteogenesis imperfecta types I-IV, Ehlers-Danlos syndrome type VIIA, Ehlers-Danlos syndrome Classical type, Caffey Disease and idiopathic osteoporosis. Reciprocal translocations between chromosomes 17 and 22, where this gene and the gene for plate-let-derived growth factor beta are located, are associated with a particular type of skin tumor called dermatofibrosarcoma protuberans, resulting from unregulated expression of the growth factor. Two transcripts, resulting from the use of alternate polyadenylation signals, have been identified for this gene. [provided by R. Dalgleish, Feb 2008]. NCBI.

COL3A1 - A:A

GENE FUNCTION

> COL3A1 COLLAGEN TYPE III ALPHA 1 CHAIN

6%

This gene encodes the pro-alpha1 chains of type III collagen, a fibrillar collagen that is found in extensible connective tissues such as skin, lung, uterus, intestine and the vascular system, frequently in association with type I collagen. Mutations in this gene are associated with Ehlers-Danlos syndrome types IV, and with aortic and arterial aneurysms. Two transcripts, resulting from the use of alternate polyadenylation signals, have been identified for this gene. [provided by R. Dalgleish, Feb 2008] NCBI.



FBN2 - G:G

GENE FUNCTION

> FBN2 FIBRILLIN 2



The protein encoded by this gene is a component of connective tissue microfibrils and may be involved in elastic fiber assembly. Mutations in this gene cause congenital contractural arachnodactyly. [provided by RefSeq, Jul 2008] NCBI.

COL1A1 - A:A

GENE FUNCTION

> COL1A: COLLAGEN TYPE I ALPHA 1 CHAIN

1%

This gene encodes the pro-alpha1 chains of type I collagen whose triple helix comprises two alpha1 chains and one alpha2 chain. Type I is a fibril-forming collagen found in most connective tissues and is abundant in bone, cornea, dermis and tendon. Mutations in this gene are associated with osteogenesis imperfecta types I-IV, Ehlers-Danlos syndrome type VIIA, Ehlers-Danlos syndrome Classical type, Caffey Disease and idiopathic osteoporosis. Reciprocal translocations between chromosomes 17 and 22, where this gene and the gene for platelet-derived growth factor beta are located, are associated with a particular type of skin tumor called dermatofibrosarcoma protuberans, resulting from unregulated expression of the growth factor. Two transcripts, resulting from the use of alternate polyadenylation signals, have been identified for this gene. [provided by R. Dalgleish, Feb 2008]. NCBI.

FBN2 - G:G

GENE FUNCTION

> FBN2 FIBRILLIN 2



The protein encoded by this gene is a component of connective tissue microfibrils and may be involved in elastic fiber assembly. Mutations in this gene cause congenital contractural arachnodactyly. [provided by RefSeq, Jul 2008] NCBI.

ACAN - G:G

GENE FUNCTION

> ACAN AGGRECAN



This gene is a member of the aggrecan/versican proteoglycan family. The encoded protein is an integral part of the extracellular matrix in cartilagenous tissue and it withstands compression in cartilage. Mutations in this gene may be involved in skeletal dysplasia and spinal degeneration. Multiple alternatively spliced transcript variants that encode different protein isoforms have been observed in this gene. [provided by RefSeq, Jul 2008] NCBI.

MMP12 - A:A

GENE FUNCTION

> MMP12 MATRIX METALLOPEPTIDASE 12



This gene encodes a member of the peptidase M10 family of matrix metalloproteinases (MMPs). Proteins in this family are involved in the breakdown of extracellular matrix in normal physiological processes, such as embryonic development, reproduction, and tissue remodeling, as well as in disease processes, such as arthritis and metastasis. The encoded preproprotein is proteolytically processed to generate the mature protease. This protease degrades soluble and insoluble elastin. This gene may play a role in aneurysm formation and mutations in this gene are associated with lung function and chronic obstructive pulmonary disease (COPD). This gene is part of a cluster of MMP genes on chromosome 11. [provided by RefSeq, Jan 2016] NCBI.

SCIENTIFIC EVIDENCE





+ GENETIC INFO

MY ANALYSIS

To date, scientific evidence regarding the genetic factors associated with the risk of suffering a torn anterior cruciate ligament is supported by dozens of scientific studies involving thousands of participants of both genders, mainly of European origin, including an active and sedentary population. Determination of injuries was undertaken by specialist doctors.

Some of these genetic associations have been replicated in more than one population and they include genes related to the composition and properties of collagen fibres. More specifically, they are related to regulation of the collagen fibril diameter, lateral growth during fibrillogenesis, adjustments in the connective tissue's strength and elasticity, and the bundling of elastic fibres in the regeneration process to provide the tissues with strength and elasticity.



MENISCUS INJURY



Your percentage



Attention Risky area

MY GENETICS

Your genetic composition is associated with being 1.3 TIMES MORE LIKELY to suffer a TORN MENISCUS and being likely to experience POORER POST-SURGERY RECOVERY in knee function than the recovery reported for the rest of the population.

WHY ANALYSE MENISCUS INJURIES?

The menisci are two fibrocartilaginous structures (internal and external) situated in the knee joint. The internal meniscus is injured more frequently. Their function is to stabilise the knee, absorbing the impacts that occur during exercise.

Knowledge of the athlete's individual tendency to suffer from this type of injury may be of interest due to its disabling effect (pain, inflammation, joint locking) as well as the risk certain exercises in training may represent.



WHAT DOES OPTIMAL MENISCI FIBROCARTILAGINOUS RESISTANCE MEAN IN PRACTICE FOR THE ATHLETE?

- > Protects the cartilage from excessive joint wear and tear (arthrosis).
- > Provides cushioning against compression and torsion forces that impact the knee (distribution of forces).
- > Helps stabilise the knee joint.
- **> Reduction** in the effect that mechanical stress, derived from exercise, has on the menisci and, therefore, a reduction in the risk of suffering from an injury related to this area.

+INFO FOOTBALL

To know more, consult the following chapters in your Informative Manual:

- > Torn anterior cruciate ligament.
- > Non-contact injuries.
- > Injury prevention. Recommendations.

GDF5 - T:T

GENE FUNCTION

> GDF5 GROWTH DIFFERENTIATION FACTOR 5

This gene encodes a secreted ligand of the TGF-beta (transforming growth factor-beta) superfamily of proteins. Ligands of this family bind various TGF-beta receptors leading to recruitment and activation of SMAD family transcription factors that regulate gene expression. The encoded preproprotein is proteolytically processed to generate each subunit of the disulfide-linked homodimer. This protein regulates the development of numerous tissue and cell types, including cartilage, joints, brown fat, teeth, and the growth of neuronal axons and dendrites. Mutations in this gene are associated with acromesomelic dysplasia, brachydactyly, chondrodysplasia, multiple synostoses syndrome, proximal symphalangism, and susceptibility to osteoarthritis. [provided by RefSeq, Aug 2016] NCBI.

SCIENTIFIC EVIDENCE







+ GENETIC INFO

MY ANALYSIS

To date, scientific evidence regarding the genetic factors associated with the risk of suffering a meniscus injury is supported by two unique scientific studies involving more than 500 male participants of Asian origin, including Chinese soldiers and civilians aged 20, of average height (170 cm) and weight (66 kg), with a body mass index of 22,2.

Injury determination was carried out by specialist doctors using, among other techniques, the results of nuclear magnetic resonance and diagnosis via arthroscopy.

This association includes the GDF5 gene and it relates to the fibrocartilaginous structures' capacity for development, regeneration and resistance as this is a growth factor that acts by regulating bone and joint development, where it plays an important role in tendon and ligament regeneration.

With regard to the level of scientific evidence on this association, you will have observed that we have only classified it with two stars. This is owed to the fact that although research performed to date agrees with the expected conclusions in terms of the function of GDF5, only a physically active Asian population has been analysed.

In the future, you will receive updated results for a European population thanks to the research project we are undertaking via the Gendurance Project, in collaboration with the University of the Basque Country and a high number of athletes, on the influence of the GDF5 gene in the likelihood of suffering from a meniscus injury.

> BONE QUALITY	39
> BONE MINERAL DENSITY	42
> CORTICAL BONE VOLUME	45
> MUSCLE MASS	48
> FAST-TWITCH MUSCLE FIBRES	51
> SLOW-TWITCH MUSCLE FIBRES	54
> VASTUS LATERALIS LENGTH	57
> SUBCUTANEOUS FAT MASS	60
> TOTAL FAT MASS	63
> ABDOMINAL GIRTH	66
> OBESITY TENDENCY	69
> ELASTICITY	71



BONE QUALITY



Your percentage



Average
Within population's standard results

MY GENETICS

Your genetic composition is associated with a tendency to present BONE QUALITY SIMILAR to the bone quality described for the general population.

WHY ANALYSE BONE QUALITY?

This term refers to a new concept that is included in current assessments of osteoporosis, allowing for an assessment of the bone beyond strictly quantitative measures (bone amount) and via an approach that includes factors that are directly involved in bone resistance, such as bone remodelling (regulated by hormonal and mechanical factors), geometry, architecture and mineralisation.

Therefore, it will allow for more information when establishing the risk level of a fracture occurring as other variables different to bone density are considered. These include age (against the same bone density figures) and the therapeutic effect of medicines, which decrease the risk of fracture without altering bone density values, acting on bone remodelling, for example.



WHAT DOES GREATER BONE QUALITY?

- **> Lower risk** of suffering from osteoporosis and, therefore, bone fractures due to this cause.
- > Reduction in the effect that mechanical stress, derived from exercise, has on the bones and, therefore, a reduction in the risk of suffering a bone fracture due to stress or strain.

+INFO FOOTBALL

To know more, consult the following chapters in your Informative Manual:

> Bone tissue

COL1A1 - G:T

GENE FUNCTION

> COL1A: COLLAGEN TYPE I ALPHA 1 CHAIN

This gene encodes the pro-alpha1 chains of type I collagen whose triple helix comprises two alpha1 chains and one alpha2 chain. Type I is a fibril-forming collagen found in most connective tissues and is abundant in bone, cornea, dermis and tendon. Mutations in this gene are associated with osteogenesis imperfecta types I-IV, Ehlers-Danlos syndrome type VIIA, Ehlers-Danlos syndrome Classical type, Caffey Disease and idiopathic osteoporosis. Reciprocal translocations between chromosomes 17 and 22, where this gene and the gene for platelet-derived growth factor beta are located, are associated with a particular type of skin tumor called dermatofibrosarcoma protuberans, resulting from unregulated expression of the growth factor. Two transcripts, resulting from the use of alternate polyadenylation signals, have been identified for this gene. [provided by R. Dalgleish, Feb 20081. NCBI.

COL1A1 - A:A

GENE FUNCTION

> COL1A: COLLAGEN TYPE | ALPHA 1 CHAIN

This gene encodes the pro-alpha1 chains of type I collagen whose triple helix comprises two alpha1 chains and one alpha2 chain. Type I is a fibril-forming collagen found in most connective tissues and is abundant in bone, cornea, dermis and tendon. Mutations in this gene are associated with osteogenesis imperfecta types I-IV, Ehlers-Danlos syndrome type VIIA, Ehlers-Danlos syndrome Classical type, Caffey Disease and idiopathic osteoporosis. Reciprocal translocations between chromosomes 17 and 22, where this gene and the gene for platelet-derived growth factor beta are located, are associated with a particular type of skin tumor called dermatofibrosarcoma protuberans, resulting from unregulated expression of the growth factor. Two transcripts, resulting from the use of alternate polyadenylation signals, have been identified for this gene. [provided by R. Dalgleish, Feb 2008]. NCBI.

SCIENTIFIC EVIDENCE * * *







+ GENETIC INFO

MY ANALYSIS

To date, scientific evidence regarding the genetic factors associated with intrinsic bone quality is supported by a series of scientific studies involving thousands of participants of both genders, of European origin, and including a middle-aged population.

Determination of injuries was undertaken by specialist doctors.

The genetic association observed includes the COL1A1 gene, which encodes the mainly protein component of tendons and ligaments, as well as bone structure. In addition, this gene was previously associated in dozens of scientific studies with a wide variety of sports injuries.

In particular, these relate to associations made between polymorphisms of the COL1A1 gene and bone quality, meaning an effect on the risk of bone fracture was observed, not simply due to its action on bone mineral density, but also due to its action on the elastic properties of the bone tissue. In short, these are polymorphisms that appear to affect complex mechanisms related to changes in bone mass and quality.



> LACTOSE INTOLERANCE	75
> GLUTEN INTOLERANCE	78
> WEIGHT LOSS - DIET LOW IN CARBOHYDRATES / HIGH IN FAT	91
> WEIGHT LOSS - LOW IN FAT / HIGH IN CARBOHYDRATES	94
> WEIGHT LOSS - BALANCED DIET	97
> WEIGHT LOSS - MEDITERRANEAN DIET	100
> WEIGHT GAIN - HIGH CONSUMPTION OF CARBOHYDRATES	103
> WEIGHT GAIN - HIGH CONSUMPTION OF FAT	106
> FOOD PREFERENCES	109
> APPETITE REGULATION	112
> HYPERTENSION SALT SENSITIVITY	115



LACTOSE INTOLERANCE



Your percentage



Attention Risky area

MY GENETICS

Your genetic composition is associated with a tendency to present a HIGH RISK of suffering from LACTOSE INTOLERANCE to a greater or lesser degree.

WHY ANALYSE LACTOSE INTOLERANCE?

"The result of this analysis is aimed at primary lactose intolerance that is permanent and has genetic causes."

Lactose is the main carbohydrate present in milk and its by-products (cheese, ice cream, yoghurt, dairy desserts, etc.). Lactose intolerance is a very widespread alteration in the global population and it stems from the inability of the small intestine to digest lactose due to an absence or deficiency of lactase (enzyme that transforms lactose into simpler sugars to facilitate its intestinal absorption).

The most frequent symptoms of this intolerance are gas, pain, abdominal distension, nausea, vomiting, diarrhoea, etc. Less specifically, it may cause tiredness, breakouts of the skin, muscle and joint pain, etc.

An early diagnosis of this intolerance is important, mainly as a lack of awareness, and therefore a delay in the adoption of suitable dietary measures, may cause more serious complications (injuries to the intestinal mucosa).



An athlete with a lactose intolerance must strictly control his or her diet. This is because the appearance of the symptoms described, even if to a lesser degree, may notably alter the normalised practise of physical exercise.

As such, if it is suspected that the athlete suffers from this intolerance, it is important that the appropriate study is carried out and, if it is confirmed, the pertinent restrictive dietary measures are adopted.

MY SPORT

A HIGHER LACTOSE TOLERANCE MEAN IN PRACTICE FOR THE ATHLETE?

> Consumption of products (milk, dairy by-products, protein supplements, etc.) that contain this carbohydrate without suffering greatly from the adverse effects (gastrointestinal symptomatology) that may impact the athlete's health and sports performance.

+INFO FOOTBALL

To know more, consult the following chapters in your Informative Manual:

Lactose intolerance

LCT - C:C

GENE FUNCTION

> LCT LACTASE

31%

The protein encoded by this gene belongs to the glycosyl hydrolase 1 family of proteins. The encoded preproprotein is proteolytically processed to generate the mature enzyme. This enzyme is integral to the plasma membrane and has both phlorizin hydrolase activity and lactase activity. Mutations in this gene are associated with congenital lactase deficiency. Polymorphisms in this gene are associated with lactase persistence, in which intestinal lactase activity persists at childhood levels into adulthood. [provided by RefSeq, Jan 2016] NCBI.

SCIENTIFIC EVIDENCE



+ GENETIC INFO

MY ANALYSIS

To date, scientific evidence regarding the genetic factors associated with intrinsic bone quality is supported by a series of scientific studies involving thousands of participants of both genders, of European origin, and including a middle-aged population.

Determination of injuries was undertaken by specialist doctors.

The genetic association observed includes the COL1A1 gene, which encodes the mainly protein component of tendons and ligaments, as well as bone structure. In addition, this gene was previously associated in dozens of scientific studies with a wide variety of sports injuries.

In particular, these relate to associations made between polymorphisms of the COL1A1 gene and bone quality, meaning an effect on the risk of bone fracture was observed, not simply due to its action on bone mineral density, but also due to its action on the elastic properties of the bone tissue. In short, these are polymorphisms that appear to affect complex mechanisms related to changes in bone mass and quality.



> INCREASE IN	VO2 MA	X HIIT.			119
> INCREASE	VO2	MAX	-	AERO	BIC
TRAINING					122
> INCREASE BI	CEPS ST	RENG1	H 1RM		125
> INCREASE	ISOMET	RIC	STREM	IGTH	IN
BICEPS					128
> MUSCLE HYP	PERTROP	HY			131
> CONVERSION	OF MUS	CLE F	BRES.		134
> WEIGHT LOS	S				137
> SUBCUTANE	OUS FAT	REDU	CTION.		140
> INCREASE C	ORTICAL	BONE	VOLUN	1E	143
> REDUCTION I	N HEART	RATE			146

> REDUCTION IN BLOOD PRESSURE	149
> REDUCTION OF TRIGLYCERIDES	152
> INCREASE HDL CHOLESTEROL	155
> INCREASE INSULIN SENSITIVITY	158
> ANGIOGENESIS	162
> MITOCHONDRIAL BIOGENESIS	165
> LEFT VENTRICULAR HYPERTROPHY	168
> INCREASE LACTATE THRESHOLD	171
> REDUCTION SYSTEMIC INFLAMMATION	174
> INCREASE SYSTOLIC VOLUME	177



INCREASE IN VO2 MAX



Your percentage



Attention Risky area

MY GENETICS

Your genetic composition, in comparison with the general population, is associated with a tendency to present a 0-5% LOWER AVERAGE INCREASE IN VO2 MAX as a result of the adaptive response that occurs due to high-intensity interval training (HIIT).

WHY ANALYSE VO2 MAX IN HIIT?

VO2 max is defined as the variable that reflects maximum consumption of oxygen during physical exercise. Therefore, it determines the athlete's aerobic capacity, indicating the maximum level of oxygen that may be captured and employed by the muscles in order to obtain the energy necessary via oxygen pathways.

It is mainly limited by the cardiorespiratory system's capacity to transport oxygen to the active musculature and, to a lesser degree, by the skeletal muscle's capacity to use it.

Although VO2 max has traditionally been considered the most influential factor in the endurance athlete's performance, today it is widely recognised that the percentage of VO2 max that may be used during exercise is a much more decisive indicator for the athlete.

Half of VO2 max will mainly depend on cardiovascular factors determined genetically (heritability), while the other half is a response to training.

Its increase is usually frequently assessed as a reflection of the positive effect of aerobic endurance training.



WHAT DOES AN INCREASE IN VO2 MAX MEAN IN PRACTICE FOR THE ATHLETE?

- **Improved aerobic** performance, which leads to a greater ability to compete over a longer period of time, mainly using the aerobic metabolic pathway to obtain energy.
- > Less muscle fatique.
- **Improved post-exercise** recovery. For example, in relation to the oxygen debt or the amount of oxygen required after exercise to:
 - Metabolise the lactate accumulated.
 - Replenish oxygen supplies in the blood (haemoglobin) and muscle (myoglobin).
 - Re-establish ATP and phosphocreatine stores.

+INFO FOOTBALL

To know more, consult the following chapters in your Informative Manual:

> VO2 max

PUM2-RHOB - A:G

GENE FUNCTION

> COL1A: COLLAGEN TYPE I ALPHA 1 CHAIN

This polymorphism is located between PUM2 and RHOB genes.

RHOB encodes a ras homolog family member B. PUM2 encodes a protein that belongs to a family of RNA-binding proteins. The encoded protein functions as a translational repressor during embryonic development and cell differentiation. This protein is also thought to be a positive regulator of cell proliferation in adipose-derived stem cells. Alternate splicing results in multiple transcript variants. [provided by RefSeq, Sep 2013].



GENE FUNCTION

> KCNH7 POTASSIUM VOLTAGE-GATED CHANNEL SUBFAMILY H MEMBER 7

Voltage-gated potassium (Kv) channels represent the most complex class of voltage-gated ion channels from both functional and structural standpoints. Their diverse functions include regulating neurotransmitter release, heart rate, insulin secretion, neuronal excitability, epithelial electrolyte transport, smooth muscle contraction, and cell volume. This gene encodes a member of the potassium channel, voltage-gated, subfamily H. This member is a pore-forming (alpha) subunit. There are at least two alternatively spliced transcript variants derived from this gene and encoding distinct isoforms. [provided by RefSeq, Jul 2008] NCBI.

ASB3 - C:T

GENE FUNCTION

> ASB3 ANKYRIN REPEAT AND SOCS BOX CONTAINING 3

The protein encoded by this gene is a member of the ankyrin repeat and SOCS box-containing (ASB) family of proteins. They contain ankyrin repeat sequence and SOCS box domain. The SOCS box serves to couple suppressor of cytokine signalling (SOCS) proteins and their binding partners with the elongin B and C complex, possibly targeting them for degradation. Alternatively spliced transcript variants have been described for this gene. [provided by RefSeq, Jan 2011] NCBI.







CDH2 and DSC3 - T:T

GENE FUNCTION

> This polymorphism is located between CDH2 and DSC3 genes.



CDH2 encodes a classical cadherin and member of the cadherin superfamily. Alternative splicing results in multiple transcript variants, at least one of which encodes a preproprotein is proteolytically processed to generate a calcium-dependent cell adhesion molecule and glycoprotein. This protein plays a role in the establishment of left-right asymmetry, development of the nervous system and the formation of cartilage and bone. [provided by RefSeq, Nov 2015].

DSC3 encodes a calcium-dependent glycoprotein that is a member of the desmocollin subfamily of the cadherin superfamily. These desmosomal family members, along with the desmogleins, are found primarily in epithelial cells where they constitute the adhesive proteins of the desmosome cell-cell junction and are required for cell adhesion and desmosome formation. The desmosomal family members are arranged in two clusters on chromosome 18, occupying less than 650 kb combined. Mutations in this gene are a cause of hypotrichosis and recurrent skin vesicles disorder. The protein can act as an autoantigen in pemphigus diseases, and it is also considered to be a biomarker for some cancers. Alternative splicing of this gene results in multiple transcript variants. [provided by RefSeq, Apr 2014].

UST - G:G

GENE FUNCTION

> UST URONYL 2-SULFOTRANSFERASE



Uronyl 2-sulfotransferase transfers sulfate to the 2-position of uronyl residues, such as iduronyl residues in dermatan sulfate and glucuronyl residues in chondroitin sulfate (Kobayashi et al., 1999 [PubMed 10187838]).[-supplied by OMIM, Mar 2008] NCBI.

SCIENTIFIC EVIDENCE



+ GENETIC INFO

MY ANALYSIS

The results reported are based on a study of your genetic composition in relation to the increase in VO2 max in response to high-intensity interval training (HIIT).

This study includes analysis of the following collection of genes: PUM2-RHOB, KCNH7, ASB3, CDH2 and UST.

The association of these genes with an increase in VO2 max was made following a genome-wide analysis, although this study has not yet been replicated in different populations, meaning only data corresponding to a Korean population exists.

Therefore, and despite the fact that the level of scientific evidence on the same is high, it is necessary to continue undertaking research to replicate these results in a European population and to shed light on the mechanism of action via which these genes act on this phenotype.

PUM2 has been associated with cell proliferation and differentiation in various tissues, including the adipose tissue. RHOB has been indirectly related to muscle contraction and the transport of molecules associated with inflammatory processes.

ASB3 has been associated with inhibiting the activity of TNFa, which is also related to various phenotypes in the field of sports. On the other hand, both CDH2 and DSC3 are cadherins (molecules dependent on the calcium metabolism) with an important role in adhesion between cells.

Similarly, KCNH7 forms a part of a type of potassium channel dependent on voltage and has been associated with regulating the release of neurotransmitters, heart rate, insulin secretion, neuronal excitability, electrolyte transport, smooth muscle contraction and cell volume.



Finally, UST transfers sulphates in molecules, such as chondroitin sulphate, and is also related to regulating the structure and elasticity of cartilage.

Analysis of this collection of genes explains 26% of the remaining variance, including differences owed to gender, age or prior fitness.

Said endurance training consisted of three weekly sessions over the course of nine weeks. It began at the heart rate calculated in each case for 60-84% VO2 max and consisted of a 40-second warm up and three 20-second HIIT series with a 40-second rest.



> MUSCLE DAMAGE - INFLAMMATION	181
> MUSCLE DAMAGE - MUSCLE CONTRACTION	184
> MUSCLE DAMAGE - FREE RADICALS	187
> FATIGUE - HIGH-INTENSITY (ALACTIC)	190
> FATIGUE - HIGH-INTENSITY (LACTIC)	193
> FATIGUE - MEDIUM-INTENSITY	196
> FATIGUE CONCENTRIC - STRENGTH IN QUADRICEPS	199
> LACTATE POST-EXERCISE	202
> DOMS STIFFNESS	205
> SLEEP QUALITY	208



MUSCLE DAMAGE - INFLAMMATION



Your percentage



Average
Within population's standard results

MY GENETICS

Your genetic composition is associated with a tendency to present an INFLAMMATORY RESPONSE SIMILAR to the response described for the general population in the process of damaged tissue regeneration as a consequence of the localised muscle damaged that occurs during exercise.

WHY ANALYSE MUSCLE DAMAGE INFLAMMATION?

For the athlete, carrying out physical activity represents a scenario of mechanical stress in which he or she may suffer muscle damage due to:

- Microtraumas that occur as a consequence of impacts against the ground, for example (running, jumping).
- Micro-tears that are caused as a result of the intense muscle contractions generated during training, mainly eccentric training exercises.

On the other hand, when faced with these stimuli of mechanical nature or structural overloading of the tissues, the body reacts by initiating an inflammatory response.

In this regard, if the inflammation involves a beneficial effect a priori, by participating in the process of regenerating damaged tissue, which is sometimes very high (overexpression of a gene) or can become chronic, it may be harmful and even cause greater damage to the tissues affected.



WHAT DOES AN OPTIMAL INFLAMMATORY RESPONSE MEAN IN PRACTICE FOR THE ATHLETE?

- > Better post-exercise recovery process (muscle recovery times are shortened). This is extremely important from a sports perspective as well as with regard to its contribution to preserving the athlete's health.
- > Less muscle fatigue as a reduced inflammatory response may delay the appearance of fatigue (caused by this reason).
- > Reduced possibility of increasing the degree of muscle damage as a consequence of an exaggerated inflammatory reaction.
- > Less regeneration of damaged tissue during physical activity.
- > Reduced increase in inflammatory plasma values (PCR).
- > Reduced intensity and greater delay in the appearance of symptoms of pain and loss of strength and function, which accompany the localised inflammatory response generated by the muscle damage caused by exercise.
- > Improved muscular endurance. Better response capacity to the introduction of higher training loads, due to being more capable of withstanding the extra muscle tension this involves.

+INFO FOOTBALL

To know more, consult the following chapters in your Informative Manual:

- > Inflammation.
- > Muscle Damage.

IL6 - G:G

GENE FUNCTION

> IL6 INTERLEUKIN 6

This gene encodes a cytokine that functions in inflammation and the maturation of B cells. In addition, the encoded protein has been shown to be an endogenous pyrogen capable of inducing fever in people with autoimmune diseases or infections. The protein is primarily produced at sites of acute and chronic inflammation, where it is secreted into the serum and induces a transcriptional inflammatory response through interleukin 6 receptor, alpha. The functioning of this gene is implicated in a wide variety of inflammation-associated disease states, including suspectibility to diabetes mellitus and systemic juvenile rheumatoid arthritis. Alternative splicing results in multiple transcript variants. [provided by RefSeq, Dec 2015] NCBI.

TNF-ALFA - G:G

GENE FUNCTION

> TNF



This gene encodes a multifunctional proinflammatory cytokine that belongs to the tumor necrosis factor (TNF) superfamily. This cytokine is mainly secreted by macrophages. It can bind to, and thus functions through its receptors TNFRSF1A/TNFR1 and TNFRSF1B/TNFBR. This cytokine is involved in the regulation of a wide spectrum of biological processes including cell proliferation, differentiation, apoptosis, lipid metabolism, and coagulation. This cytokine has been implicated in a variety of diseases, including autoimmune diseases, insulin resistance, and cancer. Knockout studies in mice also suggested the neuroprotective function of this cytokine. [provided by RefSeq, Jul 2008].

SCIENTIFIC EVIDENCE 🌟 🌟





+ GENETIC INFO

MY ANALYSIS

The results reported are based on the study of your genetic composition in relation to the inflammatory response caused following muscle damage that occurs during sports practice.

This study includes analysis of the following collection of genes: IL6 and TNFa.

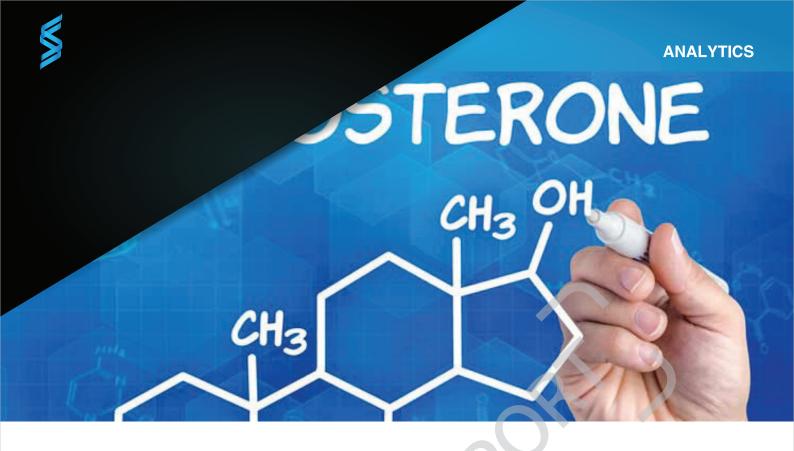
IL6 is known as interleukin 6. It is mainly expressed in immune system cells but it is also expressed in the muscle in response to muscle contraction, for which reason it is called a "myokine". It presents anti-inflammatory properties by inhibiting, among others, production of TNFα.

Meanwhile, TNFα refers to the tumour necrosis factor alpha. This is a cytokine or cell signalling molecule that participates in systemic inflammation, promoting the production of C-reactive protein and, therefore, favouring inflammation. It may also have anti-inflammatory effects via its impact on regulating its receptors. It is mainly expressed in active macrophages, although it may be produced by other types of cells, such as neutrophils. IL6 and TNFα participate in the second phase of muscle damage, which takes place post-exercise, as they are related to the inflammatory response. During this phase, leukocytes infiltrate the damaged muscle cells and remain in this position for days or even weeks.

Although some controversy exists in this area, it is also assumed that neutrophils migrate from the region in which muscle damage has occurred in the phase prior to the inflammatory phase (during the practice of exercise), contributing to degradation of the damaged muscle by producing reactive oxygen species (ROS) that attract macrophages to the damaged area.



> TESTOSTERONE	212
> INSULIN SENSITIVITY	2 15
> TOTAL CHOLESTEROL	218
> HDL CHOLESTEROL	221
> LDL CHOLESTEROL	224
> TRIGLYCERIDES	227
> C-REACTIVE PROTEIN	230
> CREATINE KINASE	233
> CREATININE	236
> WHITE BLOOD CELLS - LEUKOCYTES	239
> RED BLOOD CELLS - ERYTHROCYTES	242
> RED BLOOD CELLS - HAEMOGLOBIN	245
> HEMOCHROMATOSIS	248
> IRON	251
> FERRITIN	254
> TRANSFERRIN	257



TESTOSTERONE



Your percentage



Average
Within population's standard results

MY GENETICS

Your genetic composition is associated with presenting a CONCENTRATION OF TESTOSTERONE IN THE BLOOD SIMILAR to that of the general population.

WHY ANALYSE TESTOSTERONE?

Testosterone is a sexual steroid hormone (androgen) that is usually considered a male hormone but in reality is produced both in the male's testicles and in the female's ovaries.

However, its plasma concentration is far superior in men (approximately ten times higher).

Knowledge of your individual genetic tendency with regard to testosterone concentration in the blood will allow the athlete and/or professional responsible for requesting a blood analysis to use an individual reference point that enables the results to be assessed from a genetic perspective, therefore permitting more extensive and more rigorous knowledge of the athlete.

It is recommended that the athlete, especially during certain stages of the season (high volume of training), undergo routine plasma testosterone controls (full and free) in order to prevent negative effects (both in terms of performance and health) derived from low levels of testosterone.



WHAT DOES A HIGHER CONCENTRATION OF TESTOSTERONE IN THE BLOOD MEAN IN PRACTICE FOR THE ATHLETE?

HEALTH:

> Lower risk of injury (for example, stress fractures) due to an increase in bone density.

SPORT:

- > Lower risk of injury (for example, stress fractures) due to an increase in bone density.
 - Moderate increase in muscle mass.
 - Reduction in body fat.
 - Increase in strength.
- **Improved post-exercise** recovery as damage caused to the muscle fibres during training is repaired more efficiently.

+INFO FOOTBALL

To know more, consult the following chapters in your Informative Manual:

> Testosterone

SHBG - G:G

GENE FUNCTION

> SHBG SEX HORMONE BINDING GLOBULIN

57%

This gene encodes a steroid binding protein that was first described as a plasma protein secreted by the liver but is now thought to participate in the regulation of steroid responses. The encoded protein transports androgens and estrogens in the blood, binding each steroid molecule as a dimer formed from identical or nearly identical monomers. Polymorphisms in this gene have been associated with polycystic ovary syndrome and type 2 diabetes mellitus. Alternative splicing results in multiple transcript variants. [provided by RefSeq, Jan 2014] NCBI.

ACTN3 - C:C

GENE FUNCTION

> ACTN3 ACTININ ALPHA 3 (GENE/PSEUDOGENE)

31%

This gene encodes a member of the alpha-actin binding protein gene family. The encoded protein is primarily expressed in skeletal muscle and functions as a structural component of sarcomeric Z line. This protein is involved in crosslinking actin containing thin filaments. An allelic polymorphism in this gene results in both coding and non-coding variants; the reference genome represents the coding allele. The non-functional allele of this gene is associated with elite athlete status. [provided by RefSeg, Feb 2014] NCBI.

GCKR-T:T

GENE FUNCTION

> GCKR GLUCOKINASE REGULATOR

16%

This gene encodes a protein belonging to the GCKR subfamily of the SIS (Sugar ISomerase) family of proteins. The gene product is a regulatory protein that inhibits glucokinase in liver and pancreatic islet cells by binding non-covalently to form an inactive complex with the enzyme. This gene is considered a susceptibility gene candidate for a form of maturity-onset diabetes of the young (MODY). [provided by RefSeq, Jul 2008] NCBI.



SCIENTIFIC EVIDENCE * * *



+ GENETIC INFO

MY ANALYSIS

The results reported are based on the study of your genetic composition in relation to the concentration of testosterone in the blood.

This study includes analysis of the following collection of genes: ACTN3, SHBG and GCKR.

ACTN3 is one of the most widely studied proteins in Sports Genetics. ACTN3 is α-actinin-3, a sarcomeric structural protein. This protein helps support the myofibrillar structure of the sarcomere, interacting with a high number of structural, metabolic and signalling proteins and exercising a protective function on the muscle, mainly during the undertaking of eccentric movements. Indeed, the polymorphism analysed in this study is associated with differences between individuals in the amount of testosterone found in the blood.

SHBG regulates the bioavailability of sex hormones such as testosterone and estradiol. The polymorphism analvsed in SHBG in this genetic study allows differences to be established between individuals regarding the relative risk of presenting testosterone values in the blood under 300ng/dl, an amount deemed reduced for men.

The union of SHBG to sex hormones inhibits the action of the latter, preventing them from entering the cell.

An association has been reported between the polymorphism analysed in the GCKR gene in this genetic study and the amount of SHBG that, as mentioned previously, inhibits the action of testosterone. Finally, GCKR acts on the intracellular availability of glucose by regulating the activity of glucokinase, also known as the "glucose sensor".



> ANTIOXIDANTS	260
> CAPSAICIN - FAT LOSS	263
> GREEN TEA - FAT LOSS	266
> CHROMIUM PICOLINATE - FAT LOSS	269
> CAFFEINE - CONCENTRATION/EFFECT	272
> CAFFEINE - METABOLISING SPEED	275
> OMEGA-3	
REDUCTION IN TOTAL CHOLESTEROL	278
> OMEGA-3	
- WEIGHT LOSS	281
> OMEGA-3	
- INCREASED HDL CHOLESTEROL	284
> OMEGA-3	
- REDUCTION OF TRIGLYCERIDES	287

> VIT B6	290
> VIT B9 (FOLIC ACID)	293
> VIT B12	296
> VIT A	299
> VIT C	302
> VIT D	305
> CALCIUM	308
MAGNESIUM	311



MUSCLE DAMAGE - INFLAMMATION



Your percentage



Attention Risky area

MY GENETICS

Your genetic composition, in comparison with the rest of the population, is associated with a tendency to present a REDUCED DETOXIFYING EFFECT against free radicals in response to ANTIOXIDANT supplementation.

WHY ANALYSE THE ANTIOXIDANT EFFECT?

During exercise (and mainly when this is performed regularly) at a high intensity and over an extended period of time there will be an increase in free radical production that may cause oxidative damage in the muscle tissue.

Occasionally the athlete turns to antioxidant supplementation to complement and strengthen a diet that may be insufficient in terms of antioxidant supply.

However, it is recommended that this form of supplementation take place during the competition phase where the aim is to improve performance and achieve better post-exercise recovery, and not during training, in initial stages of the season, in which physiological adaptations to training are occur.



WHAT DOES A GREATER ANTIOXIDANT EFFECT MEAN IN PRACTICE FOR THE ATHLETE?

- > Less oxidative stress.
- > Less muscle fatigue.
- Lower risk of suffering from a greater degree of muscle damage and/or a muscle injury (caused by this reason) during physical activity.
- > Better post-exercise recovery process (less muscle damage).

+INFO FOOTBALL

To know more, consult the following chapters in your Informative Manual:

> Free radicals and antioxidants

SOD2 - T:T

GENE FUNCTION

> SOD2 SUPEROXIDE DISMUTASE 2



This gene is a member of the iron/manganese superoxide dismutase family. It encodes a mitochondrial protein that forms a homotetramer and binds one manganese ion per subunit. This protein binds to the superoxide byproducts of oxidative phosphorylation and converts them to hydrogen peroxide and diatomic oxygen. Mutations in this gene have been associated with idiopathic cardiomyopathy (IDC), premature aging, sporadic motor neuron disease, and cancer. Alternative splicing of this gene results in multiple transcript variants. A related pseudogene has been identified on chromosome 1. [provided by RefSeq, Apr 2016] NCBI.

SCIENTIFIC EVIDENCE



+ GENETIC INFO

MY ANALYSIS

The results reported are based on a study of your genetic composition in relation to detoxifying capacity in response to antioxidant supplementation.

The study includes analysis of the SOD2 gene.

SOD2 is a mitochondrial enzyme related to the muscle metabolism in general and muscle damage in particular.

This is mainly due to its antioxidant effect in light of an increase in the production of free radicals during exercise.

An increase in antioxidant capacity is a characteristic adaptation in response to exercise. It responds, in part, to the need to reduce the amount of free radicals produced during exercise.

A limited amount of free radicals is necessary for suitable muscle contraction but when exercise is intense or takes place over an extended period of time, free radical compensation systems are overwhelmed and may cause tissue and DNA damage.

The results correspond to a study performed on a healthy European population that took a daily antioxidant supplement capsule for six weeks, with the following composition per capsule: 100µg selenium, 450µg vitamin A (300µg retinol, 150µg beta-Carotene), 90mg vitamin C and 30mg vitamin E.



315
318
321
324
327
330
333
336
339
342
345
348
351
354



TESTOSTERONE



Your percentage



Above average Positive Highlight

MY GENETICS

Your genetic composition, in comparison with the general population, is associated with a tendency to present 6.7% MORE CONCENTRIC STRENGTH in a study of BICEPS STRENGTH/ONE-REPETITION MAXIMUM.

WHY ANALYSE CONCENTRIC STRENGTH?

In general, strength is a muscle quality that allows for movements, such as throwing an object or moving the body. The appropriate level of strength is required in order to carry out activities such as maintaining the posture, standing, walking, jumping, running, etc.

In terms of sports performance, it is directly related to two essential aspects: sports execution techniques and speed of movement.

With regard to eccentric and concentric dynamic muscle strength, measurement is usually performed using the one-repetition maximum (1RM) method, and one or several muscle groups can be assessed (bench press, press, biceps curl, squat, quadriceps extensions, etc.).



WHAT DOES GREATER DYNAMIC STRENGTH MEAN IN PRACTICE FOR THE ATHLETE?

- > Increased dynamic muscle strength encompasses both eccentric and concentric strength.
- > Greater ability to move the body or throw an object.
- > Improved sports performance by facilitating sports execution techniques and speed of movement.
- > Greater ability to overcome very high resistance in a single action.
- **> Allows for the introduction** of increased training loads with a greater range of safety and efficiency in their handling.
- **> Strength is essential** in a large majority of exercises performed in any sports discipline. However, and by way of example, in this specific case measurement is carried out via one-repetition maximum with biceps exercise.

+INFO FOOTBALL

To know more, consult the following chapters in your Informative Manual:

> Strength

CCL2 - C:C

8%

GENE FUNCTION

> CCL2

This gene is one of several cytokine genes clustered on the q-arm of chromosome 17. Chemokines are a superfamily of secreted proteins involved in immunoregulatory and inflammatory processes. The superfamily is divided into four subfamilies based on the arrangement of N-terminal cysteine residues of the mature peptide. This chemokine is a member of the CC subfamily which is characterized by two adjacent cysteine residues. This cytokine displays chemotactic activity for monocytes and basophils but not for neutrophils or eosinophils. It has been implicated in the pathogenesis of diseases characterized by monocytic infiltrates, like psoriasis, rheumatoid arthritis and atherosclerosis. It binds to chemokine receptors CCR2 and CCR4. [provided by RefSeq, Jul 2013]

SCIENTIFIC EVIDENCE



+ GENETIC INFO

MY ANALYSIS

To date, scientific evidence related to the genetic factors associated with the degree of strength in the biceps, in the form of a one-repetition maximum, is supported by the largest scientific study carried out in this area, involving 1300 participants from both genders, mainly of European origin, and including young adults of normal weight.

Determination of strength was undertaken by sports science specialists.

With regard to the evidence of genetic associations, a total of 500 polymorphisms were analysed, with significant differences in the degree of strength in the biceps, in the form of a one-repetition maximum, found in encoding genes for chemokines, cytokines with chemotactic activity related to the immune system and the inflammatory response.

As such, it is biologically plausible that this gene and its polymorphism will play a key role in the muscle metabolism and in its adaptation to the undertaking of physical activity.