



dnaskin[®]

Welcome

Example

to your DNA Skin report

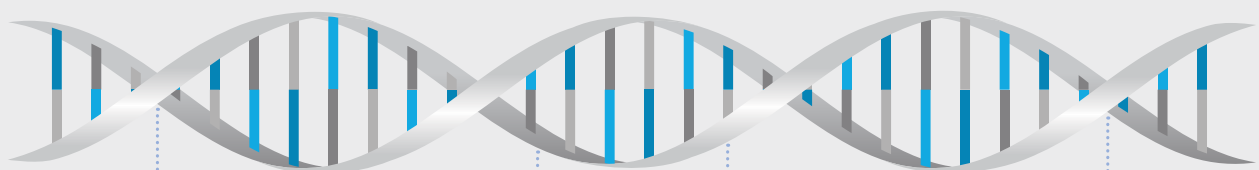
Date of birth: 01 Jan 2001

Date reported: 07 Dec 2021

Sample number: 12345678

Referring practitioner: Private

DNA Skin is a genetic test that offers insight into key areas that influence skin ageing, offering personalised topical, nutraceutical, diet, and lifestyle recommendations for improved outcomes.



Firmness & elasticity



Sun (UV) -ageing



Pigmentation



Skin sensitivity



Acne and breakouts



Antioxidant status

Genetics and personalised medicine

Genes are segments of DNA that contain the instructions your body needs to make each of the many thousands of proteins required for life. Each gene is comprised of thousands of combinations of “letters” (called bases) which make up your genetic code. The code gives the instructions to make the proteins required for proper development and function.

Genetic variations (small differences in our DNA) can affect the expression of a gene, thereby affecting metabolic processes that are important for maintaining cellular health and how we respond to environmental interventions such as diet, lifestyle, supplements, and medication. Knowledge of these genetic variations offers unparalleled insight into your biological systems, allowing your healthcare practitioner to recommend precise interventions aimed at helping you reach your goals and achieve optimal health.



NORMAL GENE

Genotype resulting in expected response to interventions

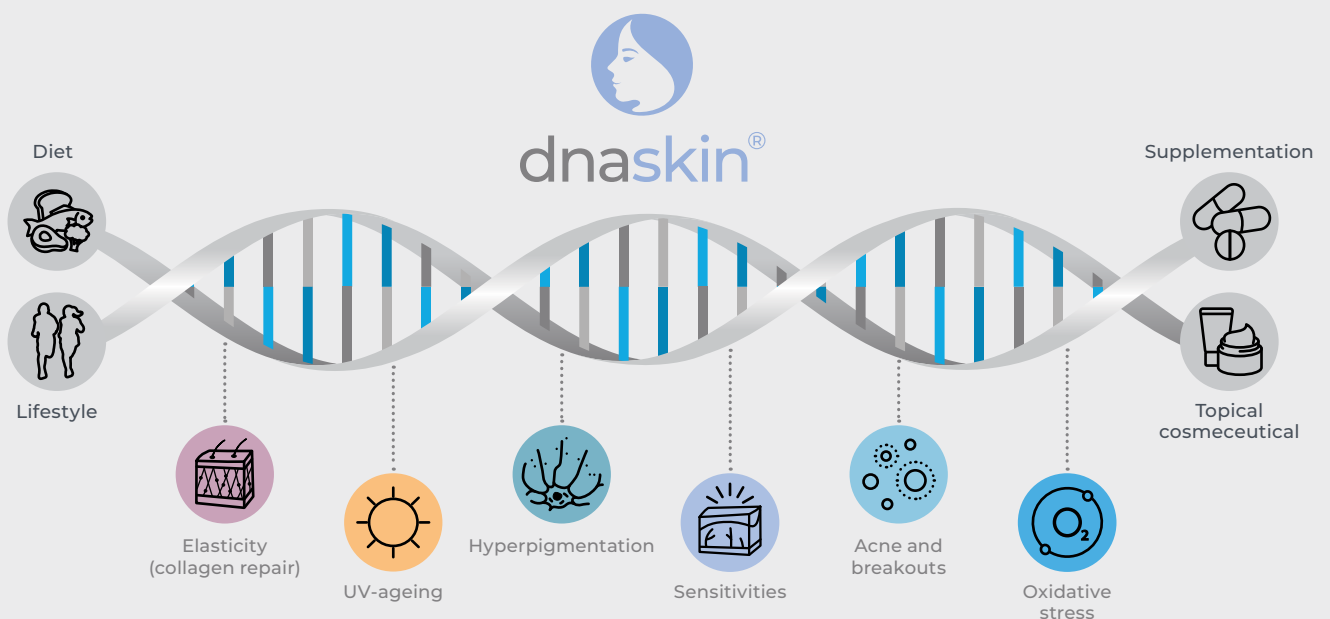


VARIANT GENE

Genotype resulting in altered functioning of a biological area and a need for personalised skin care recommendations

Personalised medicine and skincare

In general, the health of your skin can be measured by considering its appearance through any visual signs of ageing – wrinkles and fine lines, hyper/dyspigmentation, and course skin texture. Your ethnicity, age, UV-exposure, nutrient intake, cosmeceutical use, exposure to environmental pollutants, stress levels, weight management and exercise routine all contribute toward the status of your skin’s health. Within each of these factors, genetics has a major role to play in determining the interindividual variability that exists in the ageing process as well as your response to environmental input to improve skin health. DNA Skin reports on your unique genetics and how gene variations that you may carry could affect your skin and the way it ages. This unique genetic information is your own perfect “skin assistant”. You can use the “skin assistant” information to make personalised skincare decisions. Insight into the gene variations that you carry assists in personalising and optimising your lifestyle, diet, beauty supplements and topical skincare regime.



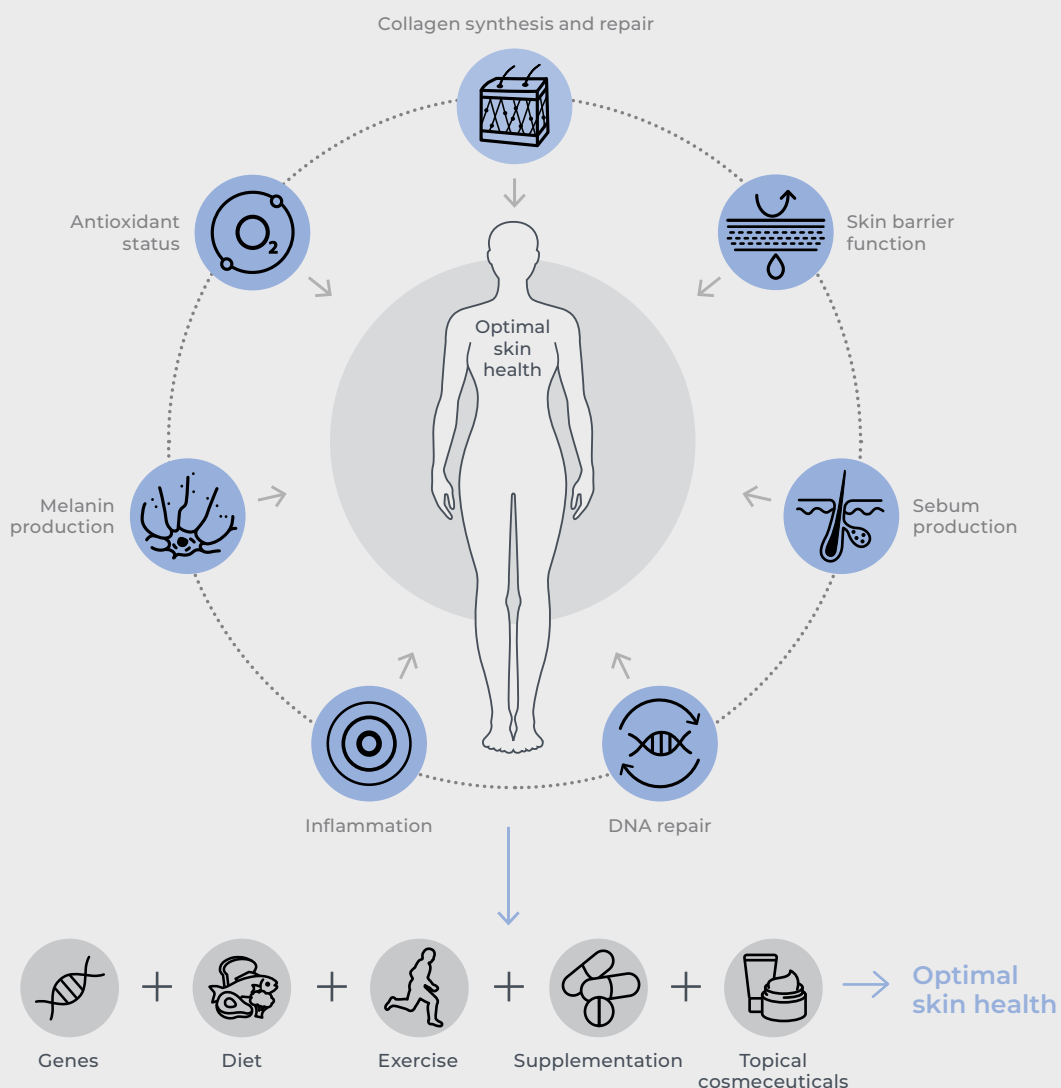
Understanding healthy skin ageing

Your skin's health results from a complex interplay between your genes (your blueprint) and what you are exposed to on a daily basis and is thus polygenic and multifactorial in nature. This means that whilst your susceptibility to skin concerns has a strong genetic contribution, many other factors also play a significant role, including environmental exposure to toxins and stress, gut health, hormonal health and age, exercise behaviour, and dietary intake. Therefore, both genes and environment should be considered to better understand the mechanisms behind skin ageing.

The underlying biological mechanisms that have key functions in skin ageing include:







- Collagen breakdown & repair – influencing skin elasticity
- Antioxidant defence & detoxification – influencing skin barrier function and formation of fine wrinkles
- DNA repair mechanisms – protecting from ultraviolet (UV) ageing
- Melanin production – influencing (hyper)pigmentation and risk for freckles/solar mottling
- Moisture retention – influencing barrier function and sensitivities
- Sebum production – important in acne control, and
- Inflammation – playing a major role in being prone to acne and (dys)pigmentation.

DNA SKIN PROVIDES INSIGHTS INTO KEY AREAS FOR IMPROVED SKIN AGEING



Result summary

Outcomes: Skin priorities

CATEGORY	IMPACT
 Elasticity	
 Antioxidant status and environmental pollution	
 UV-ageing	
 Pigmentation	
 Breakouts	
 Sensitivities	








Summary recommendations

Based on your priority area outcomes, we have provided summary recommendations for the key area's you should be focusing on for optimal skin health.



Genotype results

No Impact
 Low Impact
 Moderate Impact
 High Impact
 Beneficial Impact

BIOLOGICAL AREA	GENE NAME	GENE VARIATION	RESULT	GENE IMPACT
 Collagen breakdown and repair	COL1A1	1546 G>T	GG	
	MMP1	1G>2G	1G/2G	
 Antioxidant defence and detoxification	CAT	-262 C>T	CC	
	GPX1	C>T	CT	
	SOD2	47 T>C (Val16Ala)	CC	
	EPHX1	C>T	CT	
	GSTP1	313 A>G	AG	
	NQO1	T>C	CC	
 DNA repair mechanisms	XRCC1	Q399R (T>C)	CC	
	XRCC1	R280H (G>A)	AA	
	hOGG1	C>G	CG	
	TERT	C>T	CT	
 Melanin production	ASIP	G>A	AA	
	ASIP	G>T	TT	
	IRF4	C>T	CT	
	MATP (SLC45A2)	1122 C>G	CC	
	MC1R	Arg160Trp (C>T)	TT	
	MC1R	C>T	CT	
	TYR	A>G	AG	
 Inflammation	CRP	G>A	GG	
	IL6	-174 G>C	GG	
	IL6R	A>C	CC	
	TNFA	-308 G>A	GG	
 Sebum regulation	FST	G>A	GG	
 Skin moisture control	TEWL	G>A	AA	

Your results and recommendations



Elasticity

Collagen is the lovely protein that makes up approximately 75% of skin and is responsible for keeping it youthful looking and firm. Elastin, another protein-based fiber in your skin is responsible for its ability to stretch and bounce back to its original form. Collagen loss starts at 18-29 years of age, after 40 you can lose around 1% per year, and at around 80 years collagen synthesis in the body can decrease 75% overall in comparison to that of young adults. Certain variations in genes will result in an increase in the breakdown of existing collagen and less synthesis of new collagen.



Collagen breakdown and repair results

Genotype result table:

GENE NAME	GENE VARIATION	RESULT	GENE IMPACT
COL1A1	1546 G>T	GG	
MMP1	1G>2G	1G/2G	



Antioxidant status | Environmental pollution

Environmental pollutants include sunlight, smoke and water contaminants. Pollutants generate free radicals in your body and skin. The end product of free radicals cause skin to produce “self-destruct” enzymes, such as collagenase which destroys collagen, and elastase which destroys elastin. When there is an imbalance in your system (the free radicals outnumber all your antioxidants) this causes oxidative stress. Oxidative stress in turn is linked to premature skin ageing.

There are three types of antioxidants that form part of your defense system:

- Enzymatic antioxidants and detoxification enzymes found naturally in your body.
- The antioxidants your body makes when you feed it the right building blocks.
- Antioxidants from the food you eat and topical antioxidants you apply to your skin.

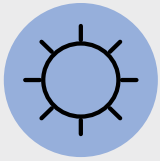
Variations in genes associated with your own enzymatic antioxidant response against free radicals make you more susceptible to the damaging effects of free radicals caused by environmental pollutants and prone to premature ageing related to oxidative stress. Detoxifying genes help to neutralise and excrete a lot of these environmental pollutants and toxins to which you are exposed, reducing the risk of free radicals forming and damaging your skin.



Antioxidant defence and detoxification results

Genotype result table:

GENE NAME	GENE VARIATION	RESULT	GENE IMPACT
CAT	-262 C>T	CC	
GPX1	C>T	CT	
SOD2	47 T>C (Val16Ala)	CC	
EPHX1	C>T	CT	
GSTP1	313 A>G	AG	
NQO1	T>C	CC	



UV-ageing

Sun (UV)-ageing is caused by UVA and UVB sun rays. UVA is linked to skin ageing. UVB is the main risk factor for burning and cancer.

UVA(geing): UVA is the most prevalent form of UV radiation on earth. UVA presents with equal intensity during all daylight hours. It penetrates clouds, fog and glass. UVA rays play a smaller role in sunburn than UVB rays. That doesn't mean UVA isn't dangerous. UVA is the chief tanning ray (also used for tanning in sunbeds). Any sign of a tan means DNA damage.

UVB(urning): UVB rays are the chief culprit behind skin reddening and sunburn. UVB rays cannot penetrate through glass and vary in intensity. For this reason sunburn depends on weather, the time of day and location. UVB damages the skin's outermost layers. You will know this as the familiar peeling after burning. UV-ageing presents as skin with a leathery and coarse texture, sagging and wrinkling, discolouration and uneven pigmentation, skin abnormalities including skin cancer.



DNA repair mechanisms results

Genotype result table:

GENE NAME	GENE VARIATION	RESULT	GENE IMPACT
XRCC1	Q399R (T>C)	CC	
XRCC1	R280H (G>A)	AA	
hOGG1	C>G	CG	
TERT	C>T	CT	



Pigmentation

Hyperpigmentation, amongst others, includes freckling and solar mottling/sunspots which mostly affects lighter skin tones on the Fitzpatrick scale (1-3) and post inflammatory hyperpigmentation (PIH) which is more prevalent in darker skin tones on the Fitzpatrick scale (4-6).

The Fitzpatrick skin tone scale:



I
Light, pale white



II
White, fair



III
Medium white to olive



IV
Olive, mid brown



V
Brown, dark brown



VI
Very dark brown, black

Freckling and solar mottling

Uneven pigment or solar mottling is a symptom of cumulative sun damage. Solar mottling includes areas of uneven, dark splotches on your face. Sun and age (liver) spots are related to this type of pigmentation.

Post inflammatory hyperpigmentation

Post-inflammatory hyperpigmentation (PIH) is caused by inflammation after a scrape, minor injury, or pimples. An immune response to the inflammation triggers melanin overproduction.

Other types: Hormonal imbalance and pregnancy pigmentation, referred to as melasma. Side effects of certain medications can also be contributing factors.

Sun exposure worsens all types of hyperpigmentation including PIH. Gene variations tested include those related to the predisposition of developing solar mottling and a tendency for freckling. Genetic predisposition for inflammation, which in turn increases your tendency for PIH is also tested.



Melanin production results

Genotype result table:

GENE NAME	GENE VARIATION	RESULT	GENE IMPACT
ASIP	G>A	AA	
ASIP	G>T	TT	
IRF4	C>T	CT	
MATP (SLC45A2)	1122 C>G	CC	
MC1R	Arg160Trp (C>T)	TT	
MC1R	C>T	CT	
TYR	A>G	AG	

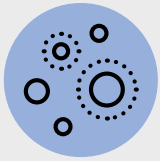


Inflammation results

Genotype result table:

GENE NAME	GENE VARIATION	RESULT	GENE IMPACT
CRP	G>A	GG	
IL6	-174 G>C	GG	
IL6R	A>C	CC	
TNFA	-308 G>A	GG	





Breakouts

Acne is an inflammatory skin condition dependant on various factors including the amount or the way your skin sheds dead cells, the presence of an acne forming bacteria, called Propionibacterium acnes (P. acnes), found at the bottom of your follicles, and the amount of sebum you produce. Genetics play a key role in these determining factors namely the number, size, and productivity of your sebaceous glands. Hormones, also influenced by gene variation and other factors, have an added impact on skin oiliness.

Oiliness = more food for P. acnes

P. acnes multiplies constantly - making new bacteria - but is also constantly kept in check by oxygen that kills it off. Clogged pores, filled with sticky cells, block oxygen from penetrating to the very bottom of the follicle which in turn allows P. acnes to flourish unchecked. In a short time, the follicle becomes swollen with bacteria, dead cells, solidified sebum, and inflammation. The result is acne. Gene variations tested are associated with a higher odds of developing acne.



Inflammation and sebum regulation results

Genotype result table:

GENE NAME	GENE VARIATION	RESULT	GENE IMPACT
TNFA	-308 G>A	GG	
FST	G>A	GG	



Sensitivities

Sensitive skin is highly reactive to treatments and environmental factors. At least 40% of people consider themselves to be in this category. In addition to being more reactive, sensitive skin can also have skin barrier function defects. Skin types 1-3 on the Fitzpatrick system are thinner. Thin skin is more likely to have barrier issues. An impaired barrier allows for easier penetration of irritants. This leads to inflammation and redness. Burning, stinging, and itching, therefore, go hand-in-hand with sensitive skin.



I
Light, pale
white



II
White,
fair



III
Medium
white to olive

NOTE: A hypersensitive allergic reaction is something different. It is often confused with having sensitive skin. A hypersensitive allergic reaction is the body's immune system rejecting a particular substance. These substances include food, airborne substances or topical cosmetic ingredients. A hypersensitive reaction triggers an immediate response. The result is skin reddening (often a fine red rash), swelling or hives. Hypersensitivity is not linked to an impaired skin barrier.



Inflammation and skin moisture control results

Genotype result table:

GENE NAME	GENE VARIATION	RESULT	GENE IMPACT
CRP	G>A	GG	
IL6	G>C	GG	
IL6R	A>C	CC	
TNFA	-308 G>A	GG	
TEWL	G>A	AA	

Gene descriptions

Collagen breakdown and repair: Elasticity



COL1A1

COL1A1 encodes a component of the type 1 collagen, which is the main collagen found in skin. It is important in maintaining the integrity of the extracellular matrix. The G allele of the COL1A1 gene is associated with decreased collagen production.



MMP1

MMP1, encoding matrix metalloproteinase 1, is responsible, in part, for the degradation of the extracellular matrix. During skin ageing, there is an upregulation of MMP1, leading to increased breakdown of collagen in the skin. The variant is associated with increased collagen breakdown, and therefore increased susceptibility for wrinkling.

Antioxidant defence and detoxification: Antioxidant status and environmental pollutants



CAT

CAT encodes the antioxidant enzyme, catalase, which is most highly expressed in the liver, kidney and erythrocytes. The enzyme is responsible for the rapid conversion of hydrogen peroxide to water and oxygen, where one molecule of this enzyme can catalyse more than 1 million hydrogen peroxide molecules per second. The variant is associated with decreased CAT activity leads to increased concentrations of hydrogen peroxide, hence leading to increased oxidative stress.



GPX1

Glutathione peroxidase 1 (GPx1) is the most abundant of the selenoperoxidase enzymes, and is expressed in almost all tissues in the body. It is responsible for catalysing the conversion of hydrogen peroxide into water, as well as reducing fatty acid hydroperoxides and peroxynitrite using glutathione as a substrate, and thus helps to maintain redox balance. The variant is associated with decreased enzyme activity and increased risk for oxidative stress.



SOD2

The SOD2 enzyme destroys the free radicals which are normally produced within cells and which are damaging to biological systems. The enzyme thus has important anti-oxidant activity within the cell, especially within the mitochondria.



EPHX1

EPHX1 encodes Epoxide Hydroxylase, which is a critical detoxification enzyme that converts epoxides from the degradation of aromatic compounds to trans-dihydrodiols which can be conjugated and excreted from the body. Sources of these compounds include smoke and chemical cleaners. Epoxide hydrolase functions in both the activation and detoxification of epoxides. The variant leads to decreased activity of the enzyme and is associated with increased oxidative stress and may also increase risk for skin sensitivity and premature ageing.



GSTP1

GSTP1 encodes Glutathione S-Transferase P1, which is a phase 2 detoxification enzyme. Glutathione S-transferases are a family of enzymes that play an important role in detoxification by catalysing the conjugation of many hydrophobic and electrophilic compounds with reduced glutathione. It is involved in efficiently detoxifying toxic compounds so that they can be safely metabolised and converted to water. The variant leads to decreased enzyme activity and is associated with increased risk of skin sensitivities.



NQO1

NQO1 encodes NADP(H:) quinone oxidoreductase 1, which is primarily involved in the detoxification of potentially mutagenic and carcinogenic quinones derived from tobacco smoke, diet and oestrogen metabolism. NQO1 also protects cells from oxidative stress by maintaining the antioxidant forms of ubiquinone and vitamin E. The variant leads to significant decreased activity of the enzyme and is associated with increased risk for oxidative stress.

DNA repair mechanisms: UV-ageing



hOGG1

hOGG1 encodes the enzyme, 8-oxoguanine DNA glycosylase 1. This is the main enzyme that is responsible for repairing the 8-oxoguanine DNA mutation caused by 8-oxo-G radicals as a result of exposure to reactive oxygen species. The variant is associated with decreased ability to repair damaged DNA, thus increasing risk for accelerated ageing, as well as diseases related to oxidative stress.



TERT

TERT codes for telomerase reverse transcriptase, a catalytic subunit of the enzyme, telomerase, which is essential in maintaining telomeres (the repeated segments of DNA found at the ends of chromosomes). Telomeres protect chromosomes from abnormally sticking together or breaking down, and so the enzyme is responsible for preventing degradation of the chromosomal ends following multiple rounds of replication. The C allele of the SNP is associated with a decrease in telomere length, and contributed toward increased risk for non-melanoma skin cancers.



XRCC1

XRCC1 encodes the protein X-Ray Repair Cross-Complementing Protein 1. It is involved in the efficient repair of DNA single-strand breaks formed by exposure to ionizing radiation, UV rays from the sun. The variant is associated with improved activity of the protein, therefore conferring protection against DNA damage caused by UV exposure.

Melanin production: Hyperpigmentation & environmental pollutants



ASIP

ASIP, encoding Agouti Signalling Protein, is involved in regulating the production of melanin by acting as an inhibitor to the Melanocortin 1 Receptor. Variations in the ASIP gene are associated with over-expression of the gene, and unbalanced melanin production, leading to higher pheomelanin, which predisposes to a fair skin type, offering less protection for the skin against UV rays and sun exposure.



IRF4

This gene encodes interferon regulatory factor 4, which is related to regulation of melanin production. A variant in this gene is strongly associated with sensitivity of skin to sun exposure, freckles, blue eyes and brown hair color.



MATP (SLC45A2)

MATP, also called SLC45A2 (solute carrier family 45 member 2), provides instructions for making a protein that is located in melanocytes. The function of this protein is to transport molecules necessary for the normal function of melanosomes, which are the structures in melanocytes where melanin is produced. A variant is associated with altered functioning of this transporter and increased susceptibility to hyperpigmentation.



MC1R

MC1R encodes the protein, Melanocortin 1 Receptor, which plays an important role in normal pigmentation. The receptor is primarily located on the surface of melanocytes, which are cells that produce a pigment called melanin. A variation in this gene is associated with increased production of a pigment that leads to a lighter, or fairer skin tone and increased predisposition to freckles. Individuals with this variant have a poorer tanning ability and have an increased risk of skin damage caused by sun exposure.



TYR

TYR encodes the enzyme, tyrosinase, which is located in melanocytes and is involved in the first step of melanin production. A variant in this gene is associated with fair skin and predisposition to hyperpigmentation and skin abnormalities.

Inflammation: Hyperpigmentation, breakouts and sensitivities



CRP

CRP encodes the proinflammatory marker, C-Reactive Protein, which assists in complement binding to foreign and damaged cells and enhances phagocytosis by macrophages. It is also important in immunity regulation. The variant leads to higher levels of CRP being produced, and is linked to disorders related to chronic low-grade inflammation.



IL6

IL6 encodes Interleukin 6, which is a pro-inflammatory cytokine that plays a crucial role in inflammation and regulates expression of CRP. The variant leads to increased expression and is associated with higher baseline levels of CRP.



IL6R

IL6R encodes for the Interleukin 6 receptor, which influences the proinflammatory IL6 cytokine action. The variant has been associated with increased risk for chronic low-grade inflammation.



TNFA

TNFA encodes for Tumour Necrosis Factor Alpha, which is a pro-inflammatory cytokine involved in regulating inflammation. The variant leads to increased expression of TNFA, which may lead to increased risk for chronic, low-grade inflammation, acne formation, and skin sensitivity.

Sebum regulation: Breakouts



FST

FST encodes for the protein, follistatin, which has an inhibitory effect on TGF-Beta. The variation in this gene is related to alterations in the structure and maintenance of the skin, in particular the pilosebaceous unit, partly by inhibiting TGF-Beta, more sebum can be produced, which results in increased risk for acne development.

Skin moisture control: Sensitivities

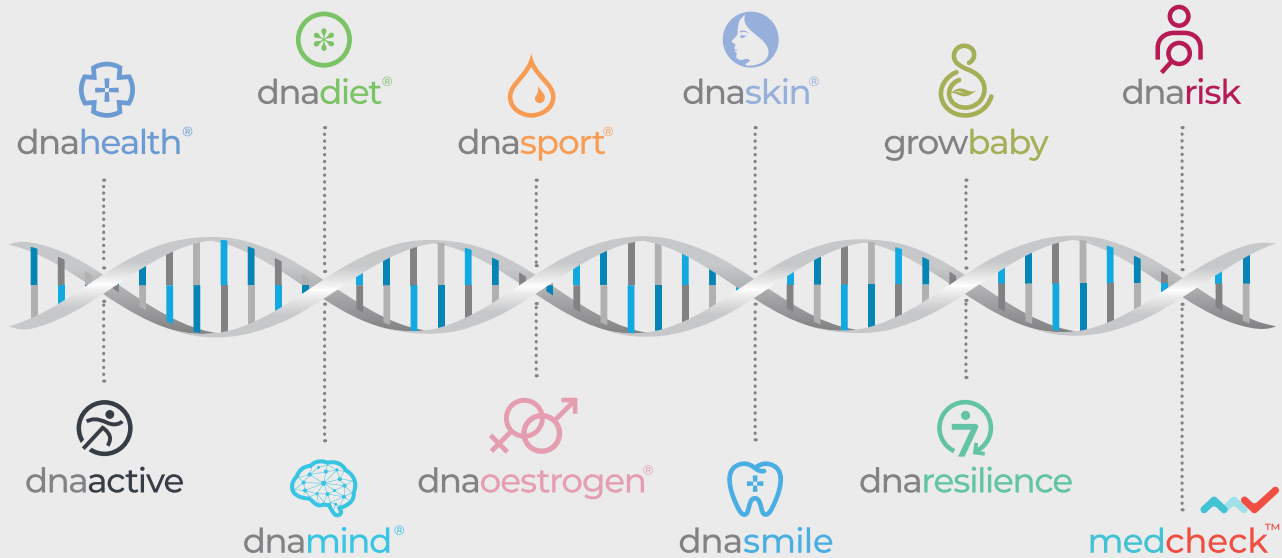


TEWL

This gene is related to the amount of moisture that evaporates through the skin, which is known as transepidermal water loss. A variant in this gene has been associated with accelerated loss of water from the skin, which can lead to high predisposition for a dry, sensitive skin.

A lifetime of optimal health awaits you

Your genes do not change, which means our laboratories will only ever need one sample* from you. Throughout your life, as your health goals and priorities change, we can continue to provide valuable health insights from this single sample* to support your unique health journey.



*Requires finger prick blood spot sample collection

Our Commitment

DNALYSIS Biotechnology is continuously developing new tests with the highest standards of scientific rigour. Our commitment to ensuring the ethical and appropriate use of genetic tests in practice means that gene variants are only included in panels once there is sound motivation for their clinical utility and their impact on health outcomes.

ADVANCED | **ACTIONABLE** | **APPROPRIATE**
technology interventions use in practice

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Risks and Limitations:

DNALYSIS Biotechnology has a laboratory with standard and effective procedures in place for handling samples and effective protocols in place to protect against technical and operational problems. However as with all laboratories, laboratory error can occur; examples include, but are not limited to, sample or DNA mislabelling or contamination, failure to obtain an interpretable report, or other operational laboratory errors. Occasionally due to circumstances beyond DNALYSIS Biotechnology's control it may not be possible to obtain SNP specific results.

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