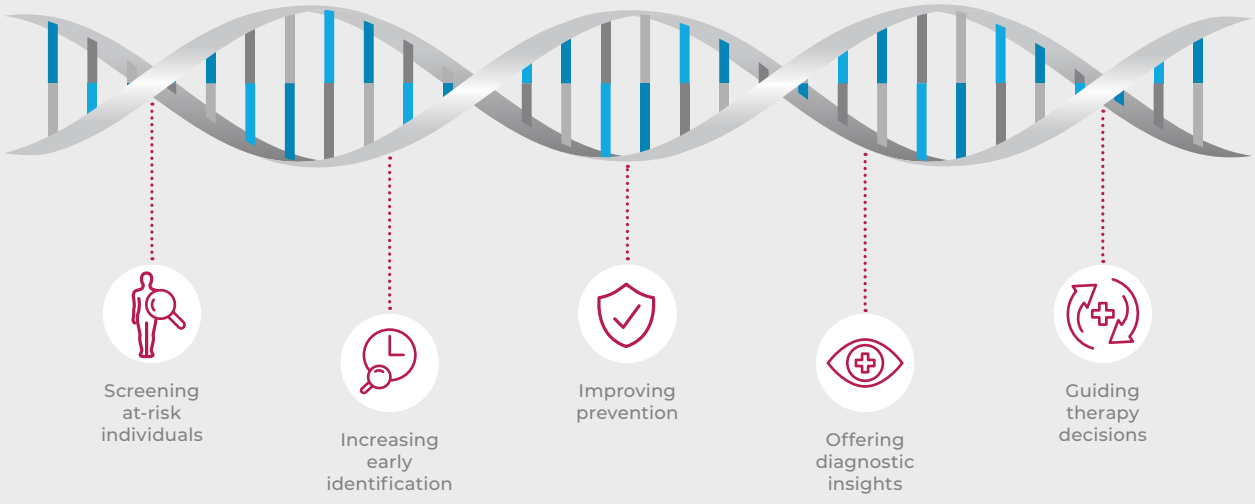
dnarisk

Sample Report

HLA-Related Autoimmunity Report

Date of birth: 01 Jun 9999	Date reported: 25 Oct 2024	Sample number: xxx
Referring practitioner: Private		

DNA Risk from DNAlysis is a category of gene tests that report on the risk of certain disorders by analysing associated genetic mutations. DNA Risk gene tests will assist in screening at-risk individuals, increasing early identification, improving prevention, offering diagnostic insights, and guiding therapy decisions.



Autoimmune Diseases and Genetics

Autoimmune diseases occur when the immune system fails to distinguish self from non-self, resulting in a breach of tolerance. This heterogenous group of diseases share common genetic risk factors and several pathophysiological mechanisms, resulting in overlapping clinical manifestations which target specific organs or multiple organ systems, causing inflammation and tissue damage.

Various genetic and environmental factors have been found to contribute to autoimmune disorders. In almost all patients presenting with an autoimmune disease, the prevalence is increased in first-degree relatives and is even higher in monozygotic twins. While an array of genes, involved in both the innate and adaptive immune response, have been linked to various autoimmune diseases, one of the strongest associations has been with the Major Histocompatibility Complex (MHC) region.

The gene products of the MHC are termed Human Leukocyte Antigens (HLAs) and these play a pivotal role in the antigen presentation of self and non-self peptides and the regulation of innate and adaptive immune responses. HLA profiles can influence autoimmunity risk or protection, by either binding pathogenic- or self-peptides more or less efficiently, resulting in the loss of tolerance.

Various environmental factors, such as smoking, vitamin D deficiency and infections, have been implicated in the development of autoimmunity in genetically susceptible individuals.

DNA Risk: HLA-Related Autoimmunity

DNA Risk: HLA-Related Autoimmunity looks at gene variants related to common autoimmune diseases and provides healthcare practitioners 'diagnostic insight' for patients already suffering from an autoimmune disorder, or who are identified as at-risk due to a family history of disease and exposure to environmental risk factors. This test analyses thousands of gene variants within the MHC and HLA gene region to report on HLA types, specific to HLA class I (HLA-A, -B, -C), and HLA class II (HLA-DPA1, -DPB1, -DQA1, -DQB1, -DRB1, -DRB3, -DRB4, and -DRB5) loci, and MHC related gene variants associated with common autoimmune diseases.

THE AUTOIMMUNE DISEASES DISCUSSED IN THIS REPORT INCLUDE:

- Alopecia areata (AA)
- Ankylosing spondylitis (AS)
- Coeliac disease (CD)
- Graves' disease (GD)
- Hashimoto thyroiditis (HT)
- Idiopathic membranous nephropathy (IMN)
- IgA nephropathy (IgAN)
- Lyme borreliosis
- Multiple sclerosis (MS)
- Rheumatoid arthritis (RA)
- Systemic lupus erythematosus (SLE)
- Vitiligo

The report provides insights into environmental contributors and highlights the HLA alleles (listed in descending order of associated risk) and MHC related gene variants associated with the disorder based on the available literature.

* A known probabilistic algorithm is used to assign each of the HLA alleles. The probability score is the probability that the corresponding call is correct. A threshold combined probability score of 0.7 is used.

* HLA risk alleles are described according to the published literature, where certain risk alleles are reported in specific population groups. Detail on population-specific HLA risk alleles can be viewed in the 'HLA and Autoimmune Diseases' white paper.

Genotype Results

HLA CLASS I		
A	01:01	24:02
B	07:02	08:01
C	07:01	07:02

HLA CLASS II		
DPA1	01:03	01:03
DPB1	04:01	04:02
DQA1	01:10	05:01
DQB1	02:01	06:03
DRB1	03:01	13:01

No risk variant detected Risk variant detected

MHC and HLA SNPs associated with Idiopathic Membranous Nephropathy (IMN)

RS NUMBER	GENOTYPE	GENE IMPACT
rs2187668	TC	+
rs9275596	CC	+
rs2856717	AA	+
rs7763262	TC	+

MHC and HLA SNPs associated with IgA Nephropathy (IgAN)

RS NUMBER	GENOTYPE	GENE IMPACT
rs9275224	AA	○
rs2856717	AA	○
rs9275596	CC	○
rs660895	AA	○
rs7763262	TC	+
rs1883414	GG	+
rs2523946	CC	+

Autoimmune Disorders

Alopecia Areata (AA)

AA is one of the most prevalent autoimmune diseases. In AA, abnormal immune damage targeted to the hair follicle results in non-scarring hair loss that typically begins as patches, which can increase in size and progress to cover the entire scalp (alopecia totalis, AT), and body (alopecia universalis, AU).

RISK ALLELES

Your genotype results:

HLA CLASS II		
DRB1	03:01	13:01

HLA risk genotypes:

HLA CLASS II
DRB1*04:01
DRB1*16

CONTRIBUTING ENVIRONMENTAL EXPOSURE

Vitamin D deficiency may be a risk factor for the development of AA as it can result in over-secretion of IFN-γ and play a role in the collapse of immune privilege of the anagen hair bulb due to the increased follicular expression of MHC class I and II molecules. Vitamin D exerts its effect via the vitamin D receptors (VDR), which are integral in maintaining hair follicle integrity.

IMPROVING OUTCOMES: DIET & LIFESTYLE

Attaining and maintaining adequate vitamin D levels is linked with beneficial autoimmune outcomes. Early evidence suggests that treatment with calcipotriol 0.005% ointment (50 ug calcipotriol monohydrate/mL), a synthetic derivative of vitamin D, may yield promising results.

Ankylosing Spondylitis (AS)

AS is an immune-mediated, rheumatic disease characterised by inflammatory spinal pain and gradual immobility. Patients with AS have an increased risk of developing inflammatory bowel disease, acute anterior uveitis, and psoriasis. They are also prone to cardiovascular disease and pulmonary complications.

RISK ALLELES

Your genotype results:

HLA CLASS I		
B	07:02	08:01

HLA risk genotypes:

HLA CLASS I

B*27:05
B*27:04
B*27:02
B*27:07

CONTRIBUTING ENVIRONMENTAL EXPOSURE

Infection with enteropathic pathogens, such as *Klebsiella pneumoniae* may trigger the development of AS in genetically susceptible individuals.

IMPROVING OUTCOMES: DIET & LIFESTYLE

Attaining and maintaining adequate vitamin D levels, following a low-starch diet, and incorporating regular therapeutic exercise into a healthy management plan has been shown to improve outcomes.

Coeliac Disease (CD)

CD is one of the more common autoimmune disorders, mainly affecting the small bowel and is triggered by gluten ingestion in genetically susceptible individuals. Clinical manifestation of CD may present as classical (malabsorption, diarrhoea, weight loss) or non-classical, extra-intestinal symptoms (iron-deficiency anaemia, ataxia, dermatitis herpetiformis, dental enamel hypoplasia), while some individuals remain asymptomatic.

RISK ALLELES

Your genotype results:

HLA CLASS II		
DQA1	01:10	05:01
DQB1	02:01	06:03

HLA risk genotypes:

HLA CLASS II
DQA1*05; DQA1*05 with DQB1*02; DQB1*02
DQA1*03 with DQB1*03:02
DQA1*02 with DQB1*02
DQA1*05 with DQB1*03:01

CONTRIBUTING ENVIRONMENTAL EXPOSURES

CD is triggered, in genetically susceptible individuals, by the ingestion of gliadin, a component of gluten (found in wheat), as well as other prolamins found in barley, and rye. Female sex, a pro-autoimmune genetic background, viral infections, an inappropriate adaptive immune response, and an imbalanced gut microbiome, may also play a role in the pathogenesis to CD.

IMPROVING OUTCOMES: DIET & LIFESTYLE

A gluten free diet (GFD), which entails strict avoidance of all products containing the prolamin proteins from wheat (gliadin), barley and rye, is the only effective treatment for CD. Screen and correct nutritional deficiencies. Following a GFD under the guidance of a registered dietician who is familiar with CD is strongly advised.

Graves' Disease (GD)

GD is an autoimmune disorder that affects the thyroid, typically presenting in patients between the ages of 40 and 60 years. It is the most common cause of hyperthyroidism, affecting 1.0 – 1.6% of the general population and results from a failure to maintain immune tolerance to thyroid antigens. GD is also associated with goiter, ophthalmopathy and dermopathy.

RISK ALLELES

Your genotype results:

HLA CLASS I		
B	07:02	08:01
C	07:01	07:02
HLA CLASS II		
DPB1	04:01	04:02
DQA1	01:10	05:01
DRB1	03:01	13:01

HLA risk genotypes:

HLA CLASS I
B*08
B*35:01
B*46:01
C*07

HLA CLASS II
DRB1*03
DQA1*05
DPB1*05:01
DRB1*08
DRB1*14:03
DRB1*15:01
DRB1*16:02

In those carrying both the *HLA-DRB1*07* and the *HLA-DRB1*03* risk alleles, the **protective effect** of *HLA-DRB1*07* appears to cancel out *HLA-DRB1*03* susceptibility.

CONTRIBUTING ENVIRONMENTAL EXPOSURES

A key environmental trigger for GD, is infection by Yersinia Enterocolitica (YE) due to the high-affinity binding sites of YE for TSH as well as TSHR antibodies. High exposure to thiocyanate, nicotine and benzopyrene from heavy cigarette smoking is known to affect thyroid function and decrease the levels of TSH. Vitamin D deficiency increases GD risk and recurrence as it results in the hyperactivation of B cells, an increased production of autoantibodies and suppressed regulatory T cell (Treg) function. Iodine deficiency is another risk factor for developing GD as it is required for the production of thyroid hormone.

IMPROVING OUTCOMES: DIET & LIFESTYLE

GD hyperthyroidism is treated by reducing thyroid hormone synthesis, using anti-thyroid drugs (ATD), reducing the amount of thyroid tissue with radioactive iodine (RAI) treatment or total thyroidectomy. Attain and maintain adequate iodine and vitamin D levels.

Hashimoto Thyroiditis (HT)

HT also known as chronic lymphocytic thyroiditis or goitrous autoimmune thyroiditis, is the most common autoimmune thyroid disorder and is characterised by the destruction of thyroid tissue by antibody-mediated immune processes. The diagnosis of HT is challenging in that signs, symptoms and laboratory findings may show normal to hyperthyroid values, due to the destruction of thyroid cells being intermittent early on in the disease.

RISK ALLELES

Your genotype results:

HLA CLASS I		
A	01:01	24:02
HLA CLASS II		
DQA1	01:10	05:01
DQB1	02:01	06:03
DRB1	03:01	13:01

HLA risk genotypes:

HLA CLASS I

A*2
A*02:07

HLA CLASS II

DRB1*03
DRB1*04
DRB1*04:03
DRB1*08
DQA1*03
DQB1*04

DQA1*03:011
DQA1*03:012
DQA1*04:01
DQB1*03:01
DQB1*03:04
DQB1*03:03

CONTRIBUTING ENVIRONMENTAL EXPOSURES

Excess dietary iodine intake, deficiency of vitamin D and selenium, and intestinal bacterial dysbiosis can cause insults to the thyrocytes, resulting in the exposure of new or cryptic epitopes on thyroglobulin (TG) and thyroid peroxidase (TPO) molecules, which then go on to serve as autoantigens.

IMPROVING OUTCOMES: DIET & LIFESTYLE

Medical management with thyroid hormone replacement is the fundamental treatment in HT, with levothyroxine (L-T4) treatment the established first choice as a standard of care. Nutrition can support the medical treatment of HT. Ensure adequate good-quality protein intake, attain and maintain optimal nutritional status of zinc, magnesium, selenium and vitamin D. Increase intake of mono- and omega 3 poly- unsaturated fat sources. It may be prudent to consider avoidance of lactose and gluten-containing foods.

IgA Nephropathy (IgAN)

IgAN is the most prevalent primary chronic glomerular disease world-wide. The diagnosis is made by kidney biopsy, which shows predominant deposition of IgA-containing immune complexes in the glomerular mesangium, leading to glomerulonephritis, glomerular sclerosis and progressive loss of kidney function.

RISK ALLELES

Your genotype results:

HLA CLASS I		
A	01:01	24:02
B	07:02	08:01
HLA CLASS II		
DQA1	01:10	05:01
DQB1	02:01	06:03

HLA risk genotypes:

HLA CLASS I	HLA CLASS II	
A*11:01	DQB*05:01	DQA1*01:01
B*40:01	DQB1*03:01	DQB*03:02

IgAN associated MHC & HLA SNPs:

RS NUMBER	GENOTYPE	GENE IMPACT
rs9275224	AA	
rs2856717	AA	
rs9275596	CC	
rs660895	AA	
rs7763262	TC	+
rs1883414	GG	+
rs2523946	CC	+

CONTRIBUTING ENVIRONMENTAL EXPOSURES

Helminth invasion and mucosal infections from bacteria such as *Staphylococcus aureus (S. aureus)* are a trigger for IgAN in genetically susceptible individuals, where increased synthesis of poorly O-galactosylated IgA1 (Gd-IgA1) in circulation leads to production of autoantibodies against Gd-IgA1, and the formation of immune complexes containing pathogenic O-galactosylated IgA1, resulting in mesangial deposition of these immune complexes activating mesangial cells and subsequently impairing glomeruli.

IMPROVING OUTCOMES: DIET & LIFESTYLE

Lifestyle modifications include dietary sodium restriction due to associated sodium-sensitive hypertension, and regular (3 times per week), moderate intensity, physical exercise, which is associated with a decreased risk of end stage renal function. Weight management is imperative to avoid accelerated progression and outcomes of IgAN related to being overweight.

Idiopathic Membranous Nephropathy (IMN)

IMN is the most common cause of nephrotic syndrome in adults and is now recognised as an organ–specific autoimmune disease. It is characterised by immune complex depositions on the extra-capillary side of glomerular basement membrane.

RISK ALLELES

Your genotype results:

HLA CLASS II		
DRB1	03:01	13:01

HLA risk genotypes:

HLA CLASS II
DRB1*03:01
DRB1*15:01

IMN associated MHC & HLA SNPs:

RS NUMBER	GENOTYPE	GENE IMPACT
rs2187668	TC	+
rs9275596	CC	+
rs2856717	AA	+
rs7763262	TC	+

CONTRIBUTING ENVIRONMENTAL EXPOSURES

Bacterial infections, such as those of the Clostridium species, and a high level of exposure to reactive oxygen species (ROS) produced by polycyclic aromatic hydrocarbons and transition metals, facilitate production of PLA2R1 (M-type phospholipase A2 receptor) antibodies, leading to an altered immune response in genetically susceptible individuals, and thus may be a trigger for IMN.

IMPROVING OUTCOMES: DIET & LIFESTYLE

In terms of lifestyle modifications, it is advised that patients decrease their sodium intake to 3g per day, and restrict dietary protein intake to 0.8g/kg body weight/day, which has been shown to slow the progression of renal disease.

Lyme Borreliosis

Lyme borreliosis, which is caused by the bacterial agent, *Borrelia burgdorferi*, transmitted to the host by a bite from the *Ixodes* tick, is regarded as a multi-system, inflammatory disease that affects the skin, nervous system, cardiovascular system, muscles and joints. While most patients present at an early stage of infection and can be treated with antibiotics, a subset of patients will go untreated and develop Lyme arthritis months after being infected. Lyme arthritis can be successfully treated with antibiotic therapy, however in some cases, persistent and excessive joint inflammation continues for several months to years after antibiotic therapy and this is termed antibiotic-refractory Lyme arthritis. The refractory outcome is likely an interplay between the pathogen, host genetic and immunologic factors.

RISK ALLELES

Your genotype results:

HLA CLASS II		
DRB1	03:01	13:01

HLA risk genotypes:

HLA CLASS II

DRB1*01:01
DRB1*01:02
DRB1*03:05
DRB1*04:01
DRB1*04:02
DRB1*04:04
DRB1*04:05
DRB1*16:01
DRB1*17

CONTRIBUTING ENVIRONMENTAL EXPOSURES

Borrelia burgdorferi, transmitted to the host by a bite from the *Ixodes* tick, is the cause of Lyme borreliosis. With regards to antibiotic-refractory Lyme arthritis, retained *B. burgdorferi* antigens may play a role in inflammation. In addition, immune reactivity with multiple autoantigens, as well as excessive inflammation and immune dysregulation are probably required. Hypercholesterolemic patients, where cholesterol is more accessible for *Borrelia* to acquire it, may experience more severe, prolonged symptoms.

IMPROVING OUTCOMES: DIET & LIFESTYLE

Besides the use of antimicrobial agents as first line therapy for Lyme disease, and the extended courses of antibiotics recommended at multiple dosages and varying durations for refractory Lyme arthritis, symptomatic treatment with nonsteroidal anti-inflammatory agents or corticosteroids is common practice.

The cholesterol status of patients who present with Ixodes tick bite should be noted for disease outcome and management purposes. Dietary modification to manage cholesterol levels is advised.

Multiple Sclerosis (MS)

Multiple sclerosis (MS) is a T-cell–mediated, chronic autoimmune disease of the central nervous system (CNS), which is characterised by inflammation, demyelination, and neurodegeneration. The course of MS is highly varied and unpredictable, where the disease is characterised initially by episodes of reversible neurological deficits, followed by progressive neurological deterioration over time.

RISK ALLELES

Your genotype results:

HLA CLASS II		
DQB1	02:01	06:03
DRB1	03:01	13:01

HLA risk genotypes:

HLA CLASS II
DRB1*15:01
DRB1*03:01
DQB1*06:02

CONTRIBUTING ENVIRONMENTAL EXPOSURES

Exposure to viral agents such as *Epstein Barr virus (EBV)*, and nitric oxide, through active or passive smoking, have been associated with the onset of MS in genetically susceptible individuals. The foreign agents may have a nuclear antigen that is structurally homologous with myelin sheet components. Thus, when immune cells are activated by these pathogens, myelin sheath lesions will form. Vitamin D deficiency, via modulation of inflammation and immune response is a contributing factor, as is deficiency in folate or vitamin B12 that may result in raised homocysteine (hcy) levels, which plays a role in myelin sheath degeneration.

IMPROVING OUTCOMES: DIET & LIFESTYLE

Regular exercise training is associated with a small improvement in walking mobility in MS sufferers. Preliminary evidence suggests that green tea catechins such as (2)epigal-locatechin-3-gallate (EGCG) may improve symptoms of fatigue, common in MS, while vitamin D supplementation was shown to reduce gadolinium-enhancing lesions. Daily intake of a multi-nutrient formula containing a balanced mixture of specific omega-3 and omega-6 poly-, and mono-unsaturated fatty acids (PUFAs and MUFAs), and saturated fatty acids (SFAs), vitamin A, vitamin E and γ-tocopherol can significantly reduce the annualised relapse rate and the risk of sustained disability progression.

Rheumatoid Arthritis (RA)

RA is a common, chronic inflammatory, autoimmune disease that is characterised by synovitis and the localized destruction of cartilage and bone. Affected individuals gradually develop articular damage and functional disability.

RISK ALLELES

Your genotype results:

HLA CLASS II		
DPB1	04:01	04:02
DRB1	03:01	13:01

HLA risk genotypes:

HLA CLASS II

High-risk shared epitope (SE) alleles:

DRB1*04:01
DRB1*04:04
DRB1*10
DRB1*01:01
DRB1*04:05
DRB1*04:10
DRB1*01

Novel risk alleles:

DPB1*04:01
DPB1*06:01

CONTRIBUTING ENVIRONMENTAL EXPOSURES

There is a strong association between smoking and RA risk in genetically susceptible individuals, where smoking enhances SE-dependent immune reaction to citrullinated proteins, enhancing anti-cyclic citrullinated peptide formation. Excess weight and obesity may contribute to RA predisposition via increased inflammation and raised oestrogen levels. Infections from *Porphyromonas gingivalis (P. gingivalis), Proteus mirabilis (P. mirabilis),* and *Epstein–Barr virus (EBV*) have been found to contribute to the pathogenesis of RA. A diet that drives inflammation and disturbes intestinal microbial homeostasis has also been considered a risk factor for RA.

IMPROVING OUTCOMES: DIET & LIFESTYLE

Smoking cessation could reduce seropositive RA risk, but it will not fully negate the risk. Exercise therapy (>20 minutes/day, >1 hour/week) can modulate the Th1/Th2 and natural killer cells levels and produce hormones including epinephrine and norepinephrine, lowering systemic inflammation. Improving gut microbiota, following a fibre-rich, Mediterranean style diet is related to improved patient outcomes.

Systemic Lupus Erythematosus (SLE)

SLE is a multi-system, chronic, autoimmune inflammatory disorder, affecting women of child-bearing age, with a female to male ratio of 9:1. SLE presents with diverse clinical manifestations ranging from autoimmune haemolytic anaemia, leukopenia, and thrombocytopenia to nephritis, arthritis, dermatitis and neuropsychiatric involvement.

RISK ALLELES

Your genotype results:

HLA CLASS I		
A	01:01	24:02
B	07:02	08:01
HLA CLASS II		
DQA1	01:10	05:01
DQB1	02:01	06:03
DRB1	03:01	13:01

HLA risk genotypes:

HLA CLASS I
A*29
B*51

HLA CLASS II
DQA1*01:02
DRB1*03:01
DRB1*15:01
DRB1*07
DRB1*08:01
DRB1*08:03
DRB1*09:01
DQB1*02
DQB1*06:02
DQB1*06
DQB1*03:01

The **protective effects** of *HLA-DRB1*13:02* and *HLA-DRB1*14:03* are dominant over the predisposing risk effects of *HLA-DRB1*15:01*

CONTRIBUTING ENVIRONMENTAL EXPOSURES

Environmental factors that have been suggested to be associated with the development of SLE in genetically susceptible individuals include exposure to silica, current smoking, vitamin D deficiency and *Epstein Barr Virus (EBV)* infection.

IMPROVING OUTCOMES: DIET & LIFESTYLE

Attaining and maintaining optimal vitamin D levels may be effective in decreasing disease activity and improving fatigue. A traditional Mediterranean diet, relatively low in protein, high in fibre, polyunsaturated fatty acids, vitamins (A, B, C, D, and E), minerals (calcium, zinc, selenium, iron and copper) and polyphenols has sufficient potential to regulate the activity of the overall disease by modulating the inflammation and immune functions of SLE.

Vitiligo

Vitiligo, being autoimmune in nature, is a common depigmenting skin condition, characterised by a loss of melanocytes, which results in the appearance of milky white patches on the skin. Vitiligo frequently occurs with other autoimmune diseases, such as RA, adult-onset type 1 diabetes mellitus, psoriasis, Addison's disease and SLE.

RISK ALLELES

Your genotype results:

HLA CLASS I		
A	01:01	24:02
B	07:02	08:01
HLA CLASS II		
DRB1	03:01	13:01

HLA risk genotypes:

HLA CLASS I
A*02
A*33
A*33:01
B*13
B*27
B*44:03

HLA CLASS II
DRB1*07:01

CONTRIBUTING ENVIRONMENTAL EXPOSURES

Exposure to phenols or some form of skin trauma has the ability to disrupt melanogenesis resulting in autoimmunity and melanocyte destruction.

Chemical-induced depigmentation of the skin, through occupational hazards and household commercial skin-care products, has been recognised for over half a decade.

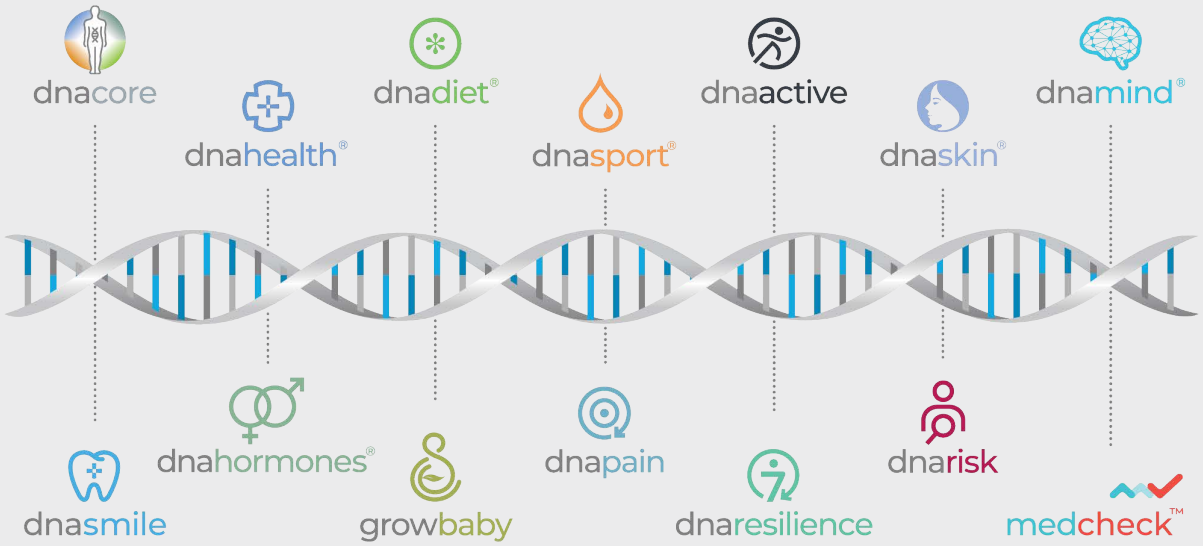
IMPROVING OUTCOMES: DIET & LIFESTYLE

Certain foods high in polyphenols such as mango, cashew, pistaschio, oak, cassava, areca nut, red chillies, cherry, raspberry, cranberry, blackberry and tea, have the ability to exacerbate and aggravate vitiligo, and should be reduced or avoided.

Attaining and maintaining optimal vitamin D levels has been shown to improve pigmentation.

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 technology | ACTIONABLE interventions | APPROPRIATE 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.