GBlongevity 100

Your Genetic Blueprint for Longevity

LONGEVITY PROFILE **Identify modifiable risk factors and enable personalized preventive treatment**

Your Longevity Index

EXCELLENT
GOOD ✓
AVERAGE
AT RISK

Your Health Insights

Comprehensive Testing

- Longevity Genetic Index PANEL
- Lipid Disorders and Heart Health COMPREHENSIVE PANEL
- Obesity COMPREHENSIVE PANEL
- Alzheimer's Disease COMPREHENSIVE PANEL
- Type 2 Diabetes COMPREHENSIVE PANEL
- Nutritional Geno PANEL

Your Action Plan

WEB AND MOBILE APP

Empower yourself with genetic insights to steer your health towards its optimal trajectory and potentially live to 100!

Clinical Case Studies

Join the Revolution

Trusted by leading cardiologists, lipidologists, and endocrinologists.

- Agatston Center for Preventive Medicine
- Baylor College of Medicine
- Baylor Scott & White Health
- Beth Israel Deaconess Medical Center
- Cedars-Sinai Medical Center
- Columbia University Medical Center
- Emory University School of Medicine
- Michigan Medicine at the University of Michigan
- Mount Sinai, Icahn School of Medicine
- New York University School of Medicine
- OhioHealth Physician Group
- Sutter Health
- The Children's Heart Clinic, Children's Minnesota
- Tulane University School of Medicine
- University of California San Diego School of Medicine
- University of Chicago School of Medicine
- University of Colorado Anschutz Medical Center
- University of Kansas School of Medicine
- University of Pennsylvania School of Medicine
- University of Texas Southwestern Medical Center
- Veterans Affairs Health System
- Weill Medical College of Cornell University

Hear from world-class experts on leveraging GB genetic testing for precision medicine and preventive health.

Recorded webinar videos: **bit.ly/gbinsight-webinars**

Personalized Lipidology: How to utilize genomic testing to diagnose and better manage lipid disorders

Dr. Michael H. Davidson
University of Chicago

Solving Clinical Dilemmas Using Genetic Testing

Dr. Arthur Agatston
The Agatston Center for Preventive Medicine

Is there evidence for expanded genetic testing in evaluation of patients with severe hypertriglyceridemia?

Dr. Cristie M. Ballantyne
Baylor College of Medicine

Hiding in Plain Sight: What genetic testing adds to the diagnosis and management of lipid disorders

Dr. James Underberg
NYU Grossman School of Medicine

Why Does a Genetic Diagnosis of Familial Hypercholesterolemia Matter?

Dr. Robert Rosenson
Icahn School of Medicine - Mt. Sinai

Letter to Physicians - Stay Ahead in Your Field

GB Longevity100 is a proactive genetic screening suite designed to help individuals pursue optimal health and extend lifespan through evidence-based methods.

The value of genetic testing lies in its ability to identify at-risk individuals early, allowing for preventive measures that can reduce or even eliminate the risk of disease. Research shows that early treatment of genetic conditions like familial hypercholesterolemia (FH) and high Lp(a) can extend health span by 15–20 years. The same applies to Alzheimer's dementia and other metabolic diseases.

GB Longevity100 analyzes six comprehensive genetic panels:

- Longevity Genetic Index Panel
- Lipid Disorders and ASCVD Panel
- Type 2 Diabetes Panel
- Obesity Panel
- Alzheimer's Disease Panel
- Nutritional Genomics Panel

This booklet presents two case studies with the following health profiles:

Case 1: A 20-year-old with a family history of early heart disease.
Case 2: A 101-year-old in excellent health.

The genetic analysis in these cases illustrates how genetic testing can uncover modifiable risk factors and provide an individualized action plan to mitigate those risks.

GB HealthWatch Team

(858) 788-9274
support@gbhealthwatch.com

Scan for test information.

bit.ly/gbinsight-doctor-intro

A 20-Year-Old with a Family History of Early Heart Disease

CASE STUDY 1

Patient Information

Age: 23

Sex: Male

The following is a sample of our GB Longevity100 Summary Report

View the online report to access the built-in knowledge base and useful links: **bit.ly/longevity100-report**

Longevity Profile

Your Genetic Index for Longevity

AT RISK

Congratulations on uncovering the genetic risk factors hidden in your DNA - a crucial step toward attaining optimal health and longevity! Taking preventive steps as early as possible in life can reduce the risk of chronic diseases, maximizing both healthspan and lifespan. Check out your personalized Action Plan for guidance on lowering your risks.

Report Summary

- **11** advantage SNPs and **3** disadvantage SNPs identified in Longevity Associated Genes
- **1** key finding(s) and **2** high risk pathway(s) identified in Heart and Vascular Health
- **1** high risk pathway(s) identified in Metabolic Health
- **1** key finding(s) identified in Cognitive Health
- **1** high risk pathway(s) identified in Diet-Nutrition and Digestive System Health
- **11** action items are recommended
- Pathogenic/likely pathogenetic variants: **1** identified

ⓘ This report is not intended to diagnose any condition and should only be used for educational or informational purposes.

ⓘ The GB-Longevity100 index provides a qualitative assessment of the likelihood of living longer based on genetic analysis of cardiometabolic diseases and dementia risk. This index does not encompass genetic analysis of cancer syndromes or other diseases that may significantly affect health and lifespan. It is important to note that lifespan and healthspan are not determined solely by genetics; factors such as diet, lifestyle, emotional well-being, risk-taking behaviors, and even luck also play critical roles in influencing the likelihood of living a longer and healthier life.

* This index calculates the cumulative effects of variants that increase or decrease longevity and presents it as a relative grade compared to your reference population.

Longevity Profile

Genetic Variants Associated with Longevity: 34 identified

Variants identified	SNP	Genotype	Effect	Impact to Longevity
SH2B3 (c.784T>C(p.Trp262Arg)) *missense*	rs3184504	C/C	Advantage	Lower blood pressure
ANKK1 (c.2137G=(p.Glu713=)) *homozygous_reference_allele*	rs1800497	G/G	Advantage	Reduced risk of obesity
CELSR2 (c.*919G>T) *3_prime_UTR*	rs12740374	G/T	Advantage	Reduced risk of heart disease
FOXO3 (c.621+25486G>T) *intron_variant*	rs2802292	G/T	Advantage	Association with increased longevity
GPX1 (c.599C>T(p.Pro200Leu)) *missense*	rs1050450	A/A	Advantage	Lower BMI and blood pressure
HNF1A (c.1501+119G=) *homozygous_reference_allele*	rs2259816	G/G	Advantage	Association with increased longevity
NOS3 (c.894T>G(p.Asp298Glu)) *missense*	rs1799983	G/G	Advantage	Lower blood pressure
PLCE1 (c.5780A>G(p.His1927Arg)) *missense*	rs2274223	A/G	Advantage	Lower blood pressure
ACE (c.2328G>A(p.Thr776=)) *synonymous_codon*	rs4343	G/A	Advantage	Reduced risk of hypertension
ADRB1 (c.145A>G(p.Ser49Gly)) *missense*	rs1801252	A/G	Advantage	Lower blood pressure
IL6 (7:g.22727026C>G) *upstream_variant*	rs1800795	C/G	Advantage	Association with increased longevity
LDLR (Exon2-5) *copy_number_loss*	NA	exon deletion	Disadvantage	Familial hypercholesterolemia
LPA (c.3947+467T>C) *intron_variant*	rs10455872	A/G	Disadvantage	Elevated Lp(a)
APOE (E4 (p.Cys130Arg)) *missense*	rs429358	T/C	Disadvantage	Alzheimer's disease risk
ABCG8 (c.55G=(p.Asp19=)) *homozygous_reference_allele*	rs11887534	G/G	Neutral	
APOA5 (c.*158C>T) *3_prime_UTR*	rs2266788	A/A	Neutral	
APOA5 (c.56C=(p.Ser19=)) *homozygous_reference_allele*	rs3135506	G/G	Neutral	
APOE (c.526C=(p.Arg176=)) *homozygous_reference_allele*	rs7412	C/C	Neutral	
BUD13 (c.358C=(p.Arg120=)) *homozygous_reference_allele*	rs10488698	G/G	Neutral	
CDKN2B-AS1 (9:g.22124478A=) *homozygous_reference_allele*	rs10757278	A/A	Neutral	
CETP (c.1264G>A(p.Val422Ile)) *missense*	rs5882	G/A	Neutral	
CHRNA3 (c.645C=(p.Tyr215=)) *homozygous_reference_allele*	rs1051730	G/G	Neutral	
CYP17A1 (c.1621+39238G=) *homozygous_reference_allele*	rs12413409	G/G	Neutral	
KL (c.1054T=(p.Phe=)) *homozygous_reference_allele*	rs9536314	T/T	Neutral	
LDLR (c.4521-5330G=) *homozygous_reference_allele*	rs1122608	G/G	Neutral	
LDLR (c.67+2015G=) *homozygous_reference_allele*	rs6511720	G/G	Neutral	
LDLR (c.81C=(p.Cys27=)) *homozygous_reference_allele*	rs2228671	C/C	Neutral	
MTHFR (c.665C=(p.Ala222=)) *homozygous_reference_allele*	rs1801133	G/G	Neutral	
MTTP (c.1981G=(p.Gly=)) *homozygous_reference_allele*	rs113337987	G/G	Neutral	
PCSK9 (c.158C=(p.Ala=)) *homozygous_reference_allele*	rs11583680	C/C	Neutral	
PCSK9 (c.1420G>A(p.Val474Ile)) *missense*	rs562556	A/A	Neutral	
PCSK9 (g.55030366T=) *homozygous_reference_allele*	rs11206510	T/T	Neutral	
PHACTR1 (c.251-149640A>G) *intron_variant*	rs9349379	A/G	Neutral	
SORL1 (c.1582G=(p.Ala=)) *homozygous_reference_allele*	rs2298813	G/G	Neutral	

Including genetic analysis reports for six panels:

Please view the following reports from the online report: bit.ly/longevity100-report

Dyslipidemia and ASCVD Comprehensive Report

Type 2 Diabetes Comprehensive Report

Obesity Comprehensive Report

Alzheimer's Disease Comprehensive Report

Nutritional Genomics Comprehensive Report

GB Longevity100 Summary Report

Health Insights

Pathogenic/likely pathogenetic variants

1 identified

Variant	Type	Genotype	dbSNP/ClinVar	Phenotype/Disease	Classification
Hypercholesterolemia and LDL cholesterol					
LDLR (Exon2-5) *copy_number_loss*	CNV	Heterozygous	NA	Familial hypercholesterolemia. (Autosomal dominant)	Pathogenic

Heart and Vascular Health (Lipid Disorders and ASCVD)

Focused Pathways:

Hypercholesterolemia	**High risk**
Hypertriglyceridemia	**Moderately high risk**
High lipoprotein (a) levels	**High risk**
Defective reverse cholesterol transport	**Risk not identified**
Sensitivity to sodium, hypertension	**Risk not identified**

See genetic details in Dyslipidemia and ASCVD Comprehensive Report

Key finding:

One pathogenic/likely pathogenic variant in the LDLR gene was detected that is indicative of heterozygous familial hypercholesterolemia (HeFH). Additionally a genetic risk for high Lp(a) was detected.

Pathogenic/likely pathogenic variants in the LDLR gene accounts for 90% of monogenic FH. HeFH puts this individual at high risk of atherosclerotic cardiovascular disease (ASCVD), particularly if untreated. People with mutation-positive HeFH are at

risk of atherosclerotic cardiovascular disease (ASCVD), particularly if untreated. People with mutation-positive HeFH are at higher (3-fold) risk of ASCVD compared to those without an FH mutation detected; this higher ASCVD risk is independent of LDL-cholesterol levels (PMID: 35143253, 27050191) and is due to the long duration of exposure to hypercholesterolemia. High Lp(a) in people with HeFH is an ASCVD risk-enhancing factor (PMID: 30071997, 34433300). High Lp(a) is a causal risk factor for atherosclerotic cardiovascular diseases (ASCVD) and calcific aortic valve stenosis (CAVS). There is a linear relationship between serum Lp(a) concentrations and risk of ASCVD and CAVS. HeFH is inherited in an autosomal dominant manner (requiring just a single P/LP variant) and therefore there is a 50% chance that other biological family members will also carry this genetic variant and have FH. Guidelines recommend: 1) Genomic cascade screening for biological family members (supported by Tier 1 evidence per the US Centers for Disease Control and Prevention (CDC) and the National Institute for Health and Clinical Excellence (NICE) in the United Kingdom). 2) Early diagnosis leads to earlier initiation of lipid-lowering therapies that studies have shown reduces ASCVD risk in midlife (PMID: 31618540).

Precision Medicine: Based on multiple cardiovascular outcome trials, such as ODYSSEY (alirocumab), OSLER and FOURIER (evolocumab) and ORION (inclisiran), the US Food and Drug Administration (FDA) approved PCSK9 inhibitors (PCSK9i), on top of diet and maximally-tolerated statins and/or ezetimibe for the treatment of HeFH (PMID: 25773607, 30403574, 32197277). Based on data from the HAUSER-RCT trial, the US FDA approved the PCSK9i, evolocumab, for the treatment of HeFH in pediatric populations ages 10-17 years (PMID: 32865373). Bempedoic acid is approved based on the CLEAR Outcomes trial (PMID: 36876740). The importance of earlier and sustained low LDL-Cholesterol levels comes from 20-year follow-up study showing that the earlier someone with HeFH is started on lipid-lowering therapies associates with reduced cardiovascular disease risk (PMID: 31618540). According to the LAPLACE, ODYSSEY and FOURIER trials, PCSK9i are shown to modestly (20-25%) reduce Lp(a) levels (PMID: 27102113, 23884353, 31948641, 30586750). Lipoprotein apheresis is currently the only FDA-approved therapy for high Lp(a) with ASCVD (PMID: 34647487).

*Key findings are for educational purposes only. We defer all medical decision making to the treating physician(s).

Metabolic Health (Diabetes, Obesity and NAFLD)

Focused Pathways:

Beta cell development and dysfunction	**High risk**
Glucose sensing and insulin secretion	**Risk not identified**
Insulin resistance	**Average risk**
Susceptibility to autoimmune diseases	**Risk not identified**
Increased appetite due to low satiety, obesity	**Average risk**
Non-Alcoholic Fatty Liver Disease (NAFLD)	**Risk not identified**

See genetic details in Type 2 Diabetes Comprehensive Report, Obesity Comprehensive Report

Cognitive Health (Alzheimer's)

CASE STUDY 1

APOE status: Increased risk*

rs429358	rs7412	APOE Genotype
CT	CC	**E3/E4**

* Among Caucasian subjects, the risk of Alzheimer's disease (AD) increased significantly with increasing APOE-E4 dose. The odds ratios (OR) were 2.6, 3.2 and 14.9 for APOE genotypes E2/E4, E3/E4 and E4/E4, respectively, whereas the OR decreased for genotypes E2/E2 and E2/E3 (OR=0.6). The association between APOE-E4 and AD was weaker among African Americans and Hispanics and much stronger among Japanese populations (OR=5.6 for E3/E4, OR=33.1 for E4/E4).

Nutritional Pathways Related to Cognitive Health:

Folate deficiency	**Risk not identified**
Vitamin B12 deficiency	**Increased risk**
Iron overload	**Risk not identified**
Functional vitamin D deficiency	**Risk not identified**

See genetic details in Alzheimer's Disease Comprehensive Report

Key finding:

Heterozygosity for the APOE4 allele that is a genetic risk factor for Alzheimer's disease (AD), was detected. The APOE4 allele is not sufficient to cause AD (is not pathogenic), but is a genetic risk factor.

Heterozygosity for the APOE4 allele is not pathogenic and is neither necessary nor sufficient for the development of Alzheimer's disease (AD). With that said, the APOE4 allele is the most important genetic risk factor for AD, in part because its relatively common in the general population (20%), depending on race/ethnicity. But risk of AD is not uniform across all populations for APOE4 heterozygotes. Caucasians and Asians have a 3-fold higher risk of developing AD compared to people who are homozygous for the neutral APOE3 allele. But people who are of African or Hispanic ancestry have lower risk of AD stemming from the APOE4 allele (PMID: 9508150, 18344711, 24632849). The APOE gene encodes apolipoprotein E (apoE) that embeds within lipid-carrying lipoproteins. In the central nervous system, apoE is the major protein found on lipid-carrying particles. ApoE facilitates the uptake of these lipoproteins by neurons. The APOE4 allele contributes to multiple pathological pathways that work together to increase risk for AD, such as increased amyloid beta production and aggregation, reduced clearance of amyloid plaques, hyperphosphorylation of tau and accumulation of neurofibrillary tangles, increased neuroinflammation, glucose dysregulation, blood brain barrier dysfunction and more (PMID: 31367008, 33340485). Heterozygosity for the APOE4 allele increases risk of Alzheimer's disease, but is not sufficient itself. Other genetic or non-genetic factors are likely necessary to cause Alzheimer's disease.

Precision Medicine: The pathology leading to Alzheimer's disease (AD) develops over decades, which means that early interventions can mitigate risk. Consensus indicates that AD risk reduction can be achieved by maintaining optimal vascular health, maintaining normal blood pressure, lipid levels, and glucose, avoid excess body weight, regular exercise, eat fresh fruits and vegetables, social engagement, avoid smoking, and regular sleep. There is some evidence that omega-3 marine oils, such as Docosahexaenoic acid (DHA), may preferentially offer cognitive benefits to people with the APOE4 allele (PMID: 37890592). For people with mild cognitive impairment (MCI) or mild dementia due to Alzheimer's disease, the US Food and Drug Administration (FDA) approved two medications: lecanemab (marketed as Leqembi in the US) and aducanumab (marketed as Aduhelm in the US). Both of these medications are antibodies that target amyloid beta. Individuals carrying the APOE4 allele are at increased risk of developing amyloid-related imaging abnormalities (ARIA), edema and hemorrhage subtypes (PMID: 34807243, 35099507, 35542992). In the combined phase 3 data from EMERGE and ENGAGE trials that

tested clinical efficacy of aducanumab, 8.2% of APOE4 carriers compared to 2.5% of noncarriers experienced symptomatic ARIA - edema and hemorrhage (PMID: 34807243). APOE4 homozygotes have greater risk of ARIA than heterozygotes who have greater risk than non-carriers. But these adverse effects are generally mild, with only 0.3% in the EMERGE and ENGAGE trials considered serious (PMID: 34807243). Similar results were reported with regards to increased risk of ARIA in people with APOE4 allele(s) receiving lecanemab (PMID: 36449413, 37357276).

*Key findings are for educational purposes only. We defer all medical decision making to the treating physician(s).

Diet-Nutrition and Digestive System Health

Focused Pathways:

Choline deficiency	**Average risk**
Folate deficiency	**Risk not identified**
Functional vitamin D deficiency	**Risk not identified**
Vitamin B12 deficiency	**Increased risk**
Long-chain omega-3/6 deficiency	**Risk of omega-6 excess**
Retinol deficiency	**Average risk**
Fat-soluble vitamin deficiency	**Average risk**
Iron overload	**Risk not identified**
Alcohol intolerance/Alcohol flush reaction	**Risk not identified**
Fructose intolerance	**Risk not identified**
Gluten intolerance	**Not likely at risk**
Lactose intolerance	**Likely intolerant**
Sensitivity to a high-carbohydrate diet, non-alcoholic fatty liver disease (NAFLD)	**Risk not identified**
Sensitivity to a high-carbohydrate diet, hypertriglyceridemia	**Slightly increased risk**
Sensitivity to a high-fat diet, hypercholesterolemia	**High risk**
Sensitivity to a ketogenic diet, high absorption of cholesterol and plant sterols	**Risk not identified**
Sensitivity to high cholesterol and high carbohydrate intake, type III hyperlipidemia	**Risk not identified**
Sensitivity to sodium, hypertension	**Risk not identified**
Reduced utilization of long-chain fatty acids for fuel	**Risk not identified**
Sensitivity to caffeine	**Fast metabolizer**

See genetic details in Nutritional Genomics Report

Action Plan

In charge of your health and longevity
Science-based recommendations to help you maximize healthspan and enhance longevity.

1 Empower yourself: take steps to live longer and healthier.

2 Contact your doctor to manage familial hypercholesterolemia (FH) combined with high Lp(a), which is a strong risk factor for heart disease.

Contact your healthcare provider for a consultation regarding familial hypercholesterolemia (FH), a genetic condition with one mutation in a gene involved in removing LDL from the blood that results in very high cholesterol levels. This type of disorder is also known as heterozygous FH (HeFH). Without treatment, FH may lead to premature cardiovascular disease. High Lp(a), on top of high LDL cholesterol levels, further increases the chance of getting heart disease. Medication is effective in managing FH, while diet and lifestyle changes, though beneficial, may not alone achieve the target cholesterol levels. Studies show that the earlier in life cholesterol levels can be lowered with medication (on top of diet), the lower chance of getting heart disease in midlife. There is a 50% chance that a first-degree blood relative will inherit the same FH variant and genetic cause of high Lp(a), therefore it's important to have close family members screened to see if they have FH and high Lp(a).
See details in **Dyslipidemia and ASCVD Comprehensive Report.**

Learn More

- Genetics of High Cholesterol
- Genetics of High Lp(a) and Cardiovascular Disease
- What is Familial Hypercholesterolemia?
- High Lp(a) increases your risk of heart attack, stroke, and aortic stenosis

3 Limit total fat, saturated fat and cholesterol intake to help keep blood cholesterol levels in check.

Your DNA analysis reveals that you may be at high risk for having high blood cholesterol (also known as hypercholesterolemia). This is due to reduced capacity to clear cholesterol from the blood. High total and LDL cholesterol levels can lead to atherosclerosis or narrowing of blood vessels, a leading cause of heart disease. Discuss with your doctor about the best means of reducing risk of hypercholesterolemia. This may require medication and change of diet. Consider a plant-based diet. Aim for less than 25% of total calories from fat and less than 7% from saturated fat. Replace saturated fat from butter and animal fats with healthy, plant-based oils. Eat fatty fish and plant-based proteins, such as tofu and nuts, and limit animal meats, especially red meat. Eat plenty of fresh fruits and vegetables for antioxidants and fiber. Limit cholesterol intake to less than 300 mg/day. Use the HealthWatch 360 app to check your saturated fat and cholesterol intake.
See details in **Nutritional Genomics Report.**

Diet and Nutrition

- Diet and Nutrition to Reduce Blood Cholesterol
- Limit foods high in saturated fat
- Limit foods high in cholesterol
- Watch out for high-fat foods
- Include more fiber-rich foods in your diet

4 Discuss with your healthcare provider about risk reduction for Alzheimer's disease due to APOE4.

Your DNA analysis reveals that you have one APOE allele, which is the most common risk factor for late-onset Alzheimer's disease (LOAD), also known as sporadic Alzheimer's disease. The APOE allele is found in about 20% of the general population. The saying "genetics is not fate" applies here: having one APOE4 allele does not mean you will get Alzheimer's disease. Knowing your increased risk early in life empowers you to take proactive steps to reduce risk as you age. The dementia process begins decades before symptoms appear. Though there is no cure, early prevention is crucial. Adopting a healthy lifestyle in young adulthood, if possible, can significantly lower your risk. This includes regular exercise, abstaining from smoking, following diets like the MIND or Mediterranean diet, controlling blood pressure, and engaging in mentally-challenging activities. Fish oils, particularly DHA, may help preserve cognitive function in those with the APOE4 allele. This is especially relevant if there is strong history of dementia in your family. The earlier you start preventive measures, the better.
See details in **Alzheimer's Disease Comprehensive Report.**

Learn More

- How does vitamin B12 protect the brain from aging?
- Eat right to reduce Alzheimer's disease risk
- Video: APOE gene and diet

Diet and Nutrition

- Nutrition and Lifestyle for Alzheimer's Prevention
- Activities for Alzheimer's Prevention
- MIND Diet for Cognitive Health
- Mediterranean Diet

5 Check your Lp(a) levels with a blood test.

If you haven't had your blood Lp(a) levels checked, contact your doctor to request one. High Lp(a) is the most common heritable risk factor for heart disease; the higher the level of Lp(a), the greater the risk of heart disease. Currently, there is no FDA-approved medication for high Lp(a), but managing other risk factors, such as body weight, blood pressure, blood sugar, achieving lipid goals, and adhering to a healthy lifestyle can help reduce your risk of heart disease. Off-label, some existing medications may moderately lower blood Lp(a) levels. Ask your doctor about these options. There is a 50% chance that a first-degree blood relative will also inherit high Lp(a), therefore it's important to have close family members screened.
See details in **Dyslipidemia and ASCVD Comprehensive Report.**

Learn More

- Genetics of High Lp(a) and Cardiovascular Disease
- High Lp(a) increases your risk of heart attack, stroke, and aortic stenosis

Diet and Nutrition

- Diet and Nutrition to Reduce Blood Cholesterol
- Top Foods to Lower Blood Pressure
- Eat more foods rich in vitamin E, a lipid antioxidant

6 Pancreatic beta cell function pathway risk was identified, which can increase risk of type 2 diabetes.

Your DNA analysis reveals that you may be at high risk for diabetes. Comprehensive genetic analysis by GBinsight indicates a high risk due to pancreatic cell dysfunction. Type 2 diabetes, or persistently high blood sugar levels, is the culmination of multiple processes gone wrong. Pancreatic beta cells are tasked with the production and secretion of insulin, which is the master controller of blood sugar levels. Genetic variants in genes that control the pancreas' insulin production and/or secretion are major risk factors for type 2 diabetes. Notably, a genetic variant (rs7903146) within the TCF7L2 gene is the most consistent genetic risk factor for type 2 diabetes and is involved in pancreatic insulin production. Very rare genetic variants that significantly slow this process is known to cause Maturity-Onset Diabetes of the Young (MODY), which is the most common form of monogenic diabetes. Discuss with your doctor about reducing risk of type 2 diabetes. Carbohydrate restriction and a high protein diet may be a means of preserving pancreatic beta cell function. Also, it's important to avoid excess body weight as this puts more strain on the pancreas. Exercise regularly. Medication may be necessary to keep blood sugar levels in check.
See details in **Type 2 Diabetes Comprehensive Report.**

7 Eat foods high in vitamin B12. Consider a supplement.

Your DNA analysis reveals that you may be at moderately higher risk of having low vitamin B12 levels. Vitamin B12 (also called cobalamin) is an important co-factor for several key biochemical reactions involving making DNA, RNA and clearing homocysteine. Low vitamin B12 also can result in high levels of homocysteine, which can increase risk of heart disease and stroke. Over time, low vitamin B12 levels increases risk of dementia. It is important to ensure you are meeting your dietary requirements for vitamin B12. Include foods high in vitamin B12, such as fish, seafood, meat, dairy and fortified foods, in your diet. Vitamin B12 is only found in animal products and fortified foods. If you are vegetarian or vegan, consider taking a vitamin B12 supplement. Check your intake using the HealthWatch 360 app.
See details in **Nutritional Genomics Report.**

Learn More

- **Symptoms of Vitamin B12 Deficiency**
- **How does vitamin B12 protect the brain from aging?**

Diet and Nutrition

- **Vitamin B12 Top Foods**
- **Even a healthy diet can lead to B12 deficiencies**

8 Restrict excessive carbohydrate intake, especially sugary foods.

Your DNA analysis reveals that you may be at moderately high risk for having high blood triglycerides (also known as hypertriglyceridemia). This is due to enhanced conversion of carbohydrates to triglycerides (a process called de novo lipogenesis). The liver produces triglycerides when carbohydrates are consumed in excess. These newly produced triglycerides are packaged into very low-density lipoproteins (VLDL) and secreted into the bloodstream. VLDL gets converted to LDL, which can lead to atherosclerosis or narrowing of blood vessels, a leading cause of heart disease. It is important to use portion control when consuming carbohydrates. Aim for no more than 50% of total calories from carbohydrates and 10% from added sugar. Limit consumption of soda, pastries and candy and foods high in refined carbs such as white bread, rice and pasta. Consider taking a vitamin B-complex and omega-3 fat supplement. Include aerobic exercise in your daily routine. Use the HealthWatch 360 app to monitor your carbohydrate intake and nutrition balance.
See details in **Nutritional Genomics Report.**

Diet and Nutrition

- **Diet and Nutrition for High Triglycerides**
- **Avoid excessive carbohydrate consumption**
- **Be conscious of consuming foods high in sugar**

9 Watch out for plant oils high in omega-6.

Your DNA analysis reveals that you have a converter genotype that increases risk of converting too much plant-based omega-6 fats from plant oils to the very long-chain, biologically omega-6 fat, arachidonic acid (AA). AA is pro-inflammatory; elevated levels are associated with increased risk of cardiovascular and inflammatory diseases. Limit consumption of plant oils high in omega-6 from foods such as peanuts, sunflower seeds, and corn oil. Instead, choose foods with plant oils lower in omega-6, such as olive and canola oils, flaxseeds, and walnuts. Use the HealthWatch 360 nutrition app to check your omega-6/omega-3 ratio. A ratio less than 6 is recommended.
See details in **Nutritional Genomics Report.**

Learn More

- **Video: How your genetics shapes your nutritional needs**
- **Are plant or animal fats better for you?**

Diet and Nutrition

- **Choose foods containing higher levels of omega-3**
- **Watch out for excessive omega-6 in plant-based oils**

10 Drinking coffee or tea may be beneficial to your health.

Caffeine is largely metabolized by the CYP1A2 enzyme in the liver. Due to genetic variation within this CYP1A2 gene, different enzymes are produced with differing properties: fast, slow and average metabolizers. Fast metabolizers process and inactivate caffeine four times faster than slow-metabolizers. Because you are a fast metabolizer of caffeine, you will clear caffeine from your body at a rapid rate. Fast metabolizers may develop tolerance to caffeine more quickly and are able to consume larger quantities of caffeine without experiencing the typical stimulatory effects that slow or average metabolizers may experience. Note: Smoking can affect CYP1A2 activity, which influences rate of caffeine metabolism.
See details in **Nutritional Genomics Report.**

Learn More

- **Genetic Trait: Caffeine Sensitivity**
- **Is coffee good or bad for your heart? Ask your genes.**

Diet and Nutrition

- **Sources of Caffeine**

11 You are likely intolerant to lactose. Limit dairy products or take Lactaid supplements.

Your DNA analysis reveals that you are likely lactose intolerant. You may experience gastrointestinal discomfort after consuming lactose-containing foods. You may benefit from limiting intake of dairy products such as milk, ice cream and cheese that contain lactose. You can also try lactose-free dairy products or dairy milk alternatives such as soy, rice or almond milk, that provide similar nutritional benefits. Track your dairy intake and health symptoms with the HealthWatch 360 app to find out if eating these foods aggravate your symptoms. Consider supplements that contain lactase enzymes, such as Lactaid or similar.
See details in **Nutritional Genomics Report.**

Learn More

- **Genetic Trait: Lactose Intolerance**

Diet and Nutrition

- **Limit foods high in lactose**

A 101-Year-Old in Excellent Health

CASE STUDY 2

Patient Information
Age: 101
Sex: Female

The following is a sample of our GB Longevity100 Summary Report

View the online report to access the built-in knowledge base and useful links: **bit.ly/longevity100-report**

Longevity Profile

Your Genetic Index for Longevity

EXCELLENT

Congratulations! You have many advantageous genetic variants for longevity. Check out your personalized Action Plan to see what you can do to further boost your health and enhance longevity.

Report Summary

- **13** advantage SNPs and **4** disadvantage SNPs identified in Longevity Associated Genes
- **13** action items are recommended
- Pathogenic/likely pathogenetic variants: **0 identified**

ⓘ This report is not intended to diagnose any condition and should only be used for educational or informational purposes.

ⓘ The GB-Longevity100 index provides a qualitative assessment of the likelihood of living longer based on genetic analysis of cardiometabolic diseases and dementia risk. This index does not encompass genetic analysis of cancer syndromes or other diseases that may significantly affect health and lifespan. It is important to note that lifespan and healthspan are not determined solely by genetics; factors such as diet, lifestyle, emotional well-being, risk-taking behaviors, and even luck also play critical roles in influencing the likelihood of living a longer and healthier life.
* This index calculates the cumulative effects of variants that increase or decrease longevity and presents it as a relative grade compared to your reference population.

Longevity Profile

Genetic Variants Associated with Longevity: 31 identified

Variants identified	SNP	Genotype	Effect	Impact to Longevity
MTTP (c.1981G>A(p.Gly661Ser)) *missense*	rs113337987	G/A	Advantage	Lower cholesterol, triglycerides, apoB
APOE (E2 (p.Arg176Cys)) *missense*	rs7412	C/T	Advantage	Lower Alzheimer's, heart disease risk
CYP17A1 (c.1621+39238G>A) *intron_variant*	rs12413409	G/A	Advantage	Reduced risk of heart disease
PCSK9 (g.55030366T>C) *intergenic_variant*	rs11206510	T/C	Advantage	Reduced risk of heart disease
SH2B3 (c.784T>C(p.Trp262Arg)) *missense*	rs3184504	T/C	Advantage	Reduced risk of heart disease
ANKK1 (c.2137G=(p.Glu713=)) *homozygous_reference_allele*	rs1800497	G/G	Advantage	Reduced risk of obesity
CELSR2 (c.*919G>T) *3_prime_UTR*	rs12740374	G/T	Advantage	Reduced risk of heart disease
FOXO3 (c.621+25486G>T) *intron_variant*	rs2802292	G/T	Advantage	Association with increased longevity
IL6 (7:g.22727026C=) *homozygous_reference_allele*	rs1800795	C/C	Advantage	Association with increased longevity
NOS3 (c.894T>G(p.Asp298Glu)) *missense*	rs1799983	G/G	Advantage	Lower blood pressure
PLCE1 (c.5780A>G(p.His1927Arg)) *missense*	rs2274223	A/G	Advantage	Lower blood pressure
ACE (c.2328G>A(p.Thr776=)) *synonymous_codon*	rs4343	G/A	Advantage	Reduced risk of hypertension
GPX1 (c.599C>T(p.Pro200Leu)) *missense*	rs1050450	G/A	Advantage	Lower BMI and blood pressure
HNF1A (c.1501+119G>T) *intron_variant*	rs2259816	G/T	Disadvantage	Increased risk of heart disease
MTHFR (c.665C>T(p.Ala222Val)) *missense*	rs1801133	A/A	Disadvantage	Increased risk of hypertension
PHACTR1 (c.251-149640A>G) *intron_variant*	rs9349379	G/G	Disadvantage	Increased risk of heart disease
CHRNA3 (c.645C>T(p.Tyr215=)) *synonymous_codon*	rs1051730	G/A	Disadvantage	Association with decreased lifespan
ABCG8 (c.55G=(p.Asp19=)) *homozygous_reference_allele*	rs11887534	G/G	Neutral	
APOA5 (c.56C=(p.Ser19=)) *homozygous_reference_allele*	rs3135506	G/G	Neutral	
APOA5 (c.*158C>T) *3_prime_UTR*	rs2266788	A/A	Neutral	
APOE (c.388T=(p.Cys130=)) *homozygous_reference_allele*	rs429358	T/T	Neutral	
BUD13 (c.358C=(p.Arg120=)) *homozygous_reference_allele*	rs10488698	G/G	Neutral	
CDKN2B-AS1 ((9p21):9:g.22124478A>G) *downstream_variant*	rs10757278	A/G	Neutral	
CETP (c.1264G>A(p.Val422Ile)) *missense*	rs5882	A/A	Neutral	
KL (c.1054T=(p.Phe=)) *homozygous_reference_allele*	rs9536314	T/T	Neutral	
LDLR (c.81C=(p.Cys27=)) *homozygous_reference_allele*	rs2228671	C/C	Neutral	
LDLR (c.67+2015G=) *homozygous_reference_allele*	rs6511720	G/G	Neutral	
LDLR (c.4521-5330G=) *homozygous_reference_allele*	rs1122608	G/G	Neutral	
PCSK9 (c.158C=(p.Ala=)) *homozygous_reference_allele*	rs11583680	C/C	Neutral	
PCSK9 (c.1420G>A(p.Val474Ile)) *missense*	rs562556	A/A	Neutral	
SORL1 (c.1582G=(p.Ala=)) *homozygous_reference_allele*	rs2298813	G/G	Neutral	

Health Insights

Pathogenic/likely pathogenetic variants

0 identified

Heart and Vascular Health (Lipid Disorders and ASCVD)

Focused Pathways:

Hypercholesterolemia	**Moderately high risk**
Hypertriglyceridemia	**Risk not identified**
High lipoprotein (a) levels	**Risk not identified**
Defective reverse cholesterol transport	**Risk not identified**
Sensitivity to sodium, hypertension	**Average risk**

See genetic details in Dyslipidemia and ASCVD Comprehensive Report

Metabolic Health (Diabetes, Obesity and NAFLD)

Focused Pathways:

Beta cell development and dysfunction	**Risk not identified**
Glucose sensing and insulin secretion	**Risk not identified**
Insulin resistance	**Risk not identified**
Susceptibility to autoimmune diseases	**Average risk**
Increased appetite due to low satiety, obesity	**Increased risk**

See genetic details in Type 2 Diabetes Comprehensive Report, Obesity Comprehensive Report

Cognitive Health (Alzheimer's)

APOE status: Reduced risk*

rs429358	rs7412	APOE Genotype
TT	CT	**E2/E3**

* Among Caucasian subjects, the risk of Alzheimer's disease (AD) increased significantly with increasing APOE-E4 dose. The odds ratios (OR) were 2.6, 3.2 and 14.9 for APOE genotypes E2/E4, E3/E4 and E4/E4, respectively, whereas the OR decreased for genotypes E2/E2 and E2/E3 (OR=0.6). The association between APOE-E4 and AD was weaker among African Americans and Hispanics and much stronger among Japanese populations (OR=5.6 for E3/E4, OR=33.1 for E4/E4).

While APOE is the single most important genetic risk factor for AD risk, it is not determinative. Other genetic and non-genetic factors can increase or decrease risk of AD. Certain rare genetic variants can increase risk even if one also carries the "protective" APOE-E2 allele. Likewise, carrying the AD risk-increasing APOE-E4 allele does not mean that development of AD is certain. Carrying certain protective genetic variants can prevent or delay onset of AD. Non-genetic factors, such as diet, lifestyle and engaging in brain-stimulating activities, also influence AD risk.

Nutritional Pathways Related to Cognitive Health:

Folate deficiency	**Increased risk**
Vitamin B12 deficiency	**Increased risk**
Iron overload	**Slightly increased risk**
Functional vitamin D deficiency	**Increased risk**

See genetic details in Alzheimer's Disease Comprehensive Report

Diet-Nutrition and Digestive System Health

Focused Pathways:

Choline deficiency	**Slightly increased risk**
Folate deficiency	**Increased risk**
Functional vitamin D deficiency	**Increased risk**
Vitamin B12 deficiency	**Increased risk**
Long-chain omega-3/6 deficiency	**Slightly increased risk**
Retinol deficiency	**Slightly increased risk**
Fat-soluble vitamin deficiency	**Average risk**
Iron overload	**Slightly increased risk**
Alcohol intolerance/Alcohol flush reaction	**Risk not identified**
Fructose intolerance	**Risk not identified**
Gluten intolerance	**Not likely at risk**
Lactose intolerance	**Likely intolerant**
Sensitivity to a high-carbohydrate diet, non-alcoholic fatty liver disease (NAFLD)	**Risk not identified**
Sensitivity to a high-carbohydrate diet, hypertriglyceridemia	**Slightly increased risk**
Sensitivity to a high-fat diet, hypercholesterolemia	**Slightly increased risk**
Sensitivity to a ketogenic diet, high absorption of cholesterol and plant sterols	**Risk not identified**
Sensitivity to high cholesterol and high carbohydrate intake, type III hyperlipidemia	**Risk not identified**
Sensitivity to sodium, hypertension	**Average risk**
Reduced utilization of long-chain fatty acids for fuel	**Risk not identified**
Sensitivity to caffeine	**Average**

See genetic details in Nutritional Genomics Report

Action Plan

Take charge of your health and longevity
Science-based recommendations to help you maximize healthspan and enhance longevity.

1 Empower yourself: take steps to live longer and healthier.

Genetics can inform disease risk and impact both healthspan and lifespan. However, many genetic risk factors are modifiable. Early preventive measures and treatments can delay or even eliminate age-related diseases, extending your disease-free years and potentially increasing life expectancy. Maintaining a healthy lifestyle-through diet, exercise, a positive outlook, social engagement, and, when necessary, supplements or medications-can significantly improve both healthspan and lifespan. To enhance longevity, you need to consistently practice healthy habits and adhere to treatments that manage lipid levels, blood pressure, and blood sugar. Start early and stay committed throughout your life. Discuss your genetic test results with your healthcare provider to identify and treat any underlying conditions, setting the foundation for maximizing your health and longevity.

Diet and Nutrition

- Dietary Guidelines for Americans
- 30-min Exercises Everyone Can Do

2 Control total fat intake and limit saturated fat and cholesterol.

3 Eat foods high in vitamin B12. Consider a supplement.

4 Reduce overeating: avoid high-caloric foods. Eat a calorie-restricted diet: higher protein and fiber.

5 Reduce risk of vitamin D deficiency. Consider a D3 supplement. Eat foods high in vitamin D.

6 Restrict excessive carbohydrate intake, especially sugary foods.

7 Be conscious of red meat in your diet.

8 Eat foods high in folate to ensure you don't develop a deficiency.

9 **Eat foods high in long-chain omega-3 and omega-6 fatty acids.**

10 **Eat foods high in choline.**

11 **Eat foods high in retinol.**

12 **Moderate amounts of coffee or tea may be beneficial to your health.**

13 **You are likely intolerant to lactose. Limit dairy products or take Lactaid supplements.**

Frequently Asked Questions

1. How is the Genetic Index for Longevity calculated?

The GB Genetic Index for Longevity calculates the cumulative effects of 30-40 genetic variants (depends on the number of unique genetic variants identified in your genomic analysis) that have been shown to influence health span and lifespan in genetic studies. This includes well-known and common variants associated with longevity, such as APOE2/APOE4, as well as variants in the FOXO3, KL, ACE, NOS3, CETP, PCSK9, and LDLR genes, etc. Rare genetic variants, which have strong evidence suggesting their effect on longevity, are also included. The Longevity index is given as one of four relative categories: Excellent, Good, Average and At-Risk. These categories are calculated as compared to your reference population (e.g. European, African, East Asian and Hispanic/Latino). It is important to remind you that this Longevity Suite does not analyze genes related to familial cancer syndromes and, therefore, these risk categories do not assess the genetic risk of cancer. For more, please see Question 8.

2. What can I learn from my longevity genetic profile?

Genetic variants associated with longer lifespan are often linked to lower blood pressure, lower blood lipids (especially cholesterol levels), lower Lp(a), lower risk of Alzheimer's disease and a lower likelihood of obesity. Conversely, genetic variants strongly associated with reduced lifespan are often linked to very high cholesterol levels, high Lp(a), and higher blood pressure-factors associated with a higher risk of cardiovascular diseases. Additionally, genetic variants associated with Alzheimer's disease influence average longevity. From the genetic variants identified in your longevity profile, you can learn which variants in your DNA are associated with increased or decreased longevity, what the biological function of these variants are, and how to mitigate the effects of genetic risk.

3. My genetic index for longevity is Average. What does 'average' mean? What can I do to boost my longevity?

Your GB Longevity100 index of "Average" indicates your genetic profile for longevity is similar to the majority in your reference population. This is because there are no detected large-effect size variants that may increase or decrease longevity. However, this average score cannot guarantee that you do not carry a large-effect sized variant affecting your longevity. It is possible that you may carry genetic variants that are not covered by the GB Longevity100 test (e.g., cancer risk), or a variant is identified but its effects are not defined. This latter scenario is common, as most rare genetic variants identified in people are classified as variants of uncertain significance or VUS (sometimes referred to as VOUS). The estimated effect of genetics on longevity is only about 20%. Taking action that mimics the genetics of those who possess an advantageous genetic longevity index, which is associated with lower blood lipid levels, lower blood pressure, and lower risks of cardiovascular and metabolic diseases, can help you achieve a healthier and longer life, even with an average genetic index for longevity.

4. My genetic index for longevity is At-Risk. How much does that truly affect my lifespan?

A low longevity index score doesn't necessarily indicate a shorter lifespan. It simply highlights the presence of certain genetic risk factors that can negatively impact health and lifespan, such as those associated with familial hypercholesterolemia (FH), high Lp(a), and/or Alzheimer's disease. A low genetic index for longevity could also suggest a lack of advantageous genetic variants compared to the average. Understanding your genetic makeup empowers you to recognize these risk factors early and take preventive measures. Consult with your healthcare providers to confirm and manage these genetic risk factors. Your Action Plan will provide guidance on how to mitigate these factors and maximize your health span and longevity.

5. My genetic index for longevity is Excellent. Does that mean I have a better chance of reaching a longer lifespan?

Yes and no. First, an "Excellent" score on the GB Longevity100 index suggests a genetic profile with more advantageous variants and fewer disadvantageous ones relative to your reference population. Your genetic profile likely provides greater protection against cardiovascular diseases and cognitive decline, suggesting a higher likelihood of a healthier and longer life. However, it's important to note that the genetic variants used by GB Longevity100 for calculating the longevity index do not include cancer risk genes. If you have a family history of cancer, you absolutely need to manage your cancer risk. Consult with your physician about additional testing for family cancer risk and follow guidelines for routine screening. It's important to reiterate here that the GB Longevity100 suite does not analyze the entire genome, and some identified genetic variants are unable to be classified due to a lack of information on whether they impact the gene. Work with your healthcare provider to manage potential risk factors, even those not detected by genetic testing.

6. I know one of my parents or grandparents lived to a very old age. Why is my longevity index low?

In general, the genetics of exceptionally long lifespans are largely unknown. Some highly advantageous genetic variants are rare and specific to certain families, which makes it difficult to extrapolate to larger populations. Due to the lack of scientific studies on these private variants, GB Longevity100 might not include them in the longevity index calculation, even if you possess one in your genome. Another explanation for a low index score in offspring with long-lived parents is that the long-lived individual(s) might have inherited the most advantageous genetic variants, and these may not have been passed to other offspring. For example, your parents may have the advantageous APOE2, and other genetic variants linked to lower lipid levels and lower risk of Alzheimer's disease and at the same time, lack genetic variants that increase risk of cardiovascular diseases. However, their offspring may not be as fortunate, inheriting a less favorable combination of genetic variants from their parents. Having a low longevity score does not necessarily mean a shorter lifespan. It can indicate that certain genetic risk factors negatively affecting health and lifespan have been identified. Consulting with your healthcare provider to manage these identified genetic risk factors is essential for achieving optimal health and a longer life.

7. Despite my family history of early death, how is it that my longevity index is good?

Firstly, a favorable genetic score for longevity suggests that more advantageous genetic variants and fewer disadvantageous ones have been identified in your genome. This indicates an increased likelihood of living a healthier and longer life. It's important to note that the GB Longevity100 suite of genetic tests does not cover cancer risk genes, and not all genes in the genome are analyzed. Some genetic variants have unknown functions, so their potential negative effects are not factored into the index score calculation. Additionally, you may be fortunate to have inherited advantageous genes from your parents without inheriting the disadvantaged ones. Furthermore, it's crucial to recognize that health and lifespan are not solely determined by genetics; poor lifestyle choices and inadequate health management can lead to poorer health and a shorter lifespan.

8. What about my risk of cancer?

The GB-Longevity100 Suite does not currently screen for familial cancer syndromes, such as Familial Breast-Ovarian Cancer caused by pathogenic variants within the BRCA1 or BRCA2 genes. However, there are many other genetic testing companies that screen for germline genetic variants found in all of our cells, identifying familial cancer susceptibility syndromes. GB Longevity100 focuses on cardiometabolic diseases that are modifiable through lifestyle changes and, in some cases, medication.

Proactive Genetic Screening Suite

LONGEVITY INSIGHTS

Longevity Index.
Genes associated with longevity.
Specific targets.

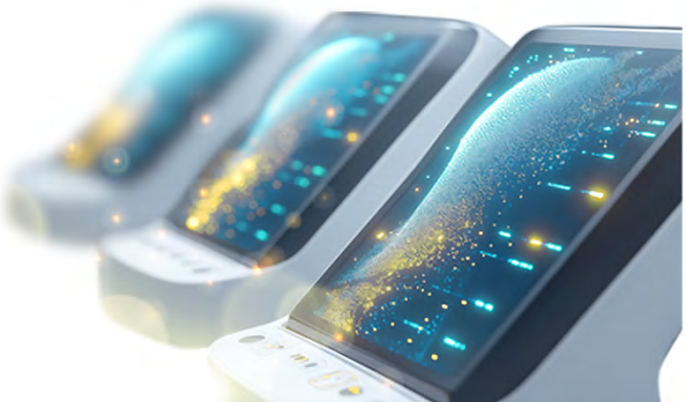
ACTION PLAN

Tailored to your DNA.
Built-in precision medicine.
Diet and Nutrition app.

HEALTH INSIGHTS

Identify risk factors for prevention:

- **Cardiovascular**
- **Metabolic**
- **Cognitive**
- **Nutrition**



NEXT-GEN TECHNOLOGY

Analyzing Six Comprehensive Genetic Panels

Longevity Genetic Index PANEL	Lipid Disorders and Heart Health COMPREHENSIVE PANEL	Type 2 Diabetes COMPREHENSIVE PANEL
Obesity COMPREHENSIVE PANEL	Alzheimer's Disease COMPREHENSIVE PANEL	Nutritional Genomics PANEL

The GB Longevity100 genetic testing and analysis suite offers comprehensive, proactive genetic screening designed to help individuals achieve optimal health and potentially live to 100. It analyzes key genetic markers associated with human lifespan, calculates a genetic index for longevity, and provides thorough screening to identify modifiable genetic risk factors linked to cardiovascular, metabolic, and cognitive health. Additionally, it offers personalized action plans tailored to each individual's genetic profile to mitigate these risks.

It’s Easy to Order

ORDER TEST

GB genetic tests are easy to order online. Contact us to setup your provider account now:
order@gbhealthwatch.com or (858) 788-9274

DNA KIT

DNA sample collection kits are free. You can request to have kits sent to your clinic or directly to your patients.

COLLECT DNA SAMPLE

We use a buccal swab for DNA sample collection.
A gentle swab of the inside of the cheek is all that is required to collect a DNA sample.

MAIL TO LAB

Mail the DNA sample to our laboratory using USPS regular mail. The DNA sample is preserved at room temperature.

LAB ANALYSIS

Next-generation DNA sequencing is performed at a CLIA-certified and licensed medical genetic testing laboratory. Bioinformatics analysis is performed by GBinsight's analysis suite.

REPORT RELEASED TO DOCTOR

Genetic reports are released to the provider's account in about two weeks. The provider can customize the report, print as a PDF, and share the report digitally with colleagues and patients. An in-depth knowledgebase is built into each report to help with interpretation of the results and their applications for clinical utility.

GB HealthWatch

6310 Greenwich Drive, Suite 100
San Diego, CA 92122

GBinsight Test Catalog

bit.ly/gbinsight-catalog

- GB6010 - GB Longevity100 Genetic Testing and Analysis Suite
- GB2030 - Dyslipidemia and ASCVD Comprehensive Panel
- GB2031 - Familial Hypercholesterolemia Panel
- GB2032 - Familial Hypertriglyceridemia Panel
- GB2010 - Type 2 Diabetes Comprehensive Panel
- GB2011 - Diabetes MODY Panel
- GB2020 - Obesity Comprehensive Panel
- GB2050 - Alzheimer's Disease Comprehensive Panel
- GB2051 - Familial Early-Onset Alzheimer Disease Panel
- GB5010 - Nutritional Genomics Comprehensive Panel

How to order

It is very easy to place orders for GBinsight genetic testing. First, set up your account online to register your DNA samples. You will use this account to access your genetic test results.

bit.ly/gbinsight-signup

Once an account is set up, you can place an order online, by email or by phone:

www.gbhealthwatch.com/gbinsight
customercare@gbhealthwatch.com
(858) 788-9274

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