SelfDecode™

Mitochondria & Energy Production Pathway

Sample Client

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Personal information

NAME

Sample Client

SEX AT BIRTH

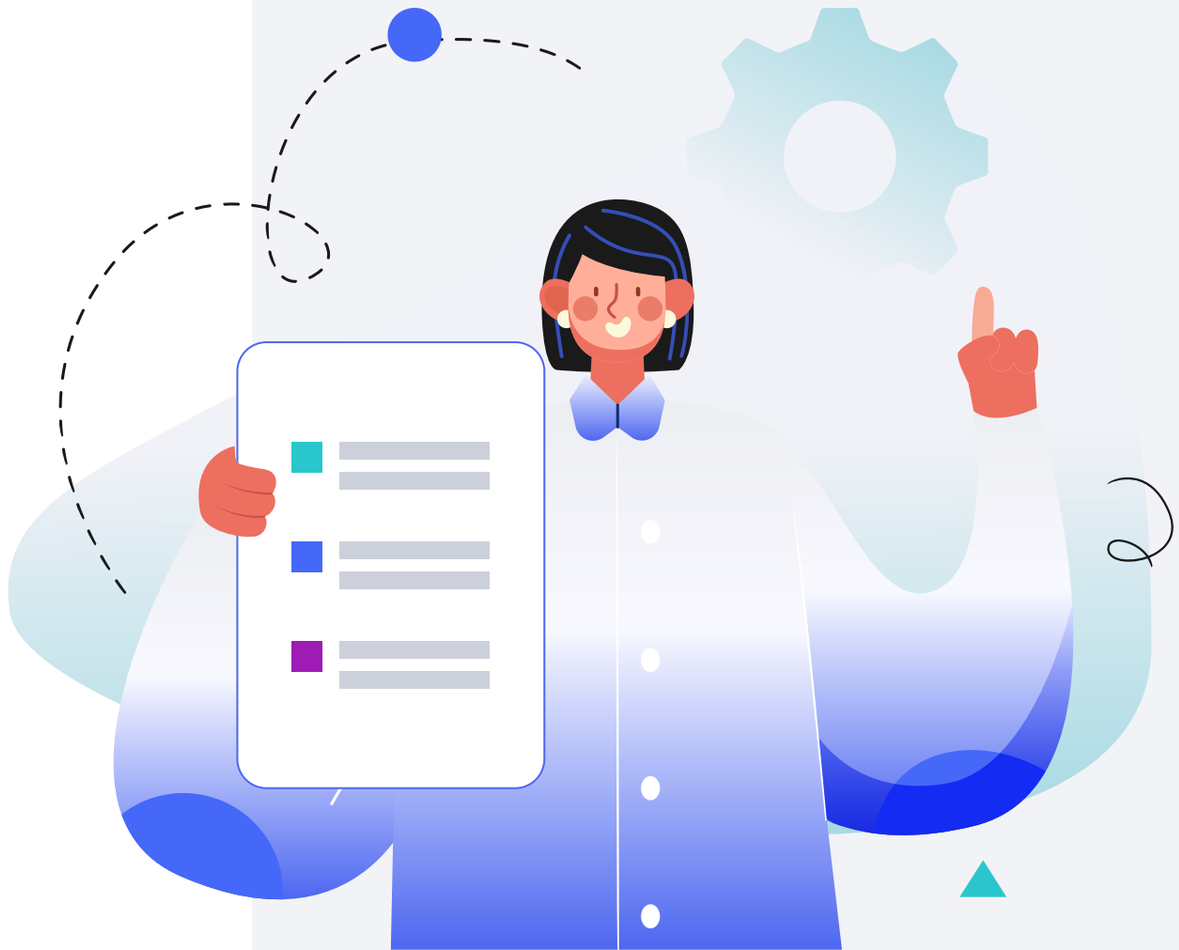
Male

HEIGHT

5ft 7" 170cm

WEIGHT

143lb 65kg



DISCLAIMER

This report does not diagnose this or any other health conditions. Please talk to a healthcare professional if this condition runs in your family, you think you might have this condition, or you have any concerns about your results.

How this works

Mitochondria are the primary energy-producing structures in nearly every cell of the body, converting the food you eat into the ATP that powers muscle contraction, brain activity, metabolism, and countless other biological processes. Because mitochondrial function touches so many systems at once, how efficiently your body produces, senses, renews, and protects its mitochondria can influence everything from athletic performance and blood sugar control to body weight, aging, and long-term disease risk.

The pathway begins with fuel. Fatty acids, glucose, and lactate from food are shuttled into the mitochondria by transporters and enzymes such as **CPT1A, SLC16A1 (MCT1), and ACADM**, which convert these fuels into acetyl-CoA. Acetyl-CoA then enters the Krebs cycle, generating the electron carriers **NADH and FADH2** that feed directly into ATP production.

These electron carriers power the electron transport chain, where genes like **NDUFB6 (Complex I) and TFAM** support the mitochondrial machinery, and **CoQ10**—produced and regenerated with help from **NQO1 and COQ2**—shuttles electrons onward through Complex III to Cytochrome C, ultimately generating ATP.

Mitochondrial activity is also closely monitored and adjusted based on the body's energy status. **NAMPT drives production of NAD+**, a molecule essential for energy metabolism, while **AMPK** (encoded by PRKAA1 and PRKAA2) senses the cell's **AMP:ATP ratio** and activates the **sirtuins SIRT1, SIRT3, and SIRT6**—proteins closely tied to metabolic health and longevity.

When the body needs more mitochondria, it relies on biogenesis: **PPARGC1A (PGC-1α)** acts as the master regulator, working alongside **PPARA, NRF1, and NFE2L2 (NRF2)** to build new mitochondria and support antioxidant defenses. At the same time, damaged mitochondria are identified and either repaired or broken down through mitochondrial recycling, a process guided by **MFN2, FOXO3, and PINK1**.

Not all mitochondrial energy is converted into ATP—some is deliberately released as heat through **uncoupling proteins UCP1, UCP2, and UCP3**, a process tied to thermogenesis and weight regulation. Meanwhile, the byproducts of energy production, including reactive oxygen species like superoxide and hydrogen peroxide, are neutralized by antioxidant enzymes such as **SOD2, GPX1, and GPX4**, protecting cells from oxidative damage.

This report analyzes your genetic variants across each stage of the mitochondrial pathway—from fuel metabolism and ATP production to energy sensing, mitochondrial renewal, thermogenesis, and antioxidant defense—so you can better understand your unique mitochondrial tendencies and take a more personalized approach to supporting energy production, metabolic health, and long-term cellular resilience.

How to Interpret Your Mitochondrial Pathway Report

The two visual diagrams on the following pages are the core of the report. They show the step-by-step biochemical flow of mitochondrial function and energy production, with each gene (and its associated SNPs) placed at the exact step where it acts. Reading these diagrams helps you understand not just how individual genes behave, but how they interact as a system.

Gene result labels tell you whether your variant is associated with:

- **Typical activity (blue)** — your gene functions within the normal expected range
- **Altered activity (orange or red)** — your variant differs from the most common form; this may mean higher or lower enzyme activity depending on the specific gene
- **Better activity (green)** — your variant is associated with a more favorable functional outcome for that step

Directional arrows on genes indicate whether a variant tends to push that step up (upregulating) or down (downregulating) relative to typical activity.

Biological compounds shown in the diagrams (e.g., glucose, lactate, CoQ10, nicotinamide) represent crucial lab markers and metabolites produced at each stage. Where lab result slots appear alongside these compounds, you can enter your own bloodwork values to see how your measured levels align with what your genetics might predict.

If a lab result is available for a specific compound, the badge color next to its name will indicate whether the result falls within the optimal range.

The **legend** in the bottom left corner explains the meaning of each element on the pathway, so please read it carefully.

Understanding the Pathway as a System

Because these genes operate as an interconnected network, your results should be read holistically rather than gene by gene. A few principles that help with interpretation:

- **Fuel transport sets the ceiling for everything downstream.** CPT1A, SLC16A1 (MCT1), and ACADM control how efficiently fatty acids, lactate, and fatty acid fragments enter the mitochondria and get converted into acetyl-CoA. If less fuel gets in — or gets in more slowly — there is less raw material for the Krebs cycle and electron transport chain to work with, regardless of how well the rest of the pathway performs.
- **NAMPT and AMPK act as the cell's energy gauge, and they set the tone for the whole pathway.** NAMPT drives NAD+ production, while AMPK (encoded by PRKAA1 and PRKAA2) senses the AMP:ATP ratio and responds to energy stress. Together, they activate the sirtuins (SIRT1, SIRT3, SIRT6) and PGC-1α. Variants here don't just affect one process — they influence how strongly the cell responds to exercise, fasting, and other energy demands across every stage that follows.
- **PPARGC1A (PGC-1α) is the master switch for building new mitochondria.** It doesn't act alone — it works together with PPARA, NRF1, and NFE2L2 (NRF2) to drive mitochondrial biogenesis and antioxidant gene expression. A variant that reduces PGC-1α activity can blunt the benefits of exercise and calorie restriction even if the genes it partners with are functioning normally, since PGC-1α is the one initiating the process.
- **CoQ10 sits at the intersection of energy production and antioxidant defense.** NQO1 and COQ2 control how much CoQ10 is produced and regenerated. Because CoQ10 is required both to shuttle electrons through the electron transport chain and to neutralize free radicals, variants that reduce CoQ10 availability can show up as two seemingly unrelated issues at once — lower ATP output and weaker antioxidant capacity.
- **Antioxidant defense is a two-step process, and both steps need to keep pace with each other.** SOD2 converts superoxide into hydrogen peroxide, but hydrogen peroxide is still reactive — GPX1 and GPX4 are needed to break it down further into harmless byproducts. High SOD2 activity paired with low GPX1/GPX4 activity can actually increase oxidative stress rather than reduce it, since the intermediate byproduct builds up faster than it can be cleared. This is the most important imbalance to look for across the antioxidant defense genes.
- **Uncoupling proteins trade energy efficiency for heat, and that trade-off has metabolic consequences either way.** UCP1, UCP2, and UCP3 divert energy away from ATP production and release it as heat instead. More uncoupling activity tends to favor fat burning and thermogenesis but can reduce ATP efficiency, while less uncoupling activity favors ATP output but may come with reduced fat-burning capacity. Neither direction is uniformly "better" — it depends on which outcome matters more for a given person.
- **Mitochondrial recycling determines whether damage accumulates or gets cleared.** MFN2, FOXO3, and PINK1 identify damaged mitochondria and either repair them through fusion or break them down through mitophagy. Because this process runs continuously in the background, variants that slow it down don't cause immediate symptoms — but they allow dysfunctional mitochondria to accumulate over time, which can compound the effects of variants elsewhere in the pathway.

Other Result Details

The **Results Overview** provides a structured table of every gene and SNP analyzed in the report, grouped by sub-pathway. Use this section to quickly locate your genotype for any specific gene. Each gene appears with its relevant SNP identifiers and your genotype. Alongside the genetic results, suggested lab markers are listed for each sub-pathway — these are the blood, urine, or saliva tests most relevant to validating or complementing your genetic findings.

The detailed **Gene–SNP Breakdown** section explains each gene and variant in depth. For every gene you will find:

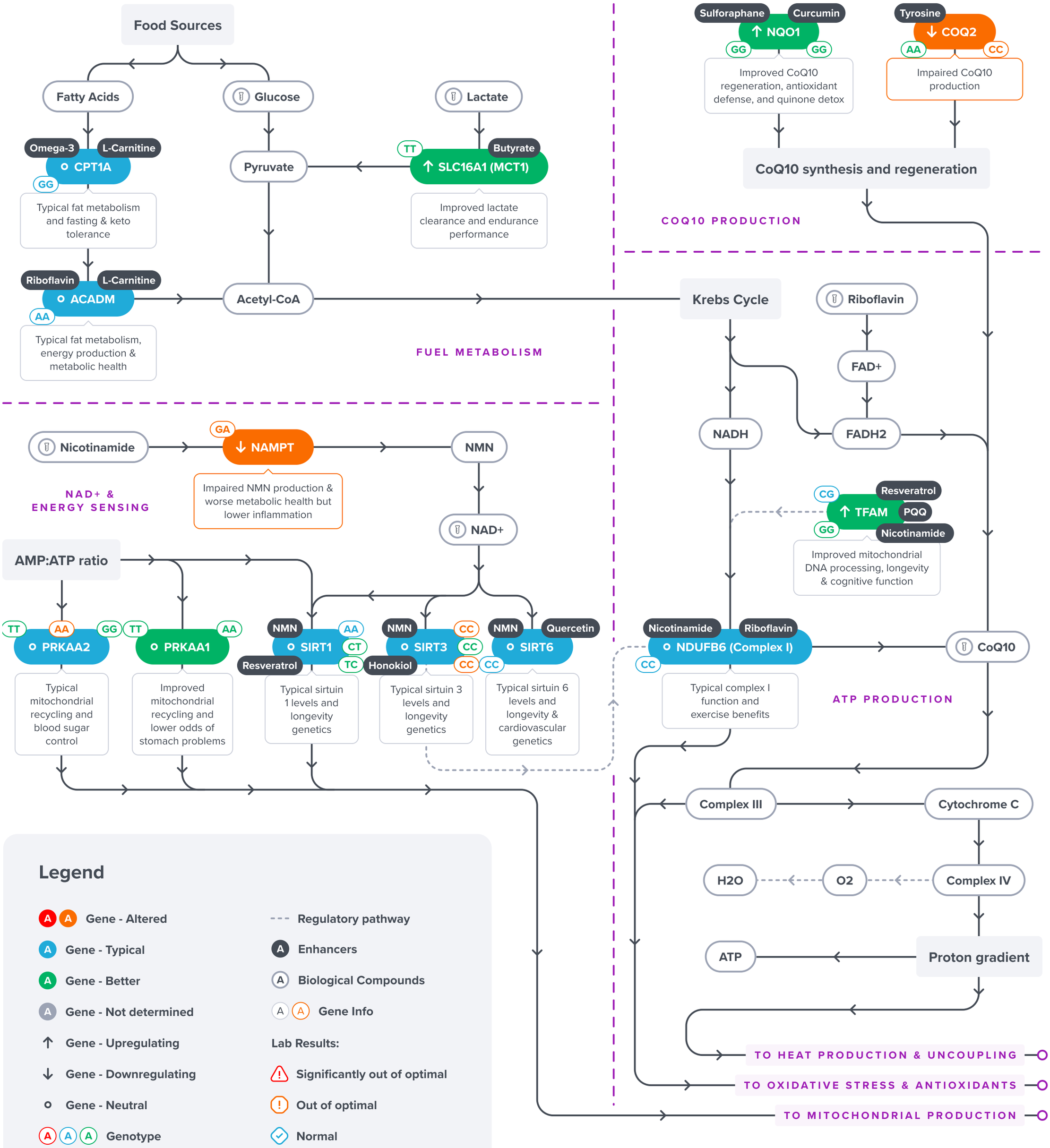
- **What the gene does** — its biological role in fuel metabolism, mitochondrial function, antioxidant defense, etc.
- **What your specific genotype means** — whether your variant is associated with higher, lower, or typical activity at that step, and what health outcomes have been linked to it in research
- **Enhancers** — nutrients or compounds that support the function of that enzyme, listed where relevant

The **Recommendations** section lists personalized dietary, lifestyle, and supplement strategies, ranked by likely impact based on your specific gene variants. Each recommendation includes the genes it is relevant to and explains the biological mechanism behind it.

The recommendations are intended as a starting point for discussion with a healthcare provider, not as a prescriptive protocol. Some suggestions (such as limiting alcohol or eating fiber-rich foods) are broadly beneficial. Others (such as specific supplements at particular doses) are more targeted and should be considered in the context of your full health picture, current medications, and lab results.

Lab Markers to Check list the laboratory tests most relevant to your genetic profile, organized by the gene(s) that make them informative for you. Running these tests allows you to see whether the functional tendencies suggested by your genetics are actually showing up in your measured physiology. Genetics describes predisposition; labs confirm whether that predisposition is being expressed under your current diet, lifestyle, and environment.

Energy Production Pathway



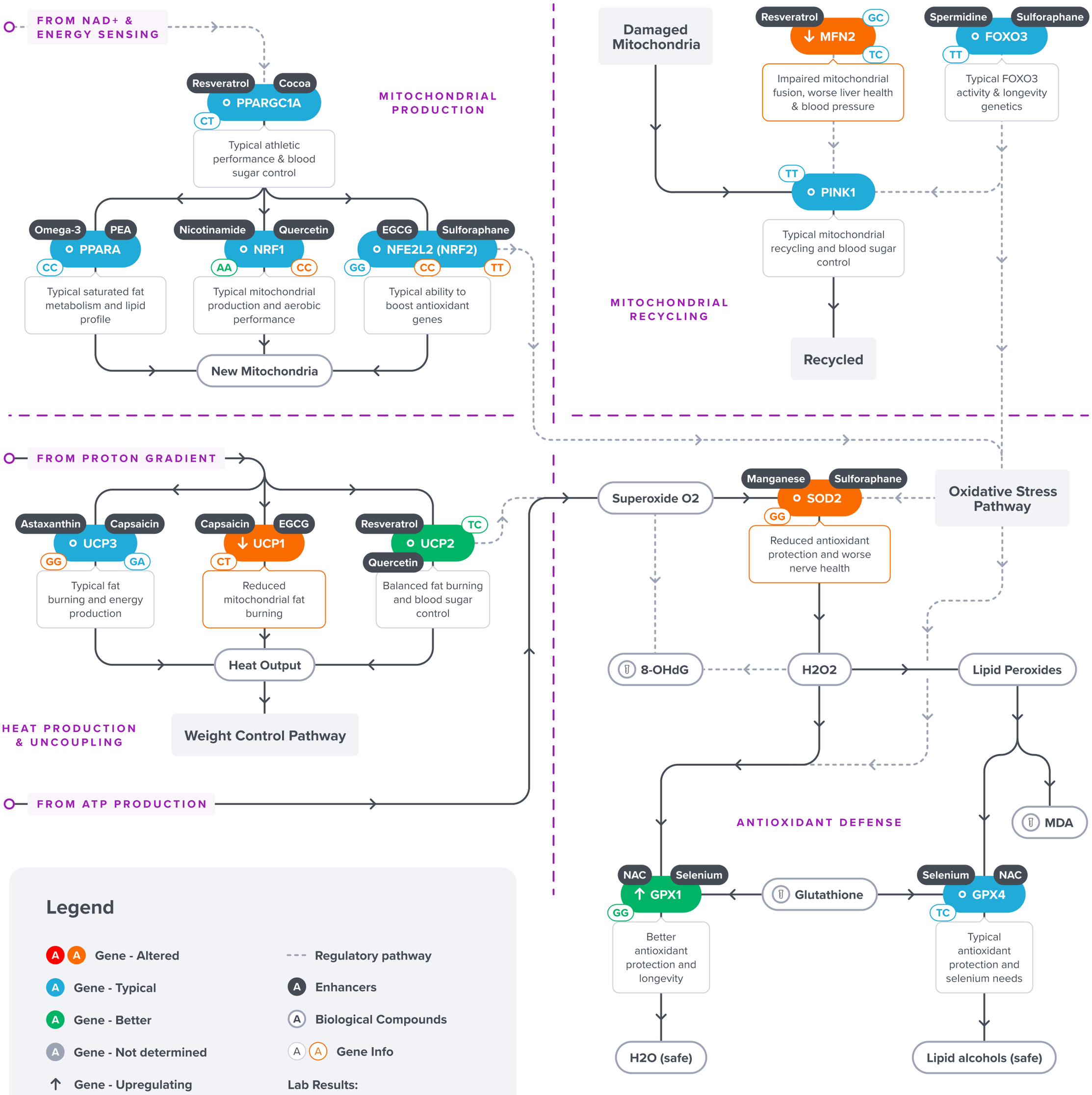
Legend

- Gene - Altered
- Gene - Typical
- Gene - Better
- Gene - Not determined
- Gene - Upregulating
- Gene - Downregulating
- Gene - Neutral
- Genotype
- Genotype not determined
- Main pathway
- Regulatory pathway
- Enhancers
- Biological Compounds
- Gene Info

Lab Results:

- Significantly out of optimal
- Out of optimal
- Normal
- Optimal
- Missing lab result

Renewal & Defense Pathway



Legend

- Gene - Altered
- Gene - Typical
- Gene - Better
- Gene - Not determined
- Gene - Upregulating
- Gene - Downregulating
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- Genotype
- Genotype not determined
- Main pathway
- Regulatory pathway
- Enhancers
- Biological Compounds
- Gene Info

Lab Results:

- Significantly out of optimal
- Out of optimal
- Normal
- Optimal
- Missing lab result

Results Overview

Fuel Metabolism

Gene - SNP Summary

ACADM	rs77931234	o AA
CPT1A	rs80356779	o GG
SLC16A1	rs1049434	↑ TT

CoQ10 Production

Gene - SNP Summary

COQ2	rs148156462	↑ AA
	rs4693075	↓ CC
NQO1	rs1800566	↑ GG
	rs1131341	↑ GG

Labs Summary

- Creatine Kinase
- Coenzyme Q10
- L-Lactate (Genova)
- Pyruvic Acid

NAD+ & Energy Sensing

Gene - SNP Summary

NAMPT	rs61330082	↓ GA
	rs11555236	↓ CC
SIRT3	rs4980329	↓ CC
	rs11246020	↑ CC
PRKAA2	rs2746342	o TT
	rs10789038	o GG
	rs2796498	o AA
SIRT6	rs107251	o CC
SIRT1	rs7895833	o AA
	rs12778366	↑ TC
	rs3758391	↑ CT
PRKAA1	rs13361707	o TT
	rs10074991	o AA

Labs Summary

- Insulin, Fasting
- 8-Hydroxy-2-deoxyguanosine, Urine
- C-Reactive Protein (CRP)
- Carnitine, Total
- F2-Isoprostanes
- HOMA-IR
- Intracellular NAD (icNAD)
- L-Lactate (Genova)
- Pyruvic Acid

ATP Production

Gene - SNP Summary

NDUFB6	rs540467	o CC
TFAM	rs1937	o CG
	rs2306604	↑ GG

Mitochondrial Production

Gene - SNP Summary

NFE2L2	rs6721961	o GG
	rs35652124	↓ TT
	rs1806649	↓ CC
NRF1	rs6949152	↑ AA
	rs2402970	↓ CC
PPARA	rs1800206	o CC
PPARGC1A	rs8192678	o CT

Labs Summary

GGT · VO2 Max · 8-Hydroxy-2-deoxyguanosine, Urine · F2-Isoprostanes · L-Lactate (Genova) · Pyruvic Acid · Total Glutathione

Heat Production & Uncoupling

Gene - SNP Summary

UCP1	rs1800592	↓ CT
UCP3	rs1800849	↓ GG
	rs647126	o GA
UCP2	rs659366	o TC

Labs Summary

TSH · VO2 Max · T3, Free (FT3) · T4 Free (FT4) · Carnitine, Total · Free Fatty Acids · L-Lactate (Genova)

Mitochondrial Recycling

Gene - SNP Summary

MFN2	rs873457	o GC
	rs1474868	o TC
FOXO3	rs12212067	o TT
PINK1	rs622525	o TT

Antioxidant Defense

Gene - SNP Summary

SOD2	rs4880	GG
GPX4	rs713041	TC
GPX1	rs1050450	↑ GG

Labs Summary

- 8-Hydroxy-2-deoxyguanosine, Urine
- C-Reactive Protein (CRP)
- F2-Isoprostanes
- Manganese, Blood

Your recommendations

Your recommendations are prioritized according to the likelihood of it having an impact for you based on your lab results, along with the amount of scientific evidence supporting the recommendation.

You'll likely find common healthy recommendations at the top of the list because they are often the most impactful and most researched.

		DOSAGE
1	Aerobic Exercise (Cardio)	1 hour
2	Coenzyme Q10 (CoQ10)	100 mg
3	Intermittent Fasting	
4	Nicotinamide Riboside (NR)	250 mg
5	B Vitamins	
6	Capsaicin	
7	Dietary Polyphenols	
8	Manganese supplements	1 mg
9	Pyrroloquinoline Quinone (PQQ)	20 mg
10	Sulforaphane	30 mg
11	Apigenin	50 mg
12	Astaxanthin	12 mg
13	Choose Healthy Fats	
14	Cold Exposure	1 minutesute
15	Green Tea Extract	250 mg
16	Hot and Cold Applications	15 minutes
17	Iodine	150 mcg
18	L-Carnitine	1 g
19	L-Tyrosine	500 mg
20	Magnolia Bark	
21	Maintain Optimal Iron Levels	10 mg
22	Mitoquinone Mesylate (MitoQ)	
23	N-acetylcysteine (NAC)	1200 mg
24	Niacin Supplements	25 mg
25	Selenium Supplements	50 mcg
26	Ubiquinol	100 mg
27	Zinc	15 mg

Aerobic Exercise (Cardio)

How to implement

Engage in at least 150 minutes of moderate-intensity aerobic exercise or 75 minutes of vigorous-intensity activity each week. Distribute this time over at least 3 days per week, avoiding consecutive days of vigorous exercise to allow for recovery.

TYPICAL STARTING DOSE

1 hour

How it helps

Aerobic/cardio exercise is one of the strongest known stimuli for mitochondrial biogenesis, robustly increasing mitochondrial density, oxidative enzyme activity, and overall aerobic capacity in skeletal muscle.

Personalized to Your Genes

↓ NAMPT	Exercise is one of the few stimuli that upregulates NAMPT itself, partially compensating for the genetic deficit.
SOD2	Regular moderate training hormetically upregulates SOD2 expression, strengthening mitochondrial antioxidant defense over time.
NFE2L2	Exercise transiently activates NRF2; consistent training keeps the antioxidant response primed despite reduced baseline capacity.
NRF1	Endurance exercise drives PGC-1a, the upstream activator of NRF1, providing the strongest natural stimulus for the biogenesis this variant impairs.
SIRT3	Endurance exercise upregulates SIRT3 in skeletal muscle, boosting mitochondrial fat oxidation and antioxidant capacity.

Coenzyme Q10 (CoQ10)

How to implement

Take a 100 mg Coenzyme Q10 (CoQ10) supplement once daily with a meal that contains fat for better absorption.

TYPICAL STARTING DOSE

100 mg

How it helps

A core electron carrier in the electron transport chain (Complex I to Complex III), essential for ATP synthesis. CoQ10 also acts as a lipid-soluble antioxidant protecting mitochondrial membranes, making it one of the most direct mitochondrial-support compounds available.

Personalized to Your Genes

↓ COQ2	200-300mg/day of ubiquinol with fat directly replaces the CoQ10 that impaired COQ2 can't produce, restoring electron transport and antioxidant function.
○ SOD2	100-200mg/day ubiquinol helps clear the mitochondrial superoxide burden that reduced SOD2 leaves unaddressed.
↓ UCP1	100-200mg/day ubiquinol supports the intact electron transport chain UCP1 relies on for brown-fat thermogenesis.
○ NRF1	CoQ10 ensures the electron transport chain components NRF1 builds can carry electrons efficiently.
○ UCP3	100-200mg/day of ubiquinol supports electron transport efficiency, offsetting the reduced energy production associated with low UCP3.

Intermittent Fasting

How to implement

Limit your daily eating to a specific window of time, typically within an 8-hour period such as from 12 pm to 8 pm, and fast for the remaining 16 hours of the day. Repeat this daily or for at least 3-4 days per week.

How it helps

Triggers mitophagy and mitochondrial biogenesis through AMPK activation and reduced insulin signaling during fasting windows, helping clear damaged mitochondria and promote metabolic flexibility. Well-supported in both animal and emerging human studies.

Personalized to Your Genes

↓ NAMPT	12-16h fasting windows raise the NAD+/NADH ratio and stimulate NAMPT expression, supporting NAD+ availability through the impaired pathway.
↓ UCP1	Fasting activates norepinephrine signaling and AMPK, both of which upregulate UCP1 in brown and beige fat.
○ SIRT3	Energy restriction is the most established natural upregulator of SIRT3 expression, directly targeting the deficit.

Nicotinamide Riboside (NR)

How to implement

Take 250-300 mg of Nicotinamide Riboside (NR) supplement daily, with or without food. It can be taken any time of the day, but maintaining a consistent routine, such as taking it every morning, is advised for best results. Continue this regimen daily for an ongoing basis to support cellular health and energy levels.

TYPICAL STARTING DOSE

250 mg

How it helps

A direct precursor to NAD+, a critical coenzyme for electron transport chain function and sirtuin-mediated mitochondrial biogenesis. NR supplementation reliably raises NAD+ levels, supporting mitochondrial energy metabolism, especially as levels decline with age.

Personalized to Your Genes

↓ NAMPT	NMN or NR (250-500mg/day) bypass the rate-limiting NAMPT step entirely, restoring NAD+ without depending on the impaired enzyme.
○ NRF1	NMN or NR (250-500mg/day) activate the SIRT1-PGC-1a axis that upregulates NRF1.
○ SIRT3	Sirtuins are NAD+-dependent, so NMN or NR (250-500mg/day) maximize the activity of the limited SIRT3 present.

B Vitamins

How to implement

Take a B vitamin complex supplement once daily, preferably with your morning meal to enhance absorption. The supplement should include a range of B vitamins such as B1 (thiamine), B2 (riboflavin), B3 (niacin), B5 (pantothenic acid), B6 (pyridoxine), B7 (biotin), B9 (folate), and B12 (cobalamin). Ensure consistency by taking it at the same time each day.

How it helps

B vitamins (B1, B2, B3, B5, B6, B12) are essential cofactors for the Krebs cycle and electron transport chain enzymes. Deficiencies directly impair ATP synthesis, making adequate B-vitamin status foundational for mitochondrial energy metabolism.

Personalized to Your Genes

↓ COQ2	B-vitamins are cofactors around the CoQ10 synthesis pathway; optimizing them supports the residual production impaired COQ2 still carries out.
NRF1	B-vitamins are cofactors for the respiratory chain enzymes NRF1 regulates, ensuring newly built mitochondria function well.

Capsaicin

How to implement

Capsaicin is available in capsule form, ideally enteric-coated, which means it dissolves in the intestine rather than the stomach, reducing the chance of irritation. Start with a low dose and increase gradually as your body adjusts. Always take it with food to minimize side effects like nausea or heartburn. If you have a sensitive stomach, acid reflux, or any digestive condition, check with your doctor before starting.

How it helps

Activates TRPV1 receptors and can stimulate mitochondrial uncoupling and thermogenesis in adipose tissue, potentially improving mitochondrial efficiency and reducing oxidative byproducts. Effects on skeletal muscle mitochondria are less established.

Personalized to Your Genes

↓ UCP1	2-6mg/day with meals activates TRPV1 receptors that stimulate sympathetic UCP1 activation and brown fat thermogenesis.

UCP3

Capsaicin stimulates UCP3 expression in skeletal muscle via sympathetic/PPAR signaling, directly targeting the downregulated gene.

Dietary Polyphenols

How to implement

Incorporate foods rich in dietary polyphenols into your daily diet. This can include consuming a variety of fruits like berries, apples, and grapes; vegetables such as onions and broccoli; nuts and seeds; as well as beverages like green tea and coffee. Aim for at least five servings of fruits and vegetables per day to achieve a beneficial intake of polyphenols.

How it helps

A broad class of plant compounds that activate Nrf2 and sirtuin pathways, promoting mitochondrial biogenesis and antioxidant defense. Polyphenol-rich diets are consistently linked to improved mitochondrial function markers across various tissues.

Personalized to Your Genes

SOD2

Polyphenol- and vitamin-rich produce provide complementary antioxidant support to protect nerves and mitochondria from oxidative damage.

NFE2L2

Green tea/EGCG, curcumin, and resveratrol activate NRF2 through complementary mechanisms, layering antioxidant gene induction.

Manganese supplements

How to implement

Take 1-5 mg of manganese supplement daily, preferably with a meal to enhance absorption.

TYPICAL STARTING DOSE

1 mg

How it helps

A cofactor for manganese superoxide dismutase (MnSOD), the primary antioxidant enzyme inside mitochondria that neutralizes superoxide radicals generated during oxidative phosphorylation. Adequate manganese status is essential for this protective mechanism.

Personalized to Your Genes

SOD2	Manganese is the essential catalytic cofactor for SOD2; optimizing it (whole grains, nuts, leafy greens, without excess) maximizes the reduced enzyme's activity.
SIRT3	SIRT3 activates SOD2, the key mitochondrial antioxidant; supplying its manganese cofactor supports this downstream defense.

Pyrroloquinoline Quinone (PQQ)

How to implement

Take 20 to 40 mg of Pyrroloquinoline Quinone (PQQ) daily as an oral supplement, ideally with meals to enhance absorption. This dosage can be maintained indefinitely as part of your daily supplement routine.

TYPICAL STARTING DOSE

20 mg

How it helps

Stimulates mitochondrial biogenesis through PGC-1alpha activation and has strong antioxidant properties that protect mitochondrial membranes and DNA from oxidative damage. Preclinical evidence is robust, human data still emerging.

Personalized to Your Genes

↓ COQ2	10-20mg/day complements CoQ10 in the mitochondrial redox system and stimulates biogenesis to support overall bioenergetics.
NRF1	10-20mg/day is one of the few compounds shown to directly stimulate NRF1-mediated mitochondrial biogenesis.

Sulforaphane

How to implement

Take a sulforaphane supplement, typically available in capsule form, with a dosage ranging from 30 to 60 milligrams per day. It is generally taken once daily, with or without food, according to the product's label instructions or a healthcare provider's advice. Continue this regimen daily for as long as you seek its benefits, but consult a healthcare provider for long-term use guidance.

TYPICAL STARTING DOSE

30 mg

How it helps

A potent Nrf2 activator from cruciferous vegetables, upregulating a broad suite of endogenous antioxidant and detoxification enzymes that protect mitochondria from oxidative and xenobiotic stress.

Personalized to Your Genes

SOD2	Broccoli sprouts or extract activate NRF2, which upregulates SOD2 and the broader antioxidant network, compensating for low baseline SOD2.
NFE2L2	Broccoli sprouts or extract (~10-30mg/day) are the most potent known natural NRF2 activator, directly stimulating the underperforming pathway.

Apigenin

How to implement

Take 50-100 mg of apigenin supplement daily, preferably with a meal to enhance absorption. This dosage is suggested based on studies for general health benefits and should be continued as long as it contributes positively to your well-being, or as directed by a healthcare professional.

TYPICAL STARTING DOSE

50 mg

How it helps

A flavonoid shown to inhibit CD38, an NAD+-consuming enzyme, thereby preserving cellular NAD+ pools available for mitochondrial redox reactions and sirtuin activity. This supports oxidative phosphorylation efficiency and may counter age-related NAD+ decline in preclinical models.

Personalized to Your Genes

↓ NAMPT

50-100mg apigenin inhibits CD38, a major NAD+ consumer, preserving the limited NAD+ produced through impaired NAMPT.

Astaxanthin

How to implement

Take an astaxanthin supplement daily, with a typical dosage ranging from 4 to 12 mg. It is best taken with a fat-containing meal to enhance absorption.

TYPICAL STARTING DOSE

12 mg

How it helps

A carotenoid antioxidant that embeds in mitochondrial membranes, protecting lipids from peroxidation and stabilizing membrane potential. It has been shown to support mitochondrial biogenesis and reduce exercise-induced oxidative damage in muscle tissue.

Personalized to Your Genes

UCP3

Astaxanthin is a potent mitochondrial antioxidant that supports the mitochondrial efficiency and fatty-acid oxidation that UCP3 facilitates.

Choose Healthy Fats

How to implement

Incorporate sources of unsaturated fats such as olive oil, avocados, nuts, seeds, and fatty fish into your daily diet. Aim for at least two servings of fatty fish per week and use olive oil for cooking and salad dressings. Replace saturated fats found in red meat, butter, and processed foods with these healthier options whenever possible.

How it helps

Replacing saturated fats with monounsaturated and polyunsaturated fats supports healthier mitochondrial membrane composition, improving fluidity and electron transport chain function while reducing lipotoxic stress associated with saturated fat overload.

Personalized to Your Genes

UCP3

Fatty acid availability upregulates UCP3 expression, targeting the gene while supplying the fuel it helps handle; use quality fats, cycled.

Cold Exposure

How to implement

Gradually expose yourself to cold temperatures for short periods, starting with 1-2 minutes and increasing up to 5-10 minutes. This can be achieved through cold showers, outdoor exposure in cold weather, or ice baths. Aim to do this 2-3 times per week.

TYPICAL STARTING DOSE

1 minutesute

How it helps

Activates brown and beige adipose tissue, increasing mitochondrial density and UCP1-mediated thermogenesis. Repeated cold exposure promotes mitochondrial biogenesis and improves metabolic flexibility through hormetic stress adaptation.

Personalized to Your Genes

↓ UCP1

Cold showers or immersion 4-5x/week are UCP1's most potent activator, upregulating its expression and inducing browning of white fat over time.

Green Tea Extract

How to implement

Take a green tea extract supplement containing 250-500 mg of EGCG (the active compound in green tea) daily, preferably with a meal to enhance absorption. This dosage is typically split into two separate doses, taken in the morning and later in the day. Continue this regimen for at least three months to observe potential health benefits.

TYPICAL STARTING DOSE

250 mg

How it helps

Rich in EGCG, a catechin that activates AMPK and PGC-1alpha, promoting mitochondrial biogenesis and fatty acid oxidation. Green tea extract has also been shown to reduce mitochondrial oxidative stress in metabolic tissues.

Personalized to Your Genes

↓ UCP1

EGCG 400mg with caffeine 100-200mg synergistically activate the sympathetic pathway driving UCP1 expression and thermogenesis.

Hot and Cold Applications

How to implement

Alternate applying a warm compress for 15-20 minutes, then a cold pack for 15-20 minutes to the affected area. Repeat this cycle 3-4 times per day for up to one week or until symptoms improve.

TYPICAL STARTING DOSE

15 minutes

How it helps

Alternating heat and cold exposure induces hormetic stress that activates heat shock proteins and mitochondrial biogenesis pathways. This combined stimulus may enhance mitochondrial resilience more than either modality alone, though direct comparative evidence is limited.

Personalized to Your Genes

NRF1

Cyclic sauna and cold immersion activate PGC-1a-NRF1 signaling, supporting mitochondrial production without exercise.

Iodine

How to implement

Take a 150 mcg iodine supplement daily, preferably with a meal to enhance absorption.

TYPICAL STARTING DOSE

150 mcg

How it helps

Essential for thyroid hormone synthesis, and thyroid hormones directly regulate mitochondrial biogenesis and metabolic rate. Iodine deficiency impairs this regulatory axis, indirectly reducing mitochondrial oxidative capacity.

Personalized to Your Genes

UCP3

Iodine is the raw material for thyroid hormone synthesis; adequacy ensures T3 drive to UCP3 isn't substrate-limited.

L-Carnitine

How to implement

Take 500 mg of L-carnitine supplement daily with a glass of water, preferably with a meal to enhance absorption.

TYPICAL STARTING DOSE

1 g

How it helps

Shuttles long-chain fatty acids into the mitochondrial matrix for beta-oxidation, a rate-limiting step in fat-based ATP production. Carnitine supplementation is well-established for supporting mitochondrial fatty acid metabolism, especially in deficient individuals.

Personalized to Your Genes

UCP3

1-2g/day shuttles fatty acids into mitochondria for oxidation, compensating for reduced UCP3-mediated fat handling in muscle.

L-Tyrosine

How to implement

Take 500-2000 mg of L-Tyrosine supplement daily, preferably 30 minutes before meals or snacks. This can be taken in one dose or divided into two doses throughout the day. Continue this supplementation daily for a duration of at least a few weeks to assess its effects on your body.

TYPICAL STARTING DOSE

500 mg

How it helps

A precursor to catecholamines and thyroid hormone, both of which regulate mitochondrial biogenesis and metabolic rate. Adequate tyrosine status supports this neuroendocrine-mitochondrial signaling axis, though direct mitochondrial mechanisms are indirect.

Personalized to Your Genes

↓ COQ2

Tyrosine is a precursor for CoQ10 biosynthesis; adequate intake removes a substrate limitation on the residual COQ2-dependent production.

Magnolia Bark

How to implement

Take a magnolia bark supplement containing 200-400mg of magnolol and honokiol, the active compounds, once or twice daily. It is best taken with food to improve absorption. Usually, magnolia bark supplements are taken for several weeks to months to experience the full benefits.

How it helps

Contains honokiol, a compound shown in preclinical studies to activate AMPK and promote mitochondrial biogenesis and autophagy (mitophagy), helping clear damaged mitochondria. Human clinical evidence remains limited.

Personalized to Your Genes

SIRT3

Honokiol is a direct SIRT3 activator that can raise SIRT3 activity, partially compensating for genetically lower SIRT3 levels.

Maintain Optimal Iron Levels

How to implement

Check your iron levels. If they are below optimal, take a 10 mg iron supplement daily, preferably with a meal to enhance absorption and reduce stomach upset.

TYPICAL STARTING DOSE

10 mg

How it helps

A required cofactor for iron-sulfur clusters and heme groups in electron transport chain complexes I, II, and III. Both deficiency and excess iron can impair mitochondrial function, making adequate (not excessive) status important.

Personalized to Your Genes

↓ UCP1

Adequate iron supports the electron transport chain that UCP1 thermogenesis depends on, maximizing residual UCP1 output.

Mitoquinone Mesylate (MitoQ)

How to implement

Take MitoQ supplement by swallowing one capsule each morning with water, preferably on an empty stomach to aid in its absorption. This should be done daily for an extended period, often several months, to notice a significant benefit in conditions related to mitochondrial dysfunction or to support overall mitochondrial health.

How it helps

A mitochondria-targeted form of CoQ10 designed to accumulate specifically within mitochondria at higher concentrations than standard CoQ10. It has shown promising results in reducing mitochondrial oxidative stress and improving vascular function in

early trials.

Personalized to Your Genes

SOD2

MitoQ is a mitochondria-targeted antioxidant that neutralizes superoxide directly within mitochondria, compensating for reduced SOD2 activity.

N-acetylcysteine (NAC)

How to implement

Take 600 mg of N-Acetylcysteine (NAC) supplement daily with water. It can be taken at any time of the day, but try to take it at the same time each day for best results.

TYPICAL STARTING DOSE

1200 mg

How it helps

A precursor to glutathione, the primary intracellular antioxidant that protects mitochondrial proteins, lipids, and DNA from oxidative damage. NAC supplementation reliably raises glutathione levels, supporting mitochondrial redox homeostasis under oxidative stress.

Personalized to Your Genes

NFE2L2

600-1200mg/day NAC or liposomal glutathione supplies precursors, compensating for the weakened NRF2-driven glutathione response.

Niacin Supplements

How to implement

Take a 25 mg niacin supplement daily, preferably with a meal to aid in absorption and minimize potential stomach upset.

TYPICAL STARTING DOSE

25 mg

How it helps

Converts to NAD+ and NADP+, essential coenzymes for nearly every step of mitochondrial energy metabolism including the Krebs cycle and electron transport chain. Niacin adequacy is foundational for mitochondrial ATP production.

Personalized to Your Genes

↓ NAMPT

Adequate dietary niacin and tryptophan feed the de novo and Preiss-Handler NAD+ pathways, offering routes that don't depend on NAMPT.

Selenium Supplements

How to implement

Take 50 mcg of selenium supplements once daily, preferably with a meal to enhance absorption.

TYPICAL STARTING DOSE

50 mcg

How it helps

A cofactor for glutathione peroxidase, a key antioxidant enzyme protecting mitochondrial membranes from lipid peroxidation. Selenium also supports thyroid hormone conversion, indirectly influencing mitochondrial metabolic rate.

Personalized to Your Genes

UCP3

Selenium is required to produce active thyroid hormone (T3), a primary transcriptional activator of UCP3.

Ubiquinol

How to implement

Take 100 to 200 mg of ubiquinol daily, preferably with a fatty meal to enhance absorption. This dosage can be adjusted based on individual health needs and under the guidance of a healthcare professional. Continue this

TYPICAL STARTING DOSE

supplementation daily for ongoing support of heart health and energy levels.

100 mg

How it helps

The reduced, more bioavailable form of CoQ10, directly participating in electron transport chain reactions and offering superior antioxidant protection to mitochondrial membranes, particularly beneficial for individuals with reduced conversion capacity.

Personalized to Your Genes

↓ COQ2

Taking CoQ10 with dietary fat and in divided doses maximizes uptake, which matters more when endogenous production is impaired.

Zinc

How to implement

Take a 15 mg zinc supplement daily, ideally with a meal to enhance absorption.

TYPICAL STARTING DOSE

15 mg

How it helps

A cofactor for numerous mitochondrial antioxidant enzymes, including copper-zinc superoxide dismutase (SOD1), which protects against oxidative damage. Zinc is also involved in mitochondrial membrane stability.

Personalized to Your Genes

UCP3

Zinc supports thyroid hormone conversion and signaling, helping maintain the T3 drive that upregulates UCP3.

COQ2

COQ2 Report

The *COQ2* gene encodes an enzyme called polyprenyl transferase that carries out the last step in the production of coenzyme Q10, a redox carrier in mitochondrial respiration [R, R].

Coenzyme Q10 plays a key role in oxidative phosphorylation, which converts the energy from food into a form that cells can use. Coenzyme Q10 also acts as an antioxidant that protects cell membranes from free radicals [R].

SNP

rs148156462

Alleles

A: Increased COQ2 activity

G: Reduced COQ2 activity

Your Genotype

↑AA

Your genotype is linked to increased COQ2 activity and improved CoQ10 production

Intro and Health Effects

The main *COQ2* polymorphism is rs148156462 (V393A). Its minor 'G' allele reduces gene expression and thus coenzyme Q10 production. This allele has been associated with an increased risk of:

- Multiple system atrophy (cerebellar type) [R, R, R]
- Parkinson's disease [R]

SNP

rs4693075

Alleles

G: Increased COQ2 activity

C: Reduced COQ2 activity

Your Genotype

↓CC

Your genotype is linked to reduced COQ2 activity and impaired CoQ10 production

Intro and Health Effects

The major 'C' allele of another variant, rs4693075, has been associated with statin-induced muscle damage. However, not all studies found this effect [R, R, R, R, R].

MFN2

MFN2 Report

The *MFN2* gene encodes a protein called mitofusin 2 that helps determine the shape and structure of the mitochondria. Specifically, mitofusin 2 controls the fusion (combining pieces) of these organelles. By doing so, it regulates the proliferation of vascular smooth muscle cells [R, R].

Mitofusin 2 is found in many types of cells and tissues, including muscles, the spinal cord, and the nerves that connect the brain and spinal cord to muscles. Within the cell, it's found in the outer membrane of the mitochondria [R].

Enhancers:

Resveratrol

SNP	Your Genotype
rs873457 Alleles **G:** Increased MFN2 activity **C:** Reduced MFN2 activity	GC **Your genotype is linked to typical MFN2 activity, typical mitochondrial fusion, liver health & blood pressure**

Intro and Health Effects

In addition, the 'C' allele of **rs873457**, the 'C' allele of **rs2336384**, and the 'T' allele of **rs4846085** have been associated with:

- Increased risk of high blood pressure, especially in men [R, R, R]
- Increased risk and worse prognosis of acute liver failure [R]
- Increased risk of fatty liver [R]

These three variants are usually inherited together, meaning you will either have all or none of them.

SNP	Your Genotype
rs1474868 Alleles **C:** Increased MFN2 activity **T:** Reduced MFN2 activity	TC **Your genotype is linked to typical MFN2 activity, typical mitochondrial fusion, liver health & blood pressure**

Intro and Health Effects

One of the main *MFN2* variants is **rs1474868**. Its minor ‘T’ allele has been associated with reduced gene expression and lower mitochondrial DNA levels, as well as with [R, R, R]:

- Increased risk of high blood pressure, especially in men [R, R]
- Increased risk and worse prognosis of acute liver failure [R]

NAMPT

NAMPT Report

The *NAMPT* gene encodes an enzyme called nicotinamide phosphoribosyltransferase that catalyzes a rate-limiting step of NAD^+ regeneration: the condensation of nicotinamide with 5-phosphoribosyl-1-pyrophosphate to yield nicotinamide mononucleotide (NMN) [R].

The NAMPT protein exists in two forms [R, R]:

- **Intracellular** (iNAMPT): found inside the cell, it's the key enzyme in NAD+ regeneration.
- **Extracellular** (eNAMPT or visfatin): secreted by cells, it acts as an adipokine and inflammatory cytokine.

NAMPT has been investigated as a drug target. While activators may act as anti-aging drugs, inhibitors are used in cancer research to selectively starve cancer cells of NAD+ [R, R, R].

SNP	Your Genotype
rs61330082 Alleles **A:** Reduced NAMPT activity **G:** Increased NAMPT activity	↓GA **Your genotype is linked to reduced NAMPT activity, impaired NMN production & worse metabolic health but lower inflammation**

Intro and Health Effects

AMPT has a "good" and a "bad" side: inside cells it is the key enzyme the body uses to produce NAD+, a molecule central to energy metabolism and mitochondrial function, while a secreted form (also called visfatin) acts as a pro-inflammatory signal.

One of the main *NAMPT* variants is **rs61330082**. Its minor 'A' allele may decrease the expression of this gene. By reducing NAD+-related metabolic support, it's been linked to [R, R]:

- Greater hypertension in type 2 diabetes patients and obese children [R, R]
- Higher blood triglycerides and VLDL-cholesterol in obese children and adults with metabolic syndrome [R, R]
- Higher cardiovascular risk in hepatitis C patients [R]
- Increased risk of prostate cancer recurrence and metastasis [R]

At the same time, by lowering the pro-inflammatory form of the protein and cardiovascular inflammation, it's linked with a **reduced risk** of [R]:

- Cardiovascular events in patients with coronary artery calcification [R]
- Bladder cancer [R]
- Esophageal squamous cell carcinoma [R]

- Non-small cell lung cancer [R]
- Liver cancer in hepatitis B patients [R]

These associations reflect a trade-off: lower NAMPT tends to be less favorable for metabolic and blood-pressure health but more favorable for inflammation and tumor-related risk. Most of these findings come from specific patient groups, so how much any of it applies depends heavily on a person's overall health context.

SOD2

SOD2 Report

Superoxide dismutases are enzymes that transform the superoxide (O2-) radical into either ordinary oxygen (O2) or hydrogen peroxide (H2O2).

Superoxide is produced as a by-product of oxygen metabolism. If left unattended, it can cause widespread cell damage. Thus, SOD is an important part of antioxidant defense in all cells exposed to oxygen [R].

SOD2 (also called MnSOD) is one of the superoxide dismutase enzymes, alongside SOD1 and SOD3. SOD2 is unique in that it requires manganese (Mn) to work, whereas the other two need copper and zinc.

SOD2 transforms the superoxide produced by the mitochondria into the less toxic hydrogen peroxide and oxygen. This allows SOD2 to clear mitochondrial reactive oxygen species (ROS) and confer some protection against cell death [R, R].

Enhancers:

Sulforaphane

SNP

rs4880

Alleles

A: Higher SOD2 levels but lower mitochondrial activity

G: Lower SOD2 levels but higher mitochondrial activity

Your Genotype

GG

Your genotype is linked to worse antioxidant nerve protection but lower odds of diabetes and cardiovascular problems

Intro and Health Effects

The SOD2 gene has many described polymorphisms. Among them, rs4880 has got most of the spotlight in SOD2 research. Its minor **allele 'G'** is associated with **decreased SOD2 levels**. However, some cell research suggests this variant is linked to **higher SOD2 mitochondrial activity** [R].

Owing to its decreased SOD2 levels, this variant has been associated with diseases such as [R, R, R, R]:

- Breast, prostate, and colorectal cancer
- Hypertension
- Sporadic motor neuron disease
- Alzheimer's disease
- Parkinson's disease
- Noise-induced hearing loss

- Cisplatin-induced ear toxicity
- Infertility
- Phthalate-induced lung damage

In contrast, the **major 'A' variant** is more common among people with [R, R, R]:

- Diabetes and its complications
- Worse cardiovascular health (with some mixed findings)

The association of this variant with longevity isn't straightforward either. While a study found the 'G' variant was more common among very elderly Danish people, the 'A' variant was prevalent among very elderly Ashkenazi Jewish men in another study [R, R].

UCP1

UCP1 Report

The *UCP1* gene encodes a protein called *uncoupling protein 1* and mainly found in the mitochondria of brown and beige fat cells. It's also called *thermogenin* because it helps generate heat by a process called *non-shivering thermogenesis*, which helps our bodies stay warm in cold environments [R, R, R, R, R].

Because it prevents excess energy from being stored as body fat (and instead turns it into heat), many researchers believe that UCP1 could protect against weight gain and obesity [R, R, R].

Enhancers:

EGCG (green tea)

SNP	Your Genotype
rs1800592	↓CT
Alleles **C:** Reduced UCP1 activity **T:** Increased UCP1 activity	**Your genotype is linked to reduced UCP1 activity and reduced mitochondrial fat burning**

Intro and Health Effects

One of the best-studied SNPs in the UCP1 gene is rs1800592. It helps determine how your body uses and stores the energy that you get from food [R].

The 'T' allele is linked to increased activity of the UCP1 gene. It's associated with a higher resting metabolic rate, higher body heat production, and less weight gain. According to some researchers, this variant helps turn more of the energy from food into heat instead of body fat (white fat) [R, R].

According to several studies, the **'C' allele** (and especially the 'CC' genotype) is associated with [R, R, R, R, R, R, R]:

- Increased weight gain and a higher chance of being obese
- Elevated blood pressure and insulin resistance
- Higher LDL cholesterol and triglycerides

In one study, people with the 'CC' genotype had less brown fat at a younger age than people with the ‘T’ allele [R].

Many researchers think that the UCP1 gene and the rs1800592 SNP are in part responsible for human adaptation to colder climates [R].

Although today we consider the 'T' allele beneficial in terms of its potential effect on body weight, this allele is essentially linked to lower metabolic efficiency. In other words, people with this allele may “waste” more of the energy that they get from food on generating body heat. It is plausible that the more efficient 'C' allele may be advantageous when food is scarce and the climate is warm [R].

ACADM

ACADM Report

The ACADM gene encodes an enzyme called medium-chain acyl-CoA dehydrogenase (MCAD) involved in fatty acid beta-oxidation. This process takes place in the mitochondria and is used to break down fats and produce energy [R, R].

MCAD is the first enzyme to act in the beta-oxidation of medium-chain fatty acids (roughly 6-12 carbons in length), making it a key gateway into mitochondrial fat-burning. When MCAD activity is insufficient, the body loses a major metabolic escape valve during fasting or metabolic stress [R].

Enhancers:

Riboflavin

SNP	Your Genotype
rs77931234	AA
Alleles **A:** Typical ACADM activity **C:** Reduced ACADM activity	**Your genotype is linked to typical ACADM activity, typical fat metabolism, energy production & metabolic health**

Intro and Health Effects

By far, the best-researched ACADM variant is rs77931234, also known as Lys329Glu or K329E. Its **minor 'C' allele greatly reduces MCAD activity**. While subjects heterozygous for this allele have MCAD activity levels that are 6-14% of those of non-carriers, the activity of those with two copies of the allele is below 2.5%. Heterozygous carriers are usually asymptomatic [R, R].

Carrying at least one copy of this variant has been associated with **mild MCAD deficiency**. While often asymptomatic, this variant has been associated with the following symptoms, especially if combined with other mutations in this gene:

- Elevated blood C8-acylcarnitine [R, R, R]
- Liver damage [R, R]
- Low blood sugar [R]
- Heart arrhythmia [R]

CPT1A

CPT1A Report

The CPT1A gene encodes a liver enzyme called carnitine palmitoyltransferase I (CPTI). Together with carnitine palmitoyltransferase II (CPT II), this enzyme is essential for long-chain fatty acid oxidation, a process that breaks them down to produce energy and ketones, especially during periods of fasting, illness, or low-carbohydrate diets [R, R, R].

CPT I is located on the outer membrane of the mitochondria, where it binds carnitine to long-chain fatty acid molecules so they can be transported across mitochondrial membranes. Once inside the mitochondria, carnitine is removed from these fatty acids so they can be broken down [R, R].

Certain variants in this gene can cause CPT I deficiency, a condition that prevents the body from using long-chain fatty acids for energy. This results in low blood sugar (hypoglycemia) and ketone (hypoketosis) levels. Moreover, the non-transported fatty acids build up in cells and damage the liver, heart, and brain [R].

Enhancers:

Omega-3

SNP	Your Genotype
rs80356779	GG
Alleles	
A: Reduced CPT1A activity	
G: Typical CPT1A activity	

Your genotype is linked to typical CPT1A activity, typical fat metabolism, and fasting & keto tolerance

Intro and Health Effects

By far, the best-researched CPT1A variant is rs80356779, also known as P479L. Its minor 'A' allele is most common in **Aboriginal populations** across coastal British Columbia, Alaska, the Canadian North, Greenland, and Siberia, suggesting a role in **adaptation to cold climates**. It decreases enzyme activity by 74% and has been linked to a mild **CPT I deficiency** [R, R, R, R].

This variant has been associated with:

- **Impaired fasting tolerance** [R]
- Reduced height [R]
- Increased risk of lung cancer [R]

On the bright side, this variant has been associated with elevated plasma HDL and apoA-I levels, as well as decreased body fat, suggesting it helps protect against atherosclerosis and obesity [R, R].

Interestingly, carriers of this variant may **require higher dietary amounts of fish and seafood to achieve their optimal omega-3 levels**. While the original populations where this allele appeared ate a diet that was already rich in these foods, the current westernization of the diet in these regions may cause health issues in carriers [R].

FOXO3

FOXO3 Report

The FOXO3 gene encodes a transcription factor, a protein that binds to DNA and regulates the activity of other genes. For instance, FOXO3 regulates insulin signalling and protein metabolism in muscles [R].

FOXO3 is involved in tumor suppression, immune function, DNA repair, and resistance to oxidative stress, which may explain its link with increased longevity [R, R, R].

FOXO3 also plays a role in managing oxidative stress. Mice without FOXO3 are highly susceptible to reactive oxygen species in their bones, resulting in decreased bone mass and increased risk of fracture (osteoporosis). The antioxidant effect of FOXO3 has not yet been investigated in aged mice [R, R].

Enhancers:

Sulforaphane

SNP	Your Genotype
rs12212067	TT
Alleles **G:** Increased FOXO3 activity **T:** Typical FOXO3 activity	**Your genotype is linked to typical FOXO3 activity & longevity genetics**

Intro and Health Effects

In the last decade or so, a large body of research has linked certain variants in the FOXO3 gene with longer lifespan. They include [R, R, R, R, R, R, R]:

- ‘G’ at rs12212067

Interestingly, the effects of some of these variants may be strongest in people who spend more time in nature [R].

The ‘G’ allele of rs12212067 has also been associated with a milder course of Crohn’s disease [R, R].

GPX4

GPX4 Report

In the intricate world of cellular metabolism, the GPX4 gene stands out as a critical guardian against oxidative stress. This gene encodes glutathione peroxidase 4 (GPX4), an enzyme pivotal in protecting cells from damage caused by reactive oxygen species (ROS). It is deeply intertwined with selenium and glutathione metabolism, making it a cornerstone of cellular health.

GPX4 supports the reduction of lipid hydroperoxides and hydrogen peroxide, converting them into harmless water and alcohol. What makes GPX4 unique among antioxidant enzymes is its reliance on selenium. Selenium is incorporated into GPX4 as the amino acid selenocysteine, critical for the enzyme's catalytic activity.

Without sufficient selenium, GPX4's function is compromised, leaving cells vulnerable to oxidative damage. GPX4's activity is also tightly linked to glutathione, the cell's master antioxidant. When glutathione levels are low, GPX4's ability to combat oxidative stress diminishes.

Enhancers:

Selenium | N-acetylcysteine (NAC)

SNP	Your Genotype
rs713041	TC
Alleles	Your genotype is linked to typical GPX4 activity, typical antioxidant protection and selenium needs
C: Typical GPX4 activity	
T: Altered GPX4 activity	

Intro and Health Effects

The main GPX4 variant is rs713041. Its "T" allele may be linked to:

- Colorectal cancer [R]
- Stroke [R]
- Poor prognosis of breast cancer [R]
- Pancreas inflammation [R]
- Vitamin B deficiencies [R]

However, some studies did not confirm the above findings [R, R].

Some studies even found protective associations between this variant and:

- High blood pressure in pregnancy [R]
- DNA damage [R]
- Heart problems in people with diabetes [R]
- Endometriosis [R]
- Alzheimer's disease [R]

Scientists are unsure about the reason behind these conflicting findings. This variant seems to be **beneficial when enough dietary selenium is available**, so people carrying it should pay special attention to selenium intake [R].

NDUFB6

NDUFB6 Report

The *NDUFB6* gene encodes an accessory subunit of mitochondrial Complex I, also known as the NADH dehydrogenase complex, which is the largest enzyme of the cellular respiratory chain [R].

This complex is located in the inner mitochondrial membrane, where it drives ATP production by oxidizing NADH to NAD+, transferring electrons to coenzyme Q10, and pumping protons across the membrane to create an electrochemical gradient [R].

The subunit encoded by *NDUFB6* is required for electron transfer activity [R].

Enhancers:

Riboflavin

SNP	Your Genotype
rs540467	CC
Alleles	Your genotype is linked to typical NDUFB6 activity, typical complex I function, and exercise benefits
C: Increased NDUFB6 activity	
T: Reduced NDUFB6 activity	

Intro and Health Effects

The best-researched *NDUFB6* variant is **rs540467**. Its 'T' allele has been associated with a **weaker response to physical activity**. Specifically with:

- Smaller beneficial effects on insulin sensitivity, body composition, and liver fat estimates in people with type 2 diabetes [R]
- Lower ATP production and insulin sensitivity improvement in relatives of type 2 diabetes patients [R, R]

NFE2L2

NFE2L2 Report

The *NFE2L2* gene is responsible for encoding a protein called NRF2, which plays a major role in your body's detoxification process. More specifically, NRF2 is responsible for activating many of your other genes that produce detox proteins [R].

It does this by binding to an area of other genes called the "antioxidant response element" (ARE), which increases gene expression [R, R].

According to some researchers, impaired NRF2 could potentially lead to several conditions, such as liver disease, Parkinson's, and cancer [R].

Enhancers:

Sulforaphane EGCG (green tea)

SNP

rs6721961

Alleles

G: Typical NFE2L2 activity and detox ability

T: Reduced NFE2L2 activity and detox ability

Your Genotype

GG

Your genotype is linked to typical NFE2L2 activity and detox ability.

Intro and Health Effects

The "T" allele at rs6721961 is one of the NFE2L2 variants that can reduce the expression and activity of NRF2. Reduced NRF2 impairs the body's ability to detox and defend itself from oxidative stress [R, R].

NFE2L2 gene variants may be linked to:

- Liver damage due to alcohol and other toxins [R, R]
- Drug and arsenic toxicity [R, R]
- Parkinson's disease (mixed evidence!) [R, R, R]
- Cancer [R, R, R, R, R, R]

SNP

rs35652124

Alleles

C: Typical NFE2L2 activity

T: Reduced NFE2L2 activity

Your Genotype

↓TT

Your genotype is linked to reduced NFE2L2 activity and reduced ability to boost antioxidant genes

Intro and Health Effects

Research has identified several variations in the NFE2L2 gene that can reduce the expression and activity of NRF2. This may impair the body's ability to detox and defend itself from oxidative stress [R, R].

NFE2L2 gene variants like **rs35652124-T** may be linked to:

- Liver damage due to alcohol and other toxins [R, R]
- Drug and arsenic toxicity [R, R]
- Parkinson's disease (mixed evidence!) [R, R, R]
- Cancer [R, R, R, R, R, R]

SNP

rs1806649

Alleles

G: Typical NFE2L2 activity

T: Reduced NFE2L2 activity

Your Genotype

↓CC

Your genotype is linked to reduced NFE2L2 activity and reduced ability to boost antioxidant genes

Intro and Health Effects

Research has identified several variations in the NFE2L2 gene that can reduce the expression and activity of NRF2. This may impair the body's ability to detox and defend itself from oxidative stress [R, R].

NFE2L2 gene variants like **rs1806649-C** may be linked to:

- Liver damage due to alcohol and other toxins [R, R]
- Drug and arsenic toxicity [R, R]
- Parkinson's disease (mixed evidence!) [R, R, R]
- Cancer [R, R, R, R, R, R]

NRF1

NRF1 Report

The *NRF1* gene encodes a transcription factor called nuclear respiratory factor 1. Together with NRF2, this protein activates the expression of nuclear genes required for mitochondrial respiration, mitochondrial DNA transcription, and cellular metabolism [R].

The NRF1 protein is also involved in heme biosynthesis [R].

Enhancers:

Omega-3

SNP	Your Genotype
rs6949152 Alleles **A:** Increased NRF1 activity **G:** Reduced NRF1 activity	↑AA **Your genotype is linked to increased NRF1 activity, improved mitochondrial production, and aerobic performance**

Intro and Health Effects

One of the main *NRF1* polymorphisms is **rs6949152**. Its major 'A' allele has been associated with a more oxidative-oriented muscular fiber type composition [R].

In line with this, people with the 'AA' genotype may **improve their aerobic capacity more** in response to aerobic training [R].

SNP	Your Genotype
rs2402970 Alleles **C:** Reduced NRF1 activity **T:** Increased NRF1 activity	↓CC **Your genotype is linked to reduced NRF1 activity, impaired mitochondrial production, and aerobic performance**

Intro and Health Effects

Another variant relevant to athletic performance is **rs2402970**. Its minor ‘T’ allele has been associated with **higher aerobic capacity** [R].

PINK1

PINK1 Report

The *PINK1* gene encodes a mitochondrial protein called PTEN-induced putative kinase 1. The function of this protein is not fully understood, but it's believed to protect cells against mitochondrial dysfunction due to stress, such as during periods of unusually high energy demands [R, R].

SNP

rs622525

Alleles

A: Increased PINK1 activity

T: Typical PINK1 activity

Your Genotype

TT

Your genotype is linked to typical PINK1 activity, typical mitochondrial recycling and blood sugar control

Intro and Health Effects

A study identified several *PINK1* variants associated with gene expression, among which **rs622525** was the key variant. Its minor 'A' allele has been associated with increased gene expression [R].

This allele has been associated with lower fasting glucose and a decreased risk of type 2 diabetes [R, R].

PPARA

PPARA Report

The PPARA gene encodes a protein called PPAR-α (short for peroxisome proliferator-activated receptor alpha). PPAR-α is part of the larger PPAR family. In general, PPARs bind to polyunsaturated fatty acids [R].

PPAR-α signals the liver to break down more fat. When you're fasting, and there aren't enough carbs in your body to burn for energy, PPAR-α helps trigger ketogenesis, the process of burning fat for energy [R, R].

The PPARA gene is important for determining your response to diet because PPAR-α is under strong influence of nutrients, especially fats. Its main natural activators are polyunsaturated fatty acids (PUFAs) [R].

Enhancers:

Omega-3

SNP

rs1800206

Alleles

G: Reduced PPARA activity

C: Typical PPARA activity

Your Genotype

CC

Your genotype is linked to typical PPARA activity, typical saturated fat metabolism, and lipid profile

Intro and Health Effects

The main PPARA SNP in nutrigenomics is rs1800206. Its minor 'G' allele (also known as the L162V polymorphism) reduces PPAR-α activity, according to the available data [R].

Carrying this allele has been associated with [R]:

- Increased heart disease risk in whites, with higher levels of triglycerides, total cholesterol, LDL, apoA1, and apoB, and decreased levels of HDL

Moreover, people carrying at least one 'G' allele who consumed high amounts of saturated fat had smaller LDL particles than those with lower intakes in a study. Based on this, they **don't seem to be well-suited for a diet high in saturated fat** [R].

A study of 2373 participants found an association between the 'G' allele and higher total and LDL cholesterol in men and apolipoprotein B in both genders [R].

A follow-up study of 2106 people from the same cohort concluded that ‘G’-allele carriers had greater triglyceride and apoC-III levels when they consumed a low-PUFA diet. But when their **PUFA intake was high, they had lower triglyceride and apoC-III levels.** The authors pointed out that the more PUFAs ‘G’-allele carriers ate, the more their triglycerides and apoC-III levels dropped--and vice versa [R, R].

PPARGC1A

PPARGC1A Report

The *PPARGC1A* gene encodes a protein called PPARG coactivator 1 alpha, or PGC-1α. PGC-1α regulates the expression of genes that help produce energy in the cell.

It is involved in the production of new mitochondria, as well as in the function of existing mitochondria. Along with *UCP3*, *PPARGC1A* helps your body stay warm by converting energy into heat [R, R].

PGC-1α increases PPARG and thyroid hormone levels. It also affects how energy metabolism changes over a 24-hour cycle (following a circadian rhythm) [R].

The expression of this gene is affected by exercise, fasting, and cold exposure [R, R].

Enhancers:

Resveratrol

SNP	Your Genotype
rs8192678	CT
Alleles	
C: Increased PPARGC1A activity	
T: Reduced PPARGC1A activity	

Your genotype is linked to typical PPARGC1A activity, typical athletic performance & blood sugar control

Intro and Health Effects

The main PPARGC1A polymorphism is rs8192678. Its minor 'T' allele decreases PPARGC1A expression and PGC-1α levels in the muscles [R, R, R].

This variant has been associated with a decreased overall athletic ability and sports performance, especially in endurance sports such as long-distance running and cycling. Moreover, carriers may benefit less from aerobic exercise for improving their aerobic capacity, gaining muscle mass, and lowering LDL cholesterol [R, R, R].

This variant has been associated with an increased risk of type 2 diabetes in European, Indian, and Chinese populations, as well as with higher blood pressure in individuals younger than 50 years old [R, R, R, R].

PRKAA2

PRKAA2 Report

The *PRKAA2* gene encodes the catalytic α-2 subunit of AMP-activated protein kinase (AMPK). This protein is a master cellular energy sensor that activates ATP production under conditions of low cellular energy [R, R].

AMPK activates enzymes that catalyze ATP production by breaking down fatty acids in the mitochondria, while switching off ATP-consuming pathways such as cell growth and proliferation. This ensures that energy homeostasis is maintained within the cell [R].

SNP

rs2746342

Alleles

G: Altered PRKAA2 activity

T: Improved PRKAA2 activity

Your Genotype

TT

Your genotype is linked to improved PRKAA2 activity, improved mitochondrial recycling, and blood sugar control

Intro and Health Effects

One of the main *PRKAA2* variants is **rs2746342**. Its minor 'G' allele has been associated with higher fasting glucose, as well as with an increased risk of type 2 diabetes and diabetic nephropathy [R, R, R].

SNP

rs10789038

Alleles

A: Altered PRKAA2 activity

G: Improved PRKAA2 activity

Your Genotype

GG

Your genotype is linked to improved PRKAA2 activity, improved mitochondrial recycling and blood sugar control

Intro and Health Effects

Another minor variant, the 'A' allele of **rs10789038**, has been associated with an increased risk of type 2 diabetes [R, R].

SNP

rs2796498

Alleles

A: Altered PRKAA2 activity

G: Improved PRKAA2 activity

Your Genotype

AA

Your genotype is linked to altered PRKAA2 activity, altered mitochondrial recycling, and impaired blood sugar control

Intro and Health Effects

Finally, the major 'A' allele of **rs2796498** has been associated with an increased risk of type 2 diabetes and diabetic nephropathy [R, R].

SIRT1

SIRT1 Report

The *SIRT1* gene encodes a sirtuin protein. Sirtuins are a group of enzymes heavily implicated in aging, cell death, inflammation, mental and physical stress resistance, and energy metabolism. They regulate and “turn off” other genes, especially those involved in the process of aging [R, R, R, R].

Many age-related and degenerative diseases are characterized by low sirtuin activity [R, R].

Sirtuin activity is also unusually low in chronic inflammation. Some researchers believe that activating the sirtuins—and especially SIRT1—may help manage obesity, atherosclerosis, diabetes, and other inflammatory diseases [R].

Given all of this, it’s not surprising that SIRT1 has been touted as a longevity gene.

Enhancers:

Resveratrol

SNP	Your Genotype
rs7895833 Alleles **A:** Typical SIRT1 activity **G:** Increased SIRT1 activity	AA **Your genotype is linked to typical SIRT1 activity, typical sirtuin 1 levels, and longevity genetics**

Intro and Health Effects

Another SNP with a large potential impact on lifespan is rs7895833: the ‘G’ allele here is almost twice as common in older people as it is in the average adult. Carriers of the ‘AA’ genotype have lower levels of this protein [R, R].

SNP	Your Genotype
rs12778366 Alleles **C:** Increased SIRT1 activity **T:** Typical SIRT1 activity	↑TC **Your genotype is linked to increased SIRT1 activity, improved sirtuin 1 levels, and longevity genetics**

Intro and Health Effects

A handful of studies have shown that people with certain SIRT1 variants are more likely to live very long lives.

One of the variations with the strongest effect so far is rs12778366: even one copy of the ‘C’ allele is linked to 30% lower all-cause mortality risk. This variant also increases glucose tolerance and SIRT1 levels [R, R].

SNP

rs3758391

Alleles

C: Typical SIRT1 activity

T: Increased SIRT1 activity

Your Genotype

↑CT

Your genotype is linked to increased SIRT1 activity, improved sirtuin 1 levels, and longevity genetics

Intro and Health Effects

The ‘T’ allele of the rs3758391 variant has been associated with reduced cardiovascular mortality and decreased cognitive decline during aging. This variant increases SIRT1 levels [R, R, R].

SIRT3

SIRT3 Report

The *SIRT3* gene encodes a sirtuin protein. Sirtuins are a group of mitochondrial enzymes heavily implicated in aging, cell death, inflammation, mental and physical stress resistance, and energy metabolism. They regulate and "turn off" other genes, especially those involved in the process of aging [R, R, R].

Many age-related and degenerative diseases are characterized by low sirtuin activity [R, R].

Sirtuin activity is also unusually low in chronic inflammation. Some researchers believe that activating the sirtuins may help manage obesity, atherosclerosis, diabetes, and other inflammatory diseases [R].

Given all of this, it's not surprising that *SIRT3* has been touted as a longevity gene.

SNP	Your Genotype
rs11555236 Alleles **A:** Increased SIRT3 activity **C:** Reduced SIRT3 activity	↓CC **Your genotype is linked to reduced SIRT3 activity, lower sirtuin 3 levels, and worse longevity genetics**

Intro and Health Effects

A handful of studies have shown that people with certain SIRT3 variants are more likely to live very long lives.
One of the variations with the strongest effect so far is rs11555236 (G477T). People homozygous for the minor 'A' allele may live longer lives. This variant has been found to increase SIRT3 levels [R, R, R].

SNP	Your Genotype
rs4980329 Alleles **C:** Reduced SIRT3 activity **T:** Increased SIRT3 activity	↓CC **Your genotype is linked to reduced SIRT3 activity, lower sirtuin 3 levels and worse longevity genetics**

Intro and Health Effects

Another SNP with a potential impact on lifespan is rs4980329: the ‘T’ allele is more common among long-lived individuals. This variant has also been associated with a decreased risk of ALS and ischemic stroke from the rupture of unstable carotid plaques [R, R, R].

SNP

rs11246020

Alleles

C: Increased SIRT3 activity

T: Reduced SIRT3 activity

Your Genotype

↑CC

Your genotype is linked to increased SIRT3 activity, improved sirtuin 3 levels and longevity genetics

Intro and Health Effects

In contrast, the ‘T’ allele of rs11246020 may decrease SIRT3 activity and has been associated with an increased risk of metabolic syndrome and stomach cancer, and higher HbA1c levels.

SIRT6

SIRT6 Report

The SIRT6 gene encodes a sirtuin protein. Sirtuins are a group of mitochondrial enzymes heavily implicated in aging, cell death, inflammation, mental and physical stress resistance, and energy metabolism. They regulate and “turn off” other genes, especially those involved in the process of aging [R, R, R].

When your cells experience stress, acetyl groups are added to proteins as a response to changes induced by inflammation and oxidation. Sirtuins remove these acetyl groups to keep the protein in service longer than usual [R, R].

Enhancers:

Quercetin

SNP	Your Genotype
rs107251	CC
Alleles **C:** Typical SIRT6 activity **T:** Reduced SIRT6 activity	**Your genotype is linked to typical SIRT6 activity, typical sirtuin 6 levels and longevity & cardiovascular genetics**

Intro and Health Effects

One of the main SIRT6 variants is rs107251. A study of ~1200 elderly participants found that carriers of at least one copy of the **minor ‘T’ allele had a 5.5- 5.9-year shorter lifespan** compared to ‘CC’ homozygotes [R].

The ‘T’ allele has been associated with an increased risk of **cardiovascular diseases** such as coronary artery disease or atherosclerosis, which may explain its link with reduced longevity [R, R, R].

UCP3

UCP3 Report

The *UCP3* gene encodes a protein called '*uncoupling protein 3*'. In the mitochondria (the main powerhouse of cells), the breakdown of nutrient fuels is *coupled* to the production of the energy molecule ATP. Members of the uncoupling protein family prevent the production of this molecule and instead cause the energy to be dissipated as heat [R, R].

UCP3 is mainly found in muscles, but is also present in smaller amounts in the heart and brown fat tissues. Fasting and acute exercise increase the expression of the *UCP3* gene [R, R, R, R, R, R, R].

SNP

rs1800849

Alleles

A: Increased UCP3 activity

G: Reduced UCP3 activity

Your Genotype

↓ GG

Your genotype is linked to reduced UCP3 activity and fat burning

Intro and Health Effects

The main UCP3 polymorphism by far is rs1800849, also known as -55C/T. A study in Pima Indians found that the **minor allele 'A' increases UCP3 expression**, and possibly the levels of the protein, in the muscles [R].

Some studies suggest that the minor variant at this polymorphism may **protect from obesity**, especially in Caucasians. The mixed results observed may be due to differences in the diet, physical activity, and genetic background of the different populations [R, R, R, R].

On the other hand, two meta-analyses confirmed the association of the 'A' variant with **increased susceptibility to type 2 diabetes in Asians** but not in Europeans [R, R].

SNP

rs647126

Alleles

A: Reduced UCP3 activity

G: Increased UCP3 activity

Your Genotype

○ GA

Your genotype is linked to typical UCP3 activity and fat burning

Intro and Health Effects

The minor variants at the rs647126 and rs2075577 polymorphisms, which are usually inherited together, were associated with **increased BMI in Dutch men**. Based on their effect, the authors of the study speculated that these variants may reduce UCP3 activity [R].

GPX1

GPX1 Report

The GPX1 gene helps make glutathione peroxidase (GPx), one of the body's key antioxidant enzymes. This enzyme converts hydrogen peroxide and glutathione into glutathione disulfide and water. By doing so, GPx helps reduce oxidative stress [R].

Increased oxidative stress opens the door for DNA damage and cell death, which in turn can lead to tissue damage and a range of health conditions [R, R, R].

Glutathione is sometimes called the "mother of all antioxidants" because of how essential it is for the body's antioxidant defenses. It plays a central role in the function of GPx [R, R].

Enhancers:

Selenium | N-acetylcysteine (NAC)

SNP

rs1050450

Alleles

A: Lower GPX1 activity

G: Higher GPX1 activity

Your Genotype

↑GG

Your genotype is linked to higher GPX1 activity, better antioxidant protection, and longevity

Intro and Health Effects

One study found a direct link between a common GPX1 variant and human longevity. According to a cohort of elderly Danish people born in 1905, the heterozygous **genotype 'AG' at rs1050450 was significantly more common in the very elderly** than in the general population [R].

Other studies have strongly suggested that the **'G' allele at rs1050450 confers higher GPx activity**, which is linked to better health outcomes [R, R].

Along with other variants (like rs1800668 and rs3811699), this variant has also been linked to [R, R, R, R, R, R, R]:

- Rheumatoid arthritis
- Kashin-Beck disease
- Heart disease
- Some types of cancer

Kashin-Beck disease (KBD) is a bone disease that causes arthritis-like joint pain. People with KBD tend to have significantly higher oxidative stress and significantly lower selenium, suggesting that the disease could be caused (at least in part) by poor GPx activity [R, R].

NQO1

NQO1 Report

The NQO1 gene encodes an enzyme also named NQO1, which is short for NADPH dehydrogenase quinone 1. The main responsibility of this enzyme is to detoxify quinones [R, R].

Quinones are a family of chemical compounds that can cause oxidative stress, cellular injury, and DNA damage. These negative effects can play a role in the development of cancer and other health conditions [R, R].

NQO1 helps detox quinones by converting them into hydroquinone, a much safer compound that generally does not cause oxidative stress. Other functions of NQO1 include converting ubiquinone (CoQ10) and vitamin E into their antioxidant forms [R].

Enhancers:

Sulforaphane Curcumin

SNP

rs1800566

Alleles

A: Reduced NQO1 activity

G: Increased NQO1 activity

Your Genotype

↑GG

Your genotype is linked to increased NQO1 activity, improved CoQ10 regeneration, antioxidant defense, and quinone detox

Intro and Health Effects

Certain NQO1 gene variants may impair the body's ability to detox by reducing the activity of this enzyme [R, R].

NQO1 gene variants may play a role in DNA damage. Research has found their associations with different types of cancer [R, R, R, R, R].

One of the main culprits is a variant called **NQO1*2**, which corresponds to rs1800566-A. People with this variant have reduced NQO1 activity [R, R].

Research shows that people with the NQO1*2 variant (rs1800566-A) are up to **7.6 times more likely to experience benzene toxicity**, even from low-level exposure [R, R, R].

SNP	Your Genotype
rs1131341 Alleles **A:** Reduced NQO1 activity **G:** Increased NQO1 activity	↑GG **Your genotype is linked to increased NQO1 activity, improved CoQ10 regeneration, antioxidant defense, and quinone detox**

Intro and Health Effects

Another important variant is **NQO1*3**, which corresponds to rs1131341-A. It leads to a slower conversion of quinone into its safer metabolites [R].

PRKAA1

PRKAA1 Report

The PRKAA1 gene encodes the catalytic α-1 subunit of AMP-activated protein kinase (AMPK). This protein is a master cellular energy sensor that activates ATP production under conditions of low cellular energy [R, R].

AMPK activates enzymes that catalyze ATP production by breaking down fatty acids in the mitochondria, while switching off ATP-consuming pathways such as cell growth and proliferation. This ensures that energy homeostasis is maintained within the cell [R].

The AMPK enzyme also plays a major role in autophagy, the process by which cells clear out damaged organelles and proteins, thus protecting cells from stress [R].

SNP	Your Genotype
rs13361707 Alleles **C:** Altered PRKAA1 activity **T:** Improved PRKAA1 activity	TT **Your genotype is linked to improved PRKAA1 activity, improved mitochondrial recycling, and lower odds of stomach problems**

Intro and Health Effects

The **'C' allele of rs13361707** has been associated with an increased risk of stomach cancer and faster progression [R, R, R, R, R, R, R].

The allele may further increase the risk conferred by H. pylori infection and high BMI [R, R, R, R].

SNP	Your Genotype
rs10074991 Alleles **A:** Improved PRKAA1 activity **G:** Altered PRKAA1 activity	AA **Your genotype is linked to improved PRKAA1 activity, improved mitochondrial recycling, and lower odds of stomach problems**

Intro and Health Effects

Usually inherited with the previous variant, the ‘G’ allele of rs10074991 has been associated with an increased risk of stomach cancer [R, R, R, R, R, R, R].

SLC16A1

SLC16A1 Report

The SLC16A1 gene encodes a protein called solute carrier family 16 member 1. This gene is sometimes known as MCT1 or monocarboxylate transporter 1 [R].

SLC16A1 transports certain compounds across the cell membrane (into and out of cells). These compounds include ketone bodies, lactate, pyruvate, and lactic acid. Because lactate and pyruvate are important breakdown products of **dietary carbohydrates**, this gene is essential in the process of **obtaining energy** from these macronutrients (glycolysis). The SLC16A1 gene is also considered one of the most important for predicting how much we exercise [R, R].

SNP	Your Genotype
rs1049434 Alleles **A:** Reduced SLC16A1 activity **T:** Increased SLC16A1 activity	↑ TT **Your genotype is linked to increased SLC16A1 activity, improved lactate clearance, and endurance performance**

Intro and Health Effects

The main SLC16A1 variant is rs1049434. Its **minor 'A' allele** encodes a protein with a 60-65% reduction in lactate transport, resulting in **higher blood lactate levels during high-intensity exercise**. Carriers may also have higher oxygen requirements and glycogen use. In line with this, the variant has been associated with **decreased endurance performance** and lower odds of reaching elite status [R, R, R, R, R, R, R, R, R, R, R, R, R, R].

However, this variant has been associated with **better performance at power- and strength-oriented sports** such as wrestling, a reduced incidence of muscle and bone injuries in athletes, and faster lactate clearance during exercise recovery [R, R, R, R].

TFAM

TFAM Report

The *TFAM* gene encodes a key transcription factor produced in the nucleus and transported to the mitochondria, where it is responsible for the transcription, replication, and structural packaging of mitochondrial DNA (mtDNA) [R].

Enhancers:

Resveratrol

SNP	Your Genotype
rs1937 Alleles **G:** Reduced TFAM activity **C:** Increased TFAM activity	○ CG **Your genotype is linked to typical TFAM activity, typical mitochondrial DNA processing, longevity & cognitive function**

Intro and Health Effects

The main *TFAM* variant is **rs1937**. Its minor 'C' allele has been associated with:

- Increased longevity [R, R]
- Decreased risk of Alzheimer's disease [R, R, R, R]
- Greater intelligence in Alzheimer's disease patients [R]

SNP	Your Genotype
rs2306604 Alleles **A:** Reduced TFAM activity **G:** Increased TFAM activity	↑ GG **Your genotype is linked to increased TFAM activity, improved mitochondrial DNA processing, longevity & cognitive function**

Intro and Health Effects

Another variant, the major ‘A’ allele of **rs2306604**, has been associated with an increased risk of Alzheimer’s and Parkinson’s disease dementia [R, R]
On the bright side, it’s linked to increased athletic performance [R]

UCP2

UCP2 Report

The *UCP2* gene encodes a protein called '*uncoupling protein 2*'. In the mitochondria (the main powerhouse of cells), the breakdown of nutrient fuels is *coupled* to the production of the energy molecule ATP. Members of the uncoupling protein family prevent the production of this molecule and instead cause the energy to be dissipated as heat [R, R].

UCP2 is found in a wide variety of tissues, such as brown and white fat, muscles, pancreatic insulin-producing cells, the immune system, the heart, and the liver. Its expression is increased when blood fatty acid levels are high, such as during fat burning or when eating a high-fat diet. Conversely, obese and diabetic people have lower UCP2 levels [R, R, R, R, R, R].

UCP2 may also protect the brain and other tissues from oxidative stress [R, R, R, R, R].

Enhancers:

Quercetin Resveratrol

SNP

rs659366

Alleles

C: Reduced UCP2 activity

T: Increased UCP2 activity

Your Genotype

TC

Your genotype is linked to balanced UCP2 activity, balanced fat burning and blood sugar control

Intro and Health Effects

The minor '**T' variant of the rs659366 increases UCP2 expression** in the fatty tissue [R, R, R].

This allele showed a protective effect from obesity in multiple studies [R, R, R, R, R, R, R, R].

However, many studies failed to confirm these results or even found the opposite ones [R, R, R, R, R, R, R, R, R, R, R, R, R].

Despite these discrepancies, **all meta-analyses agree that the 'T' variant is associated with lower obesity rates and BMI in European** but not in Asian populations [R, R, R, R].

On the other hand, this variant may have **negative effects on blood lipids, glucose control, and diabetes outcomes** [R, R, R, R, R, R, R, R, R].

Lab markers to check

ALT — 63 U/L — 4 May 2026

ApoB — 132 mg/dL — 13 Jan 2026

Hemoglobin A1c — 40 mmol/mol — 25 Mar 2026

LDL Cholesterol — 4.4 mmol/L — 25 Mar 2026

GGT — 46 IU/L — 25 Mar 2026

Personalized to Your Genes

NFE2L2

Elevated GGT reflects oxidative stress and glutathione demand, rising when NRF2 defense is impaired.

Hemoglobin A1c (%) — 5.4 % — 13 Jan 2026

Insulin, Fasting — 8.7 uIU/mL — 13 Jan 2026

Personalized to Your Genes

↓ NAMPT — Low NAD+ impairs sirtuin-driven metabolic regulation, making insulin resistance a key downstream marker.

Iron — 69 ug/dL — 13 Jan 2026

Triglycerides — 1.53 mmol/L — 25 Mar 2026

TSH — 2.64 mIU/L — 4 May 2026

Personalized to Your Genes

↓ UCP1 — T3 is a key activator of UCP1 transcription; thyroid dysfunction compounds the downregulation.

UCP3 — T3 drives UCP3 expression, so thyroid status directly modulates how much the gene is expressed.

VO2 Max — 35.1 mL/kg/min — 25 Mar 2026

Personalized to Your Genes

NRF1 — The functional gold standard for mitochondrial density and aerobic capacity that NRF1 governs.

UCP3 — Reflects muscle mitochondrial fat-oxidation efficiency, the primary functional consequence of low UCP3.

Creatine Kinase — 173 IU/L — 9 Nov 2025

Personalized to Your Genes

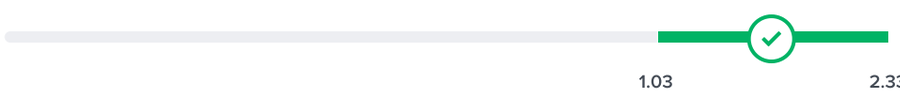
↓ COQ2

CoQ10 deficiency can cause muscle bioenergetic stress; CK flags muscular involvement worth monitoring.

Ferritin — 130 ng/mL — 13 Jan 2026

Glucose, Fasting — 81 mg/dL — 12 Jun 2026

HDL Cholesterol — 1.65 mmol/L — 25 Mar 2026



Ketones, Urine — 25 Mar 2026

T3, Free (FT3) — 3.2 pg/mL — 13 Jan 2026

Personalized to Your Genes

↓ UCP1

T3 is a key activator of UCP1 transcription; thyroid dysfunction compounds the downregulation.

UCP3

T3 drives UCP3 expression, so thyroid status directly modulates how much the gene is expressed.

T4 Free (FT4) — 11.4 pmol/L — 4 May 2026

Personalized to Your Genes

↓ UCP1

T3 is a key activator of UCP1 transcription; thyroid dysfunction compounds the downregulation.

UCP3

T3 drives UCP3 expression, so thyroid status directly modulates how much the gene is expressed.

TIBC

379 mcg/dL

9 Nov 2025

8-Hydroxy-2-deoxyguanosine, Urine

Personalized to Your Genes

SOD2

Directly quantifies the mitochondrial oxidative burden that reduced SOD2 fails to neutralize.

NFE2L2

Quantifies the oxidative damage resulting from a weakened NRF2-driven antioxidant response.

SIRT3

SIRT3 governs mitochondrial antioxidant defense via SOD2; elevated markers reveal the consequence of reduced activity.

C-Peptide

C-Reactive Protein (CRP)

Personalized to Your Genes

↓ NAMPT

Given the paradoxical lower-inflammation effect, tracking CRP clarifies the net inflammatory picture and metabolic tradeoff.

SOD2

Reflects the oxidative-inflammatory state relevant to the nerve-protection concern of this variant.

Carnitine, Total

Personalized to Your Genes

SIRT3 — SIRT3 activates fat-oxidation enzymes; abnormal acylcarnitines reflect impaired mitochondrial fat metabolism.

UCP3 — Carnitine supports the fat oxidation UCP3 facilitates; deficiency compounds the deficit and is correctable.

Coenzyme Q10

Personalized to Your Genes

↓ COQ2 — Directly measures the deficiency caused by impaired biosynthesis and tracks supplementation adequacy.

F2-Isoprostanes

Personalized to Your Genes

SOD2 — Directly quantifies the mitochondrial oxidative burden that reduced SOD2 fails to neutralize.

NFE2L2 — Quantifies the oxidative damage resulting from a weakened NRF2-driven antioxidant response.

SIRT3 — SIRT3 governs mitochondrial antioxidant defense via SOD2; elevated markers reveal the consequence of reduced activity.

Free Fatty Acids

Personalized to Your Genes

↓ UCP1 — Brown fat needs FFA substrate; impaired UCP1 fat oxidation may manifest as elevated fasting FFAs.

Helicobacter pylori, Urea Breath Test

HOMA-IR

Personalized to Your Genes

↓ NAMPT	Low NAD+ impairs sirtuin-driven metabolic regulation, making insulin resistance a key downstream marker.

IL-6

Intracellular NAD (icNAD)

Personalized to Your Genes

↓ NAMPT	Directly measures the NAD+ status that impaired NAMPT compromises, the central consequence of this variant.

L-Lactate (Genova)

Personalized to Your Genes

↓ COQ2	An elevated ratio signals electron transport chain dysfunction, a downstream consequence of CoQ10 insufficiency.
NRF1	Indicates oxidative capacity and helps calibrate training given reduced mitochondrial production. Reflects electron transport chain capacity, which declines when NRF1-driven biogenesis is impaired.
SIRT3	Reflects overall mitochondrial oxidative efficiency, which declines with reduced SIRT3 function.
UCP3	Reflects muscle mitochondrial fat-oxidation efficiency, the primary functional consequence of low UCP3.

Malondialdehyde

Manganese, Blood

Personalized to Your Genes

SOD2 — Confirms adequate cofactor status for the SOD2 enzyme, the most correctable input to its activity.

Nicotinamide (Vitamin B3)

Postprandial Glucose

Pyruvic Acid

Personalized to Your Genes

↓ COQ2 — An elevated ratio signals electron transport chain dysfunction, a downstream consequence of CoQ10 insufficiency.

NRF1 — Reflects electron transport chain capacity, which declines when NRF1-driven biogenesis is impaired.

SIRT3 — Reflects overall mitochondrial oxidative efficiency, which declines with reduced SIRT3 function.

Selenium

Total Glutathione

Personalized to Your Genes

NFE2L2

NRF2 governs glutathione synthesis; this reveals whether the body's master antioxidant is depleted.

Vitamin B2 (Riboflavin), Plasma

Glossary

8-OHdG (8-Hydroxy-2'-Deoxyguanosine)
A marker of oxidative damage to DNA, often used to assess oxidative stress levels.

Acetyl-CoA
A key molecule that links the breakdown of fats, carbohydrates, and proteins to energy production.

AMP:ATP Ratio
A measure of cellular energy status. A high ratio signals low energy and activates energy-producing pathways.

ATP (Adenosine Triphosphate)
The primary energy currency of the cell, used to power nearly all biological processes.

CoQ10 (Coenzyme Q10)
A compound that helps transport electrons in mitochondria and supports cellular energy production.

Complex I
The first protein complex of the electron transport chain, where electrons from NADH enter energy production.

Complex III
A protein complex that transfers electrons through the electron transport chain and contributes to ATP generation.

Complex IV
The final protein complex of the electron transport chain, where oxygen is used to complete energy production.

Cytochrome C
A protein that carries electrons between mitochondrial complexes and supports ATP production.

FAD+
A coenzyme involved in energy metabolism that accepts electrons during cellular respiration.

FADH2
The electron-rich form of FAD that delivers energy to the electron transport chain for ATP production.

Glutathione
The body’s main antioxidant, helping protect cells and mitochondria from oxidative damage.

H2O2 (Hydrogen Peroxide)
A reactive oxygen species produced during metabolism that can contribute to oxidative stress if not neutralized.

Krebs Cycle
A series of chemical reactions in mitochondria that generate energy-carrying molecules used to produce ATP.

Lactate
A byproduct of glucose metabolism that can be recycled and used as an energy source by cells.

Lipid Peroxides
Damaged fats created by oxidative stress that can harm cell membranes if not cleared.

MDA (Malondialdehyde)
A byproduct of lipid peroxidation commonly used as a marker of oxidative stress.

NAD+ (Nicotinamide Adenine Dinucleotide)
A coenzyme essential for energy production, DNA repair, and healthy mitochondrial function.

NADH
The electron-rich form of NAD+ that supplies energy to the electron transport chain.

Nicotinamide
A form of vitamin B3 used by the body to produce NAD+, an essential coenzyme for energy metabolism.

NMN (Nicotinamide Mononucleotide)
A precursor to NAD+ that supports cellular energy production and healthy aging.

Proton Gradient
A concentration difference of protons across the mitochondrial membrane that drives ATP production.

Pyruvate
A molecule produced from glucose that can be converted into Acetyl-CoA for energy generation.

Riboflavin (Vitamin B2)
A vitamin required to produce FAD+, a key coenzyme involved in mitochondrial energy production.

Superoxide O_2
A reactive oxygen species naturally produced during energy generation that can contribute to oxidative stress.

Uncoupling
A process where mitochondria release energy as heat instead of producing ATP, affecting metabolic efficiency.