SelfDecode™

Response to Peptides

Summary Report

REPORT CATEGORIES

- MENTAL HEALTH
- COGNITION
- INFLAMMATION & AUTOIMMUNITY
- IMMUNITY & INFECTIONS
- WEIGHT & BODY FAT
- BLOOD SUGAR CONTROL
- LONGEVITY
- INJURIES
- SEXUAL HEALTH
- REPRODUCTIVE HEALTH
- FITNESS

Sample Client

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Table of Contents

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.

Personal information

NAME

Sample Client

SEX AT BIRTH

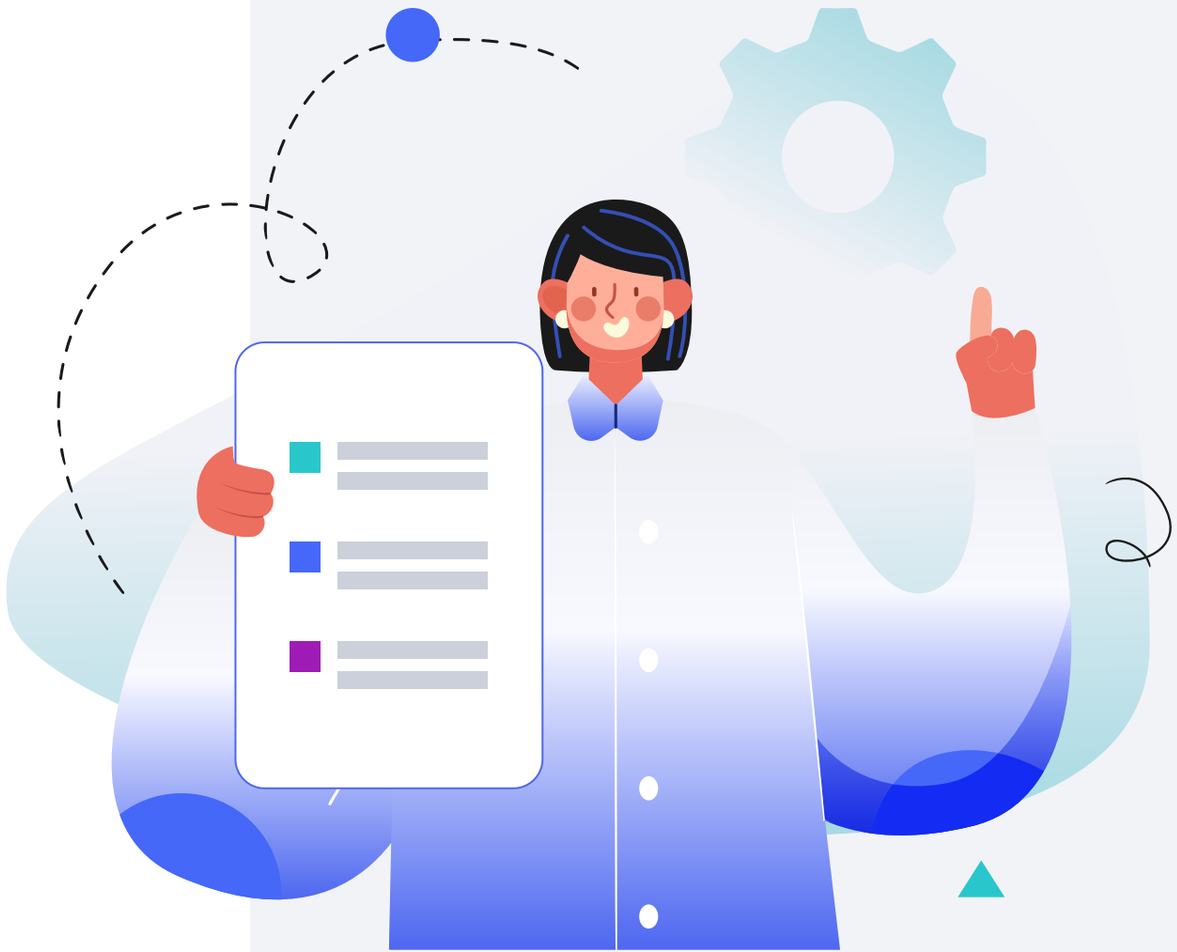
Female

HEIGHT

5ft 0" 153cm

WEIGHT

110lb 50kg



Summary

Please note: *The claims in this report haven't been verified by scientific research. They are hypotheses based on the mechanisms of these peptides. Few genetic variants have been found to modify the effectiveness of therapeutic peptides.*

Therapeutic peptides are short chains of amino acids that help regulate vital processes from healing and metabolism to mood and immunity. These naturally occurring molecules, and their synthetic analogs, can fine-tune how your body repairs tissue, manages inflammation, balances hormones, and even supports brain function.

In this report, we explore a wide range of peptides that influence key aspects of health and performance. You'll learn how different classes of therapeutic peptides — from growth and repair peptides like BPC-157 and TB-500, to nootropic peptides like Semax and Dihexa, and metabolic regulators like Tirzepatide and MOTS-c — interact with your body's cellular systems to impact immune balance, longevity pathways, and stress resilience.

Understanding how these peptides work and how your genetic profile may influence their effectiveness can offer valuable insights into optimizing your health. While peptides are not a substitute for medical treatment, learning how they align with your body's unique biology can help guide more personalized and evidence-based wellness strategies.

This summary report contains:

43 **Genetic Results**

Overview of Your Results

Cognition & Mood

WORSE RESPONSE

Response to Semax (Hypothesis)

Predisposed to a worse response to Semax

WORSE RESPONSE

Response to Synapsin (Hypothesis)

Predisposed to a worse response to Synapsin

TYPICAL RESPONSE

Response to Selank (Hypothesis)

Predisposed to a typical response to Selank

TYPICAL RESPONSE

Response to Dihexa (Hypothesis)

Predisposed to a typical response to Dihexa

TYPICAL RESPONSE

Response to Cerebrolysin (Hypothesis)

Predisposed to a typical response to Cerebrolysin

TYPICAL RESPONSE

Response to Cyclo (Hypothesis)

Predisposed to a typical response to Cyclo (His-Pro)

TYPICAL RESPONSE

Response to Pinealon (Hypothesis)

Predisposed to a typical response to Pinealon

TYPICAL RESPONSE

Response to DSIP (Hypothesis)

Predisposed to a typical DSIP response

Muscle Growth & Tissue Repair

TYPICAL RESPONSE

Response to PEG-MGF (Hypothesis)

Predisposed to a typical response to PEG-MGF

TYPICAL RESPONSE

Response to Follistatin-344 (Hypothesis)

Predisposed to a typical response to Follistatin-344

TYPICAL RESPONSE

Response to GHRP-2 (Hypothesis)

Predisposed to a typical response to GHRP-2

TYPICAL RESPONSE

Response to GHRP-6 (Hypothesis)

Predisposed to a typical response to GHRP-6

TYPICAL RESPONSE

Response to CJC-1295 (Hypothesis)

Predisposed to a typical response to CJC-1295

TYPICAL RESPONSE

Response to Thymosin Beta-4 (Hypothesis)

Predisposed to a typical response to Thymosin beta-1

BETTER RESPONSE

Response to BPC-157 (Hypothesis)

Predisposed to a better BPC-157 response

BETTER RESPONSE

Response to TB-500 (Hypothesis)

Predisposed to a better response to TB-500

BETTER RESPONSE

Response to Ipamorelin (Hypothesis)

Predisposed to a better response to Ipamorelin

BETTER RESPONSE

Response to Sermorelin (Hypothesis)

Predisposed to a better response to Sermorelin

Weight Management & Energy Metabolism

WORSE RESPONSE

Response to GLP-1 (Ozempic)

Likely worse response to GLP-1 (Ozempic)

TYPICAL RESPONSE

Response to Retatrutide (Hypothesis)

Predisposed to a typical response to Retatrutide

TYPICAL RESPONSE

Response to Tirzepatide (Hypothesis)

Predisposed to a typical response to Tirzepatide

TYPICAL RESPONSE

Response to 5-Amino-1MQ (Hypothesis)

Predisposed to a typical response to 5-amino-1MQ

BETTER RESPONSE

Response to AOD-9604 (Hypothesis)

Predisposed to a better response to AOD-9604

BETTER RESPONSE

Response to MOTS-c (Hypothesis)

Predisposed to a better MOTS-c response

BETTER RESPONSE

Response to SS-31 (Hypothesis)

Predisposed to a better SS-31 response

Inflammation & Immune Modulation

WORSE RESPONSE

Response to KPV (Hypothesis)

Predisposed to a worse response to KPV

WORSE RESPONSE

Response to Larazotide (Hypothesis)

Predisposed to a worse response to Larazotide

TYPICAL RESPONSE

Response to Thymosin Alpha-1 (Hypothesis)

Predisposed to a typical response to Thymosin alpha-1

TYPICAL RESPONSE

Response to Thymogen (Hypothesis)

Predisposed to a typical response to Thymogen

TYPICAL RESPONSE

Response to Thymalin (Hypothesis)

Predisposed to a typical response to Thyamlin

TYPICAL RESPONSE

Response to ARA 290 (Hypothesis)

Predisposed to a typical response to ARA 290

TYPICAL RESPONSE

Response to LL-37 (Hypothesis)

Predisposed to a typical response to LL-37

TYPICAL RESPONSE

Response to VIP (Hypothesis)

Predisposed to a typical response to VIP

Sexual & Reproductive Health

TYPICAL RESPONSE

Response to Bremelanotide (Hypothesis)

Predisposed to a typical response to bremelanotide

TYPICAL RESPONSE

Response to Kisspeptin (Hypothesis)

Predisposed to a typical response to Kisspeptin

BETTER RESPONSE

Response to Oxytocin (Hypothesis)

Predisposed to a better response to oxytocin

BETTER RESPONSE

Response to Melanotan II (Hypothesis)

Predisposed to a better response to Melanotan II

∞ Anti-Aging & Longevity

TYPICAL RESPONSE

Response to Epitalon (Hypothesis)

Predisposed to a typical response to Epitalon

TYPICAL RESPONSE

Response to Vilon (Hypothesis)

Predisposed to a typical response to Vilon

TYPICAL RESPONSE

Response to FOXO4-DRI (Hypothesis)

Predisposed to a typical response to FOXO4-DRI

TYPICAL RESPONSE

Response to Humanin (Hypothesis)

Predisposed to a typical response to Humanin

TYPICAL RESPONSE

Response to Small Humanin-Like Peptides (Hypothesis)

Predisposed to a typical response to SHLPs

BETTER RESPONSE

Response to GHK-Cu (Hypothesis)

Predisposed to a better response to GHK-Cu

Your Results in Details

Cognition & Mood

Your brain's ability to think clearly, stay focused, and maintain emotional balance is influenced by a complex interplay of neurotransmitters, neurotrophic factors, and cellular resilience. This section explores peptides that support cognitive performance, memory, and mood regulation — including Selank, Dihexa, Cerebrolysin, Synapsin, and more. These compounds can enhance neuroplasticity, reduce mental fatigue, and help protect neurons from oxidative or stress-related damage. Understanding how they work offers insight into optimizing brain health, emotional well-being, and long-term cognitive vitality.

WORSE RESPONSE

Response to Semax (Hypothesis)

Predisposed to a worse response to Semax

WORSE RESPONSE

Response to Synapsin (Hypothesis)

Predisposed to a worse response to Synapsin

TYPICAL RESPONSE

Response to Selank (Hypothesis)

Predisposed to a typical response to Selank

TYPICAL RESPONSE

Response to Dihexa (Hypothesis)

Predisposed to a typical response to Dihexa

TYPICAL RESPONSE

Response to Cerebrolysin (Hypothesis)

Predisposed to a typical response to Cerebrolysin

TYPICAL RESPONSE

Response to Cyclo (Hypothesis)

Predisposed to a typical response to Cyclo (His-Pro)

TYPICAL RESPONSE

Response to Pinealon (Hypothesis)

Predisposed to a typical response to Pinealon

TYPICAL RESPONSE

Response to DSIP (Hypothesis)

Predisposed to a typical DSIP response

Response To Semax (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Semax.*

The effectiveness of Semax in enhancing cognition and mood can vary based on individual genetic factors that influence neuroplasticity, neurotransmitter regulation, and neuroprotection. Genetic variants that affect BDNF expression, dopamine, serotonin, and other related pathways can modulate how well Semax works for cognitive and emotional enhancement.

BDNF (brain-derived neurotrophic factor) is a component produced mainly in brain cells. It plays many key roles that support your brain's ability to grow and learn. More precisely, BDNF helps stimulate neurogenesis (the production of new nerve cells) and synaptic plasticity (growing new connections between brain cells) [R, R, R, R].

A crucial *BDNF* gene variant is **rs6265**, also known as "**Val66Met**". It may reduce BDNF production, storage, and release in brain cells. As a result, the **"T" ("Met") allele is linked to reduced cognitive function.** Carriers of this allele may respond worse to Semax [R, R, R, R].

The *COMT* gene helps make an enzyme called *catechol-O-methyltransferase* (COMT). The COMT enzyme helps break down chemical messengers in the body. These include dopamine, epinephrine, and norepinephrine [R, R, R].

One common variant of the *COMT* gene, **rs4680**, may affect COMT enzyme activity. Some people call rs4680 the "worrier or warrior" variant. The "G" allele of this variant is linked to a higher COMT enzyme activity, resulting in a faster breakdown of chemical messengers. In individuals with this allele, Semax may have a more immediate but shorter-lasting effect on mood and cognition [R, R].

The *DRD2* gene helps make dopamine D2 receptors. Those are proteins on the surface of brain cells that bind dopamine. The

WORSE RESPONSE

Predisposed to a worse response to Semax based on 14 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
BLMH	rs2129785	TT
BLMH	rs11867581	AA
BDNF	rs6265	TC
COMT	rs4680	AG
ANKK1	rs1800497	GA
MAOA	rs6323	G
MAOA	rs1137070	T
MAOA	rs909525	C
MAOB	rs3027452	G
MAOB	rs2283729	G
MAOB	rs1799836	T
HTR2A	rs6313	GA
RNF180	rs6295	GG
DRD3	rs6280	TT

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

functions of D2 receptors are complex. They depend on the brain region and the position of receptors on brain cells. For the most part, these receptors are *inhibitory,* which means they prevent excessive dopamine release [R].

When it comes to the *DRD2* gene, the most studied variant is **rs1800497**, also known as Taq1A. Interestingly, this variant is found in another gene, *ANKK1*, which controls the activity of *DRD2*. The **"A" ("A1") allele** of this variant is linked to a **30%** lower number of D2 receptors in brain regions responsible for **motivation and reward.** This may result in a reduced response to the dopamine-enhancing effects of Semax [R, R, R].

The *DRD3* gene encodes dopamine receptor D3. While activation of certain types of dopamine receptors has been shown to promote alertness and arousal, preliminary research has reported that dopamine receptor D3 activation may induce sleepiness [R, R, R].

The 'T' allele of **rs6280** (Ser9Gly) may increase the sensitivity or production of dopamine receptor D3, which may result in excessive fatigue when activated by dopamine. On the bright side, carriers of this variant may be more responsive to the dopamine-enhancing effects of Semax [R].

The *MAOA* gene codes for *monoamine oxidase*, an enzyme that helps break down chemical messengers such as dopamine, serotonin, and norepinephrine [R].

There are multiple *MAOA* variants affecting enzyme activity. While low-activity variants lead to increased levels of the monoamine neurotransmitters dopamine, serotonin, and norepinephrine, variants with high activity decrease them. Among them, three of the most well-researched ones are **rs6323**, **rs1137070**, and **rs909525**. Their minor alleles ("G", "T", and "C" respectively) encode MAOA proteins with higher activity, potentially reducing the effects of Semax [R].

The *MAOB* gene codes for monoamine oxidase B, an enzyme that helps break down compounds such as dopamine and phenylethylamine [R].

A higher amount of monoamine oxidase B implies lower monoamine levels (due to increased breakdown), and vice versa. *MAOB* variants with increased activity, which may reduce the effects of Semax, include [R, R]:

- ‘G’ of **rs3027452**
- ‘G’ of **rs2283729**
- ‘T’ of **rs1799836**

The *HTR1A* gene helps produce a serotonin receptor, 5HT1A [R, R].

The most widely investigated *HTR1A* variant is **rs6295**. Its minor ‘C’ allele leads to a decreased overall activity in serotonin neurons. This may reduce the effectiveness of Semax [R, R].

The *HTR2A* gene helps produce another serotonin receptor, 5-HT2A. In the brain, these receptors are concentrated in the prefrontal cortex, amygdala, and hippocampus--areas implicated in learning, memory, and overall cognitive ability [R, R, R, R, R, R, R].

The most widely investigated variant is **rs6313**. Its minor ‘A’ allele increases the number of active receptors. As a result, carriers may respond better to the serotonin-enhancing effects of Semax [R, R].

Finally, 5-HTTLPR is the most well-researched polymorphism of the *SLC6A4* gene, which encodes a serotonin transporter protein. Once serotonin has been released by brain cells, SLC6A4 becomes active and moves the serotonin back into those cells to reduce the length of serotonin signals [R, R, R, R].

The S allele reduces the rate at which serotonin is recycled after a signal, ultimately lowering the circulating levels of this chemical. This may also reduce the effects of Semax. Different combinations of the **rs2129785** and **rs11867581** polymorphisms of this gene can be used to predict the genotype of this variant. While carrying the ‘T’ variant at rs2129785 and ‘A’ at rs11867581 predicts the S allele in 91% of cases, 96% of people carrying ‘T’ at rs2129785 and ‘G’ at rs11867581 and 100% of those with ‘C’ at rs2129785 and ‘A’ at rs11867581 have the L allele [R, R].

Response To Synapsin (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Synapsin.*

Synapsin acts primarily by modulating presynaptic synapsin proteins (SYN1, SYN2, SYN3) and associated pathways, influencing neurotransmitter release and synaptic plasticity. Genetic variation in these proteins, as well as in downstream regulators of synaptic function, may affect responsiveness to Synapsin therapy.

The *SYN1* gene encodes synapsin I, a neuronal phosphoprotein that associates with the cytoplasmic surface of synaptic vesicles and controls neurotransmitter release and brain cell development [R].

The 'G' variant of **rs1142636** (*SYN1*), which possibly affects brain cell development, has been associated with schizophrenia and worse response to antidepressant treatments. Carriers may also respond worse to Synapsin [R, R].

BDNF (brain-derived neurotrophic factor) is a component produced mainly in brain cells. It plays many key roles that support your brain's ability to grow and learn. More precisely, BDNF helps stimulate neurogenesis (the production of new nerve cells) and synaptic plasticity (growing new connections between brain cells) [R, R, R, R].

A crucial *BDNF* gene variant is **rs6265**, also known as "**Val66Met**". It may reduce BDNF production, storage, and release in brain cells. As a result, the **"T" ("Met") allele is linked to reduced cognitive function.** Carriers of this allele may respond worse to Synapsin [R, R, R, R].

Other variants linked to lower BDNF levels and potentially worse response to Synapsin include:

- 'T' of **rs59579819** [R]
- 'A' of **rs80238569** [R]
- 'T' of **rs75945125** [R]

WORSE RESPONSE

Predisposed to a worse response to Synapsin based on 21 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
BDNF	rs59579819	TT
BDNF	rs80238569	AA
BDNF	rs75945125	TT
CAMK2A	rs919741	CC
CAMK2A	rs891935	AA
NUDCD3	rs28505285	GG
NUDCD3	rs76395018	AA
GRIN2B	rs1019385	AA
SYN1	rs1142636	G
BDNF	rs6265	TC
SNAP25	rs3746544	GT
SNAP25	rs363039	GA
SNAP25	rs362987	AC
GRIN2B	rs12822453	GA
GRIN2B	rs1861786	AG
GRIN2B	rs220558	AG
GRIN2B	rs75940780	GG
EMP1	rs7312511	CC
ARSI	rs6880251	CC
GRIN2B	rs2268127	TT
GRIN2B	rs138009610	TT

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

The *SNAP25* gene encodes a protein called 'synaptosome-associated protein 25' that is involved in the release of neurotransmitters from brain cells to support signal transmission (*synapsis*). SNAP25 works by targeting, docking, and fusing synaptic vesicles containing them with the plasma membrane of brain cells for their release, a process called *exocytosis* [R, R].

The following variants, which lower SNAP25 levels, have been associated with mental disorders such as ADHD:

- 'T' of **rs3746544** [R, R, R, R, R]
- 'A' of **rs363039** [R, R, R, R]
- 'A' of **rs362987** [R, R, R]

They may also worsen carriers' response to Synapsin.

The *CAMK2A* and *CAMK2B* genes encode two subunits of the calcium calmodulin-dependent protein kinase CAMK2. This protein phosphorylates synapsins to facilitate vesicle mobilization and neurotransmitter release [R, R].

The following variants have been associated with better cognitive performance and educational attainment, potentially enhancing the effectiveness of Synapsin:

- 'T' of **rs919741** [R]
- 'G' of **rs891935** [R]
- 'C' of **rs6880251** [R]
- 'T' of **rs76395018** [R]

Finally, the *GRIN2B* gene encodes a glutamate receptor of the NMDA type. These receptors are involved in brain development, synaptic plasticity, and cognitive function [R].

Some variants have been associated with worse cognitive performance and educational attainment, which may worsen Synapsin response. They include:

- 'T' of **rs7312511** [R]
- 'A' of **rs1019385** [R]
- 'G' of **rs12822453** [R]
- 'A' of **rs1861786** [R, R]
- 'T' of **rs75940780** [R]
- 'C' of **rs138009610** [R]
- 'A' of **rs220558** [R]
- 'C' of **rs2268127** [R]

Response To Selank (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Selank.*

The effectiveness of Selank in enhancing cognition and mood can vary based on individual genetic factors that influence neuroplasticity, neurotransmitter regulation, and neuroprotection. Genetic variants that affect BDNF expression, dopamine, serotonin, and other related pathways can modulate how well Selank works for cognitive and emotional enhancement.

BDNF (brain-derived neurotrophic factor) is a component produced mainly in brain cells. It plays many key roles that support your brain's ability to grow and learn. More precisely, BDNF helps stimulate neurogenesis (the production of new nerve cells) and synaptic plasticity (growing new connections between brain cells) [R, R, R, R].

A crucial *BDNF* gene variant is **rs6265**, also known as "**Val66Met**". It may reduce BDNF production, storage, and release in brain cells. As a result, the **"T" ("Met") allele is linked to reduced cognitive function.** Carriers of this allele may respond worse to Selank [R, R, R, R].

The GABA-A receptor is the primary target of GABA (gamma-aminobutyric acid) in the brain. Genes encoding different parts of that receptor, such as *GABRA6* and *GABRG2*, can influence its function [R].

The *GABRG2* gene codes for the gamma-aminobutyric acid type A subunit gamma2 (GABRG2) of the GABA-A receptor. The 'T' allele of its **rs211037** polymorphism likely impairs GABA-A receptor function and has been associated with an increased risk of anxiety and seizures [R].

Another gene, *GABRA6*, encodes the alpha6 subunit of this receptor. The 'T' allele of its **rs3219151** polymorphisms, which may also impair GABA-A activity, has been associated with an

TYPICAL RESPONSE

Predisposed to a typical response to Selank based on 9 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
BLMH	rs2129785	TT
BLMH	rs11867581	AA
BDNF	rs6265	TC
COMT	rs4680	AG
ANKK1	rs1800497	GA
MAOA	rs6323	G
MAOA	rs1137070	T
MAOA	rs909525	C
MAOB	rs3027452	G
MAOB	rs2283729	G
MAOB	rs1799836	T
HTR2A	rs6313	GA
GABRB3	rs4906902	GA
RNF180	rs6295	GG
DRD3	rs6280	TT
GABRA6	rs3219151	TT
GABRG2	rs211037	TT

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

increased risk of anxiety, alcohol addiction, and stress-associated suicide [R].

Carriers of these two variants may experience stronger calming effects of Selank.

The *GABRB3* gene encodes the beta-3 subunit of the heteropentameric receptor for the neurotransmitter GABA (gamma-aminobutyric acid) [R].

The best-researched *GABRB3* SNP is **rs4906902**. Its minor 'G' allele reduces *GABRB3* gene expression and has been associated with an increased risk of mental health conditions such as addictions, schizophrenia, or depression. Carriers may benefit more from Selank [R, R].

The *COMT* gene helps make an enzyme called *catechol-O-methyltransferase* (COMT). The COMT enzyme helps break down chemical messengers in the body. These include dopamine, epinephrine, and norepinephrine [R, R, R].

One common variant of the *COMT* gene, **rs4680**, may affect COMT enzyme activity. Some people call rs4680 the "worrier or warrior" variant. The "G" allele of this variant is linked to a higher COMT enzyme activity, resulting in a faster breakdown of chemical messengers. In individuals with this allele, Selank may have a more immediate but shorter-lasting effect on mood and cognition [R, R].

The *DRD2* gene helps make dopamine D2 receptors. Those are proteins on the surface of brain cells that bind dopamine. The functions of D2 receptors are complex. They depend on the brain region and the position of receptors on brain cells. For the most part, these receptors are *inhibitory,* which means they prevent excessive dopamine release [R].

When it comes to the *DRD2* gene, the most studied variant is **rs1800497**, also known as Taq1A. Interestingly, this variant is found in another gene, *ANKK1*, which controls the activity of *DRD2*. The **"A" ("A1") allele** of this variant is linked to a **30%** lower number of D2 receptors in brain regions responsible for **motivation and reward.** This may result in a reduced response to the dopamine-enhancing effects of Selank [R, R, R].

The *DRD3* gene encodes dopamine receptor D3. While activation of certain types of dopamine receptors has been

shown to promote alertness and arousal, preliminary research has reported that dopamine receptor D3 activation may induce sleepiness [R, R, R].

The 'T' allele of **rs6280** (Ser9Gly) may increase the sensitivity or production of dopamine receptor D3, which may result in excessive fatigue when activated by dopamine. On the bright side, carriers of this variant may be more responsive to the dopamine-enhancing effects of Selank [R].

The *MAOA* gene codes for *monoamine oxidase*, an enzyme that helps break down chemical messengers such as dopamine, serotonin, and norepinephrine [R].

There are multiple *MAOA* variants affecting enzyme activity. While low-activity variants lead to increased levels of the monoamine neurotransmitters dopamine, serotonin, and norepinephrine, variants with high activity decrease them. Among them, three of the most well-researched ones are **rs6323**, **rs1137070**, and **rs909525**. Their minor alleles ("G", "T", and "C" respectively) encode MAOA proteins with higher activity, potentially reducing the effects of Selank [R].

The *MAOB* gene codes for monoamine oxidase B, an enzyme that helps break down compounds such as dopamine and phenylethylamine [R].

A higher amount of monoamine oxidase B implies lower monoamine levels (due to increased breakdown), and vice versa. *MAOB* variants with increased activity, which may reduce the effects of Selank, include [R, R]:

- 'G' of **rs3027452**
- 'G' of **rs2283729**
- 'T' of **rs1799836**

The *HTR1A* gene helps produce a serotonin receptor, 5HT1A [R, R].

The most widely investigated *HTR1A* variant is **rs6295**. Its minor 'C' allele leads to a decreased overall activity in serotonin neurons. This may reduce the effectiveness of Selank [R, R].

The *HTR2A* gene helps produce another serotonin receptor, 5-HT2A. In the brain, these receptors are concentrated in the prefrontal cortex, amygdala, and hippocampus--areas implicated in learning, memory, and overall cognitive ability [R, R, R, R, R, R, R].

The most widely investigated variant is **rs6313**. Its minor 'A' allele increases the number of active receptors. As a result, carriers may respond better to the serotonin-enhancing effects of Selank [R, R].

The *TPH2* gene codes for tryptophan hydroxylase 2, an enzyme that helps transform the amino acid tryptophan into the neurotransmitter serotonin in the brain [R].

The **rs4570625** variant has been most widely investigated. Its minor 'T' allele is believed to reduce TPH2 production, resulting in lower serotonin levels, but also increased GABA levels. Based on this, carriers may benefit less from the calming effects of Selank [R, R, R, R].

Finally, 5-HTTLPR is the most well-researched polymorphism of the *SLC6A4* gene, which encodes a serotonin transporter protein. Once serotonin has been released by brain cells, SLC6A4 becomes active and moves the serotonin back into those cells to reduce the length of serotonin signals [R, R, R, R].

The S allele reduces the rate at which serotonin is recycled after a signal, ultimately lowering the circulating levels of this chemical. This may also reduce the effects of Selank. Different combinations of the **rs2129785** and **rs11867581** polymorphisms of this gene can be used to predict the genotype of this variant. While carrying the 'T' variant at rs2129785 and 'A' at rs11867581 predicts the S allele in 91% of cases, 96% of people carrying 'T' at rs2129785 and 'G' at rs11867581, and 100% of those with 'C' at rs2129785 and 'A' at rs11867581 have the L allele [R, R].

Response To Dihexa (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Dihexa.*

Dihexa primarily acts through HGF/c-Met signaling, which promotes synaptic development and neuronal growth. While no direct human pharmacogenomic studies exist, responsiveness may vary depending on variants influencing HGF production, MET receptor function, or downstream neurotrophic signaling pathways.

The *MET* gene encodes the hepatocyte growth factor receptor, a tyrosine kinase receptor essential for embryonic development, organogenesis, and wound healing by binding to HGF/SF and its splicing forms NK1 and NK2. Mutations of this gene are associated with different types of cancer [R, R].

Variants influencing *MET* expression may modulate the strength of Dihexa's effect. Key variants potentially increasing response include:

- 'G' of **rs112270031** [R]
- 'C' of **rs1858830** [R]
- 'A' of **rs41747** [R]
- 'A' of **rs55985569** [R]

The *HGF* gene encodes the hepatocyte growth factor, a protein secreted by mesenchymal cells that binds the hepatocyte growth factor receptor to regulate cell growth, cell motility, and morphogenesis in numerous cell and tissue types, especially epithelial and endothelial tissues [R].

Variants influencing HGF expression may modulate the strength of Dihexa's effect. Key variants potentially increasing response include:

- 'C' of **rs2074725** [R]
- 'T' of **rs5745652** [R]
- 'C' of **rs5745687** [R]
- 'T' of **rs984534** [R]
- 'C' of **rs7810969** [R]

TYPICAL RESPONSE

Predisposed to a typical response to Dihexa based on 12 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MET	rs112270031	AA
MET	rs41747	TT
MET	rs55985569	GG
HGF	rs150267054	GG
MET	rs1858830	GC
HGF	rs10248271	TG
BDNF	rs6265	TC
NGF	rs6330	GA
HGF	rs984534	TT
HGF	rs7810969	CC
HGF	rs5745695	AA
HGF	rs5745687	CC
HGF	rs5745652	GG
HGF	rs2074725	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

- 'A' of **rs150267054** [R]
- 'A' of **rs5745695** [R]
- 'T' of **rs10248271** [R]

BDNF (brain-derived neurotrophic factor) is a component produced mainly in brain cells. It plays many key roles that support your brain's ability to grow and learn. More precisely, BDNF helps stimulate neurogenesis (the production of new nerve cells) and synaptic plasticity (growing new connections between brain cells) [R, R, R, R].

A crucial *BDNF* gene variant is **rs6265**, also known as "**Val66Met**". It may reduce BDNF production, storage, and release in brain cells. As a result, the **"T" ("Met") allele is linked to reduced cognitive function.** Carriers of this allele may respond worse to Dihexa [R, R, R, R].

Finally, the *NGF* gene encodes a protein called nerve growth factor. As its name suggests, NGF may promote the growth, maintenance, and survival of neurons and axons. It's also thought to help repair the myelin sheath, which is the insulating coating around the axons. Mutations in this gene are relatively common in inherited diseases of the nervous system [R, R].

Variants in *NGF* may modulate neurotrophic effects indirectly, potentially impacting Dihexa's effectiveness. For instance, the 'A' allele of **rs6330** (Ala35Val) has been linked to higher NGF levels and may enhance the effects of Dihexa [R].

Response To Cerebrolysin (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. The only genetic variant found to modify the effectiveness of Cerebrolysin is APOE ε4, and its effects are mixed.*

Cerebrolysin's effects are mediated through pathways involved in neurotrophic signaling, neuronal survival, and synaptic plasticity, including BDNF, NGF, and other neurotrophin-related pathways. While no direct human pharmacogenomic studies exist, genetic variants may influence individual responsiveness by affecting these neurotrophic pathways or receptor sensitivity.

BDNF (brain-derived neurotrophic factor) is a component produced mainly in brain cells. It plays many key roles that support your brain's ability to grow and learn. More precisely, BDNF helps stimulate neurogenesis (the production of new nerve cells) and synaptic plasticity (growing new connections between brain cells) [R, R, R, R].

A crucial *BDNF* gene variant is **rs6265**, also known as "**Val66Met**". It may reduce BDNF production, storage, and release in brain cells. As a result, the **"T" ("Met") allele is linked to reduced cognitive function.** Carriers of this allele may respond worse to Cerebrolysin [R, R, R, R].

Other variants linked to lower BDNF levels and potentially worse response to Cerebrolysin include:

- 'T' of **rs59579819** [R]
- 'A' of **rs80238569** [R]
- 'T' of **rs75945125** [R]

The *NGF* gene encodes a protein called nerve growth factor. As its name suggests, NGF may promote the growth, maintenance, and survival of neurons and axons. It's also thought to help repair the myelin sheath, which is the insulating coating around the axons. Mutations in this gene are relatively common in inherited diseases of the nervous system [R, R].

Variants in *NGF* may modulate neurotrophic effects indirectly, potentially impacting Cerebrolysin's effectiveness. For instance,

TYPICAL RESPONSE

Predisposed to a typical response to Cerebrolysin based on 11 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NTRK2	rs77562929	GG
APOE	rs7412	CC
BDNF	rs59579819	TT
BDNF	rs80238569	AA
BDNF	rs75945125	TT
NTRK2	rs4457413	CT
BDNF	rs6265	TC
NGF	rs6330	GA
NTRK1	rs6336	CC
APOE	rs429358	TT
NTRK2	rs1624327	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

the ‘A’ allele of **rs6330** (Ala35Val) has been linked to higher NGF levels and may enhance the effects of Cerebrolysin [R].

The *NTRK1* and *NTRK2* genes encode receptors for NGF and BDNF, respectively, mediating downstream signaling for neuronal growth and synaptic function [R, R].

Variants in these receptors potentially increasing responsiveness to Cerebrolysin include:

- ‘C’ of **rs6336** [R]
- ‘G’ of **rs1624327** [R]
- ‘T’ of **rs4457413** [R]
- ‘T’ of **rs77562929** [R]

The *APOE* gene makes Apolipoprotein E (ApoE), a protein critical for the transport and metabolism of fats throughout the body. Despite the important role of ApoE in heart health, most of the research has been focused on the brain. APOE is the major gene affecting the risk of late-onset (after the age of 65) Alzheimer’s disease [R, R, R, R, R].

There are three major forms (variants) of the *APOE* gene. These are called ε2, ε3, and ε4. ε4 has been linked to a higher risk of Alzheimer’s disease and neurodegeneration. In one study, Alzheimer's patients without this variant were three times more likely to respond to treatment. However, Cerebrolysin was more effective at increasing BDNF levels in ε4 carriers in another study [R, R, R, R].

Response To Cyclo (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Cyclo (His-Pro).*

Because Cyclo (His-Pro) influences monoamine neurotransmission, oxidative balance, and inflammatory signaling, genetic variants in related pathways may alter individual sensitivity or therapeutic outcomes.

The *MAOA* gene codes for *monoamine oxidase*, an enzyme that helps break down the chemical messengers dopamine, serotonin, and norepinephrine (noradrenaline) [R].

There are multiple *MAOA* variants affecting enzyme activity. While low-activity variants lead to increased levels of the monoamine neurotransmitters dopamine, serotonin, and norepinephrine, variants with high activity decrease them. Among them, three of the most well-researched ones are **rs6323**, **rs1137070**, and **rs909525**. Their minor alleles ("G", "T", and "C" respectively) encode MAOA proteins with higher activity. Due to its association with lower levels of these neurotransmitters, these variants may reduce the effects of Cyclo (His-Pro) [R].

The *MAOB* gene codes for monoamine oxidase B, an enzyme that helps break down compounds such as dopamine and phenylethylamine [R].

A higher amount of monoamine oxidase B implies lower monoamine levels (due to increased breakdown), and vice versa. *MAOB* variants with increased activity, which may be linked to a worse response to Cyclo (His-Pro), include 'G' of **rs3027452** and 'G' of **rs2283729** [R, R].

The *SLC6A3* gene encodes the dopamine transporter DAT1. This protein is found on the membrane of brain cells, where it transports the neurotransmitter dopamine into the cell [R].

TYPICAL RESPONSE

Predisposed to a typical response to Cyclo (His-Pro) based on 9 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC6A3	rs6347	TT
CLPTM1L	rs11564750	GG
LPCAT1	rs27072	CC
BLMH	rs2129785	TT
BLMH	rs11867581	AA
IL6	rs1800795	GG
MAOB	rs3027452	G
MAOB	rs2283729	G
MAOA	rs6323	G
MAOA	rs1137070	T
MAOA	rs909525	C
SLC6A3	rs2550948	CT
ANKK1	rs1800497	GA
HTR2A	rs6313	GA
HTR2A	rs6311	CT
HTR2C	rs3813929	C
HTR2C	rs3813928	G
COMT	rs4680	AG
IL6	rs1524107	CT
TNF	rs1799724	TC
ESD	rs7984966	TT
STEAP1B	rs75045751	GG
ESD	rs7322347	TT
HTR2C	rs6318	G
RNF180	rs6295	GG
HTR2C	rs498207	A
HTR2C	rs498177	G
SOD2	rs4880	GG
TPH2	rs4570625	GG
SLC6A3	rs27048	TT

Several *SLC6A3* variants have been associated with an increased risk of ADHD, as well as increased impulsivity and inattention in individuals with ADHD. These include [R, R, R, R]:

- 'T' of **rs6347**
- 'G' of **rs11564750**
- 'C' of **rs27072**
- 'T' of **rs2550948**
- 'C' of **rs27048**

While the mechanism behind this association is not clear, these variants may increase the density of the dopamine transporter in certain regions of the brain. This would decrease the amount of dopamine available in these brain regions. Based on this mechanism, carriers may also respond worse to Cyclo (His-Pro) [R, R].

SLC6A4 encodes a serotonin transporter protein. Once serotonin has been released by brain cells, SLC6A4 becomes active and moves the serotonin back into those cells. In this way, SLC6A4 reduces the length of serotonin signals [R, R].

5HTTLPR is the best-researched polymorphism of the *SLC6A4* gene, which encodes a serotonin transporter protein [R, R, R, R].

Different combinations of the **rs2129785** and **rs11867581** polymorphisms of this gene are usually inherited together with the S and L alleles and can be used to predict the genotype of this variant. While carrying the 'T' variant at rs2129785 and 'A' at rs11867581 predicts the S allele in 91% of cases, 96% of people carrying 'T' at rs2129785 and 'G' at rs11867581 and 100% of those with 'C' at rs2129785 and 'A' at rs11867581 have the L allele [R].

The S allele reduces the rate at which serotonin is recycled after a signal, ultimately lowering the circulating levels of this chemical. Carriers may respond worse to Cyclo (His-Pro) [R].

Two other *SLC6A4* variants, 'G' at **rs2066713** and 'T' at **rs140701**, have been associated with higher rates of chronic fatigue syndrome. These variants may reduce SLC6A4 activity. As a result, they may boost the effects of Cyclo (His-Pro) [R, R].

The *DRD2* gene helps make dopamine D2 receptors. Those are proteins on the surface of brain cells that bind dopamine. The functions of D2 receptors are complex. They depend on the brain region and the position of receptors on brain cells. For the

GENE	SNP	GENOTYPE
BLMH	rs2066713	GG
TNF	rs1800629	GG
TNF	rs1799964	TT
BLMH	rs140701	TT

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

most part, these receptors are *inhibitory,* which means they prevent excessive dopamine release [R].

When it comes to the *DRD2* gene, the most studied variant is **rs1800497**, also known as Taq1A. This allele is linked to a **30%** lower number of D2 receptors in brain regions responsible for **motivation and reward**. People with this allele may not get enough pleasure from ordinary rewarding activities. Due to its decreased dopamine signaling, carriers may respond worse to Cyclo (His-Pro) [R, R, R].

The *TPH2* gene codes for tryptophan hydroxylase 2, an enzyme that helps transform the amino acid tryptophan into the neurotransmitter serotonin in the brain [R].

The **rs4570625** variant has been most widely investigated. Its minor 'T' allele is believed to reduce TPH2 production, resulting in lower serotonin levels. Carriers of this variant may respond worse to Cyclo (His-Pro) supplementation [R, R, R, R].

Several genes encode serotonin receptors [R, R, R, R].

The most widely investigated *HTR1A* variant is **rs6295**. Its minor 'C' allele leads to higher expression of 5HT1A autoreceptors, a lower expression of postsynaptic receptors, and decreased overall activity in serotonin neurons. In line with this, it may reduce the effects of Cyclo (His-Pro) [R, R].

Because the receptor encoded by the *HTR2A* gene (5HT2A) enhances serotonin signaling, variants increasing the number of receptors may boost the effects of Cyclo (His-Pro). Some of these variants include:

- 'A' of **rs6313** [R, R]
- 'T' of **rs6311** [R, R]
- 'T' of **rs7322347** [R, R]
- 'T' of **rs7984966** [R, R]

Several *HTR2C* variants have been found to increase 5-HT2C activity and decrease BMI, appetite, and antipsychotic-induced weight gain:

- 'T' of **rs3813929** [R]
- 'A' at **rs3813928** [R]
- 'G' at **rs6318** [R]
- 'A' at **rs498207** [R]
- 'G' at **rs498177** [R]

On the bright side, carriers may respond better to Cyclo (His-Pro).

The *COMT* gene helps make an enzyme called *catechol-O-methyltransferase* (COMT). The COMT enzyme helps break down chemical messengers such as dopamine, epinephrine (adrenaline) and norepinephrine in the body [R, R, R].

One common variant of the *COMT* gene, **rs4680**, may affect COMT enzyme activity. Some people call rs4680 the “worrier or warrior” variant. The “G” allele of this variant is linked to a higher COMT enzyme activity. As a result, carriers may show a reduced response to Cyclo (His-Pro) [R, R].

Interleukin-6 (IL-6) is a cytokine encoded by the *IL6* gene that has either proinflammatory or anti-inflammatory properties, depending on the circumstances and the tissue that secretes it. It has complicated mechanisms of effect and interacts with many other cytokines and inflammatory signals [R, R].

By far, the best-researched *IL6* polymorphism is **rs1800795** (also known as the “-174G>C” polymorphism). The major 'G' allele of this SNP has been linked with higher levels of IL-6, while the 'C' allele has been associated with lower IL-6 levels. The major ‘G’ allele may lead to a more pronounced neuroinflammatory response, potentially leading to a weaker neuroprotective response to Cyclo (His-Pro) [R].

Other variants of this gene linked to greater inflammation include:

- ‘C’ of **rs1524107** [R]
- ‘A’ of **rs75045751** [R]

The *TNF* gene encodes a protein called tumor necrosis factor-alpha (TNF-alpha or cachexin). TNF-alpha plays a central role in the immune response and inflammation, and it has been implicated in a wide variety of inflammatory disorders [R].

The **rs1800629** polymorphism (also known as *TNF* -308) is one of the most researched SNPs in the *TNF* gene. **The ‘A’ allele is associated with 6-7 times higher levels of TNF-alpha.** This may result in greater inflammation and worse response to Cyclo (His-Pro) [R].

Other variants that may increase TNF levels include:

- ‘C’ of **rs1799964** [R]

- ‘C’ of **rs1799724** [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 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].

The *SOD2* gene has many described polymorphisms. Among them, **rs4880** has received the spotlight in SOD2 research. Its minor allele ‘G’ is associated with decreased activity and worse protection against oxidative stress. Carriers may benefit more from the antioxidant effects of Cyclo (His-Pro) [R].

Response To Pinealon (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Pinealon.*

Genetic variation in neuronal plasticity, oxidative stress, inflammation, circadian, and apoptotic pathways may influence individual response to Pinealon, affecting cognition, stress tolerance, and neuroprotection.

BDNF (brain-derived neurotrophic factor) is a component produced mainly in brain cells. It plays many key roles that support your brain's ability to grow and learn. More precisely, BDNF helps stimulate neurogenesis (the production of new nerve cells) and synaptic plasticity (growing new connections between brain cells) [R, R, R, R].

A crucial *BDNF* gene variant is **rs6265**, also known as "**Val66Met**". It may reduce BDNF production, storage, and release in brain cells. As a result, the **"T" ("Met") allele is linked to reduced cognitive function.** Carriers of this allele may respond worse to Pinealon [R, R, R, R].

Other variants linked to lower BDNF levels and potentially worse response to Pinealon include:

- 'T' of **rs59579819** [R]
- 'A' of **rs80238569** [R]
- 'T' of **rs75945125** [R]

The *COMT* gene helps make an enzyme called *catechol-O-methyltransferase* (COMT). The COMT enzyme helps break down chemical messengers such as dopamine, epinephrine (adrenaline) and norepinephrine in the body [R, R, R].

One common variant of the *COMT* gene, **rs4680**, may affect COMT enzyme activity. Some people call rs4680 the "worrier or warrior" variant. The "G" allele of this variant is linked to a higher COMT enzyme activity. As a result, carriers may show a reduced response to Cyclo (His-Pro) [R, R].

TYPICAL RESPONSE

Predisposed to a typical response to Pinealon based on 9 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
BDNF	rs59579819	TT
BDNF	rs80238569	AA
BDNF	rs75945125	TT
APOE	rs7412	CC
NFE2L2	rs6721961	GG
AGPS	rs2364723	GG
AGPS	rs10497511	AA
HNRNPA3	rs13001694	GG
AGPS	rs1962142	GG
SOD2	rs4880	GG
GPX1	rs1050450	GG
STEAP1B	rs75045751	GG
TNF	rs1800629	GG
TNF	rs1799964	TT
BCL2	rs4987856	CC
CACNA1C	rs2299661	GG
BDNF	rs6265	TC
COMT	rs4680	AG
CAT	rs1001179	TC
CAT	rs7943316	TA
IL6	rs1524107	CT
TNF	rs1799724	TC
CLOCK	rs1801260	AG
CAT	rs769217	CC
NFE2L2	rs6726395	AA
APOE	rs429358	TT
NFE2L2	rs35652124	TT
CACNA1C	rs216008	CC
NFE2L2	rs1806649	CC
IL6	rs1800795	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

The *APOE* gene makes Apolipoprotein E (ApoE), a protein critical for the transport and metabolism of fats throughout the body. Despite the important role of ApoE in heart health, most of the research has been focused on the brain. APOE is the major gene affecting the risk of late-onset (after the age of 65) Alzheimer's disease [R, R, R, R, R].

There are three major forms (variants) of the *APOE* gene. These are called ε2, ε3, and ε4. ε4 has been linked to a higher risk of Alzheimer's disease and neurodegeneration. Carriers of this variant may show greater neuroprotective effects from Pinealon [R, R].

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].

Research has identified several variants in the NFE2L2 gene that can reduce the expression and activity of NRF2. Researchers have claimed that reduced NRF2 impairs the body's ability to detox and defend itself from oxidative stress. Some of these variants, which may be linked to enhanced benefits from Pinealon, include [R, R]:

- 'T' of **rs35652124**
- 'T' of **rs6721961**
- 'A' of **rs6726395**
- 'C' of **rs2364723**
- 'G' of **rs10497511**
- 'A' of **rs13001694**
- 'C' of **rs1806649**
- 'A' of **rs1962142**

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 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].

The *SOD2* gene has many described polymorphisms. Among them, **rs4880** has received the spotlight in SOD2 research. Its minor allele 'G' is associated with decreased activity and worse protection against oxidative stress. Carriers may respond better to Pinealon's antioxidant and cytoprotective actions [R].

The *CAT* gene encodes subunits of catalase, a key antioxidant enzyme in the body's defense against oxidative stress. Four identical subunits, each attached to an iron-containing molecule called a heme group, form the functional catalase enzyme [R].

The best-characterized *CAT* polymorphism is **rs1001179**, commonly referred to as -262G>A. Its minor 'T' allele may increase *CAT* expression but decrease it in people with conditions such as type 2 diabetes or chronic hepatitis C. Other well-known CAT variants with increased activity include 'T' of **rs769217** and 'A' of **rs7943316.** They may decrease the antioxidant effects of Pinealon [R, R, R, R, R].

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].

One study found a direct link between a common *GPX1* variant and human longevity. The heterozygous **genotype 'AG' at rs1050450 was significantly more common in the very elderly** than in the general population. Other studies have strongly suggested that the **'G' allele at rs1050450 confers higher GPx activity**. Carriers may benefit less from Pinealon [R, R, R].

Interleukin-6 (IL-6) is a cytokine encoded by the *IL6* gene that has either proinflammatory or anti-inflammatory properties, depending on the circumstances and the tissue that secretes it. It has complicated mechanisms of effect and interacts with many other cytokines and inflammatory signals [R, R].

By far, the best-researched *IL6* polymorphism is **rs1800795** (also known as the "-174G>C" polymorphism). The major 'G' allele of this SNP has been linked with higher levels of IL-6, while the 'C' allele has been associated with lower IL-6 levels. The major 'G' allele may lead to a more pronounced neuroinflammatory response. Carriers may benefit more from Pinealon [R].

Other variants of this gene linked to greater inflammation include:

- 'C' of **rs1524107** [R]
- 'A' of **rs75045751** [R]

The *TNF* gene encodes a protein called tumor necrosis factor-alpha (TNF-alpha or cachexin). TNF-alpha plays a central role in

the immune response and inflammation, and it has been implicated in a wide variety of inflammatory disorders [R].

The **rs1800629** polymorphism (also known as *TNF* -308) is one of the most researched SNPs in the *TNF* gene. **The 'A' allele is associated with 6-7 times higher levels of TNF-alpha.** This may result in better anti-inflammatory effect from Pinealon [R].

Other variants that may increase TNF levels include:

- 'C' of **rs1799964** [R]
- 'C' of **rs1799724** [R]

The *CLOCK* ('Circadian Locomotor Output Cycles Kaput') gene is a core component of the biological clock. It is one of the main genes responsible for human daily rhythms, also known as circadian rhythms [R].

The **rs1801260** polymorphism is the most studied SNP in the *CLOCK* gene. Its minor 'G' allele increases CLOCK activity and has been associated with abnormal circadian rhythms and shorter sleep duration. Carriers may benefit more from Pinealon [R].

The *MTNR1B* gene provides instructions for making a receptor that responds to melatonin, a hormone that helps regulate our internal body clock, also known as the circadian rhythm. This rhythm controls our sleep-wake cycle and many other processes, including how our bodies manage blood sugar. The minor "G" allele of **rs10830963** increases the expression of melatonin receptors in pancreatic beta cells. These beta cells release insulin, and when they have more melatonin receptors, they become more sensitive to melatonin's signals. Carriers may be more sensitive to the effects of Pinealon on circadian rhythms [R, R].

The *BCL2* gene encodes a protein that blocks the death of certain cells, such as lymphocytes [R].

The 'T' allele of **rs4987856** has been associated with lower BCL2 protein levels and higher stress susceptibility; Pinealon's anti-apoptotic signaling may be particularly helpful in these individuals [R].

Finally, the *CACNA1C* gene codes for part of a calcium channel ($Ca_V1.2$) in the brain that plays a role in plasticity [R].

The major alleles ‘C’ of **rs216008** and ‘C’ of **rs2299661** have been associated with a better response to calcium channel blocker monotherapy, potentially increasing the beneficial effects of Pinealon [R, R].

Response To DSIP (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of DSIP.*

The effectiveness of DSIP may be influenced by genetic variants that regulate sleep, stress response, and hormonal pathways. Variants in genes associated with the hypothalamic-pituitary-adrenal (HPA) axis, sleep regulation, and neuropeptide receptors can impact individual responses.

The *ADA* gene encodes the adenosine deaminase enzyme, one of the two enzymes that break down adenosine. Adenosine is an endogenous nucleoside found in every cell of the body. One of its key roles is to control the sleep-wake cycle by promoting sleepiness and deep sleep. ADA is activated when adenosine levels become excessive and converts it to inosine, which signals to the body to stop producing adenosine [R, R].

The 'T' allele of **rs73598374** encodes a protein with an amino acid substitution resulting in a 35% reduced ADA activity. As a result, carriers have higher adenosine levels and less inosine. Carriers may respond better to DSIP [R].

The *CRHR1* gene encodes a receptor for CRH, the first hormone of the HPA axis. Stress activates the HPA axis, resulting in cortisol release. Hence, CRH receptors play a significant role in stress response and related disorders. Contrary to CRHR2, this receptor promotes anxiety, arousal, and depression upon activation [R, R].

Variants increasing CRHR1 expression or activity have been associated with stress and anxiety, and may decrease the effectiveness of DSIP treatments. These include [R, R, R, R, R]:

- 'A' of **rs12938031**
- 'C' of **rs4792887**
- 'G' of **rs12944712**
- 'G' of **rs17689882**
- 'G' of **rs110402**
- 'G' of **rs242924**

TYPICAL RESPONSE

Predisposed to a typical DSIP response based on 18 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ARHGAP27	rs4792887	CC
MAPT	rs12944712	GG
CRHR1	rs17689882	GG
CRHR2	rs2267715	AA
CRHR2	rs2190242	AA
NR3C1	rs2918419	TT
NR3C1	rs6196	AA
ADA	rs73598374	TC
MAPT	rs12938031	GA
NR3C1	rs852977	GA
NR3C1	rs1866388	GA
NR3C1	rs6188	AC
CLOCK	rs1801260	AG
NR3C1	rs41423247	GG
MAPT	rs242924	TT
BLMH	rs2066713	GG
BLMH	rs140701	TT
MAPT	rs110402	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

The *CRHR2* gene encodes another receptor for CHR. Contrary to CRHR1, the activation of CRHR2 receptors reduces anxiety, arousal, and depression [R, R, R].

A study of 491 veterans exposed to trauma and their partners associated the 'A' allele of **rs2267715** and **rs2190242** with an increased risk of PTSD in women. The study speculated that both variants may decrease *CRHR2* activity, thereby stimulating the stress response. Based on this, they may decrease the effectiveness of DSIP [R].

The *NR3C1* gene codes for the glucocorticoid receptor. Upon activation by glucocorticoids (such as the primary stress hormone cortisol), the glucocorticoid receptor is able to regulate the production of stress-related, inflammatory proteins [R].

Variants that lower the sensitivity or activity of the glucocorticoid receptor, thus resulting in dysregulated HPA axis activity, excess cortisol release, and inflammation, have been associated with chronic fatigue syndrome and may worsen response to DSIP. They include:

- 'A' at **rs852977** [R, R, R]
- 'A' at **rs1866388** [R, R, R]
- 'C' at **rs6188** [R, R, R]
- 'T' at **rs2918419** [R, R, R]
- 'A' at **rs6196** [R, R, R]
- 'C' at **rs41423247** [R, R]

The *CLOCK* ('Circadian Locomotor Output Cycles Kaput') gene is a core component of the biological clock. It is one of the main genes responsible for human daily rhythms, also known as circadian rhythms [R].

The **rs1801260** polymorphism is the most studied SNP in the *CLOCK* gene. Its minor 'G' allele increases CLOCK activity and has been associated with abnormal circadian rhythms and shorter sleep duration. Carriers may respond worse to DSIP [R].

SLC6A4 encodes a serotonin transporter protein. Once serotonin has been released by brain cells, SLC6A4 becomes active and moves the serotonin back into those cells. In this way, SLC6A4 reduces the length of serotonin signals [R, R].

Two *SLC6A4* variants, 'G' at **rs2066713** and 'T' at **rs140701**, have been associated with higher rates of chronic fatigue syndrome and reduced SLC6A4 activity. These variants may enhance DSIP's calming and sleep-promoting effects [R].

Muscle Growth & Tissue Repair

Tissue repair, exercise recovery, and muscle growth depend on the coordination of growth factors, cellular signaling, and inflammation control. This section focuses on peptides that enhance regeneration, strength, and recovery — including BPC-157, TB-500, Sermorelin, CJC-1295, and more. These peptides can stimulate collagen production, improve muscle fiber repair, and promote growth hormone release, helping the body restore balance after stress or injury. Understanding their mechanisms can provide insight into optimizing recovery, performance, and long-term musculoskeletal health.

TYPICAL RESPONSE

Response to PEG-MGF (Hypothesis)

Predisposed to a typical response to PEG-MGF

TYPICAL RESPONSE

Response to Follistatin-344 (Hypothesis)

Predisposed to a typical response to Follistatin-344

TYPICAL RESPONSE

Response to GHRP-2 (Hypothesis)

Predisposed to a typical response to GHRP-2

TYPICAL RESPONSE

Response to GHRP-6 (Hypothesis)

Predisposed to a typical response to GHRP-6

TYPICAL RESPONSE

Response to CJC-1295 (Hypothesis)

Predisposed to a typical response to CJC-1295

TYPICAL RESPONSE

Response to Thymosin Beta-4 (Hypothesis)

Predisposed to a typical response to Thymosin beta-1

BETTER RESPONSE

Response to BPC-157 (Hypothesis)

Predisposed to a better BPC-157 response

BETTER RESPONSE

Response to TB-500 (Hypothesis)

Predisposed to a better response to TB-500

BETTER RESPONSE

Response to Ipamorelin (Hypothesis)

Predisposed to a better response to Ipamorelin

BETTER RESPONSE

Response to Sermorelin (Hypothesis)

Predisposed to a better response to Sermorelin

Response To PEG-MGF (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of PEG-MGF.*

The response to PEG-MGF may be influenced by genetic variants in genes that regulate IGF-1 production, receptor sensitivity, and muscle regeneration pathways. Variations in these genes can modify peptide efficacy, recovery rates, and muscle growth potential.

The *IGF1* gene encodes insulin-like growth factor 1 (IGF-1), a hormone that works with growth hormone to help cells multiply and regenerate.

The following variants, associated with higher IGF-1 levels, may enhance the effectiveness of PEG-MGF:

- 'T' of **rs6214** [R]
- 'A' of **rs35767** [R]
- 'C' of **rs1019731** [R]
- 'C' of **rs11111270** [R]
- 'G' of **rs11111274** [R]
- 'C' of **rs1546155** [R]
- 'A' of **rs1457596** [R]
- 'A' of **rs78929770** [R]
- 'C' of **rs703588** [R]
- 'T' of **rs978458** [R, R]

The *IGF1R* gene codes for a protein called the IGF1 receptor, which plays an important role in the body's growth and development by acting as a receptor for IGF-1 [R].

Variants resulting in higher IGF1R levels or enhanced receptor sensitivity and signal transduction efficiency may improve response to PEG-MGF. Some of them include:

- 'C' of **rs2684777** [R]
- 'G' of **rs2229765** [R]
- 'A' of **rs34516635** [R]

TYPICAL RESPONSE

Predisposed to a typical response to PEG-MGF based on 15 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IGF1	rs6214	CC
IGF1	rs35767	GG
IGF1	rs1457596	GG
WASHC3	rs78929770	CC
IGF1	rs703588	TT
IGF1R	rs2684777	TT
IGF1R	rs2229765	AA
IGF1R	rs34516635	GG
IGF1	rs1546155	CT
GHR	rs6873545	CT
ACTN3	rs1815739	TC
WASHC3	rs978458	TT
WASHC3	rs11111274	GG
IGF1	rs11111270	CC
IGF1	rs1019731	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

The *GHR* gene encodes the growth hormone receptor, found in the outer membrane of cells and most abundant in the liver [R].

By far, the most widely-researched *GHR* polymorphism consists of the presence or absence of a region called the 'exon 3'. The isoform lacking this region (deficient or d3-GHR) shows enhanced growth hormone signaling compared to the from including it (full-length or fl-GHR). The **rs6873545** SNP serves as a marker for this polymorphism, with the minor 'C' allele corresponding to d3-GHR. Based on this mechanism, carriers of this allele may respond better to PEG-MGF [R].

Finally, the *ACTN3* gene encodes a structural protein of fast-twitch muscle fibers called alpha-actinin 3. ACTN3 is well-known to affect athletic performance. Oddly, about 18% of all people are completely deficient in this protein. People without ACTN3 are less likely to excel in sports, especially in power sports [R, R, R, R].

The best-researched *ACTN3* polymorphism is **rs1815739**, also known as R577X. This variant determines whether people produce the ACTN3 protein or not. The 'T' allele introduces a premature stop codon that prevents the protein from being produced. Carriers of this allele may show reduced hypertrophic response to PEG-MGF [R, R, R].

Response To Follistatin-344 (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Follistatin-344.*

Genetic variation in muscle growth, myostatin, and regeneration pathways may influence individual response to Follistatin-344 in terms of muscle gain, repair, and metabolic effects.

The *FST* gene encodes the follistatin protein, a glycoprotein that regulates the availability of certain growth factors in the body and inhibits FSH release. The single FST gene encodes two isoforms: FST317 and FST344 [R].

The following *FST* variants have been associated with lower FST levels and lower baseline muscle growth. Carriers may benefit more from exogenous Follistatin-344 administration:

- 'C' of **rs1469101** [R]
- 'A' of **rs62370480** [R]
- 'G' of **rs3797297** [R]
- 'G' of **rs121909305** [R]

The *MSTN* gene encodes myostatin, a protein of the transforming growth factor beta superfamily that limits muscle growth [R, R].

Two variants, 'C' of **rs1805086** and 'A' of **rs72909336**, have been associated with lower MTN levels and greater muscle mass. Carriers of these variants may benefit less from Follistatin-344 administration [R, R].

The *ACVR2B* gene encodes the activin A receptor type IIB, a transmembrane protein that acts as a high-affinity receptor for activins and other growth factors like myostatin and GDF11 [R].

The 'A' allele of **rs2276541** has been associated with greater lean muscle mass, suggesting enhanced function of this receptor. This variant may also boost Follistatin-344's effects on muscle growth [R].

TYPICAL RESPONSE

Predisposed to a typical response to Follistatin-344 based on 9 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
FST	rs1469101	TT
FST	rs62370480	GA
ACVR2B	rs2276541	AG
IGF1	rs6214	CC
IGF1	rs5742692	AA
ASCL1	rs56006730	GG
WASHC3	rs78929770	CC
IGF1	rs855203	AA
WASHC3	rs5742704	TT
TMEM91	rs12461895	AA
TGFB1	rs73045269	CC
IL6	rs1800795	GG
IGF1	rs833718	GA
PPARGC1A	rs8192678	TC
COL1A1	rs1800012	AC
IL6	rs1524107	CT
ASNSD1	rs72909336	GG
FST	rs3797297	GG
MSTN	rs1805086	TT
MYO1A	rs121909305	GG
WASHC3	rs978458	TT
STEAP1B	rs75045751	GG
WASHC3	rs6219	TT
IGF1	rs6218	AA
TGFB1	rs1800472	GG
ASCL1	rs12830406	AA
PAH	rs10507151	GG
IGF1	rs1019731	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

The *TGFB1* gene codes for transforming growth factor beta 1 (TGF-beta1). TGF-beta1 is an immune messenger (cytokine) that plays a role in many cellular processes, including cell growth & division, specialization, movement, and death. By doing so, it controls bone, cartilage, and blood vessel formation, muscle and fat development, wound healing, and inflammation [R].

Certain alleles linked to higher TGFB1 expression may amplify tissue regeneration with Follistatin-344. These include:

- 'C' of **rs12461895** [R]
- 'G' of **rs1800472** [R]
- 'T' of **rs73045269** [R]

The *IGF1* gene encodes insulin-like growth factor 1, a protein that has similar functions to insulin and is involved in controlling growth and development in an organism. IGF1 is produced in the liver in response to growth hormone [R].

Variants in *IGF1* can affect IGF-1 levels, thereby influencing the therapeutic outcomes of Follistatin-344. Those that may lead to higher response to this peptide include:

- 'T' of **rs6214** [R]
- 'C' of **rs1019731** [R]
- 'G' of **rs5742692** [R]
- 'A' of **rs56006730** [R]
- 'T' of **rs978458** [R]
- 'A' of **rs78929770** [R]
- 'C' of **rs855203** [R]
- 'G' of **rs833718** [R]
- 'A' of **rs12830406** [R]
- 'G' of **rs10507151** [R]
- 'T' of **rs6219** [R]
- 'A' of **rs6218** [R]
- 'C' of **rs5742704** [R]

The *COL1A1* gene encodes part of a large molecule called type I collagen. Collagens are a family of proteins that strengthen and support many tissues in the body, including cartilage, bone, tendon, and skin. Type I collagen is the most abundant form of collagen in the human body. [R].

The best-researched *COL1A1* polymorphism is **rs1800012**. Its minor 'A' allele promotes the expression of an alternative version of the protein with increased pro-α1 chain levels, potentially increasing its mechanical strength and stability. Carriers may experience enhanced tissue regeneration from Follistatin-344 [R].

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].

The best-researched *PPARGC1A* polymorphism is **rs8192678**. Its minor 'T' allele decreases *PPARGC1A* expression and PGC-1α levels in the muscles. Carriers may benefit more from the anabolic and recovery effects of Follistatin-344 [R, R, R].

Finally, interleukin-6 (IL-6) is a cytokine encoded by the *IL6* gene that has either proinflammatory or anti-inflammatory properties, depending on the circumstances and the tissue that secretes it. It has complicated mechanisms of effect and interacts with many other cytokines and inflammatory signals [R, R].

By far, the most well-researched *IL6* polymorphism is **rs1800795** (also known as the "-174G>C" polymorphism). The major 'G' allele of this SNP has been linked with higher levels of IL-6, while the 'C' allele has been associated with lower IL-6 levels. The major 'G' allele may lead to a more pronounced inflammatory response. Carriers may benefit more from Follistatin-344's anti-inflammatory effects [R].

Other variants of this gene linked to an enhanced inflammatory response include:

- 'C' of **rs1524107** [R]
- 'A' of **rs75045751** [R]

Response To GHRP-2 (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of GHRP-2.*

Genetic variation in growth hormone signaling may influence individual response to GHRP-2, affecting GH release, muscle development, fat oxidation, and appetite modulation.

The *GHRL* gene encodes the ghrelin-obestatin preproprotein, which is cleaved to yield two peptides: ghrelin and obestatin. The "hunger hormone" ghrelin stimulates appetite, promotes eating, and increases fat storage. Recent reports suggest multiple metabolic roles for obestatin, including regulating adipocyte function and glucose metabolism [R, R, R].

The following *GHRL* variants have been associated with lower ghrelin levels. Carriers may benefit more from GHRP-2 administration to compensate for the absence of this hormone:

- 'T' of **rs34911341** [R, R]
- 'T' of **rs4684677** [R, R]
- 'C' of **rs55821288** [R]
- 'G' of **rs35682** [R]
- 'C' of **rs35680** [R]
- 'A' of **rs35683** [R]
- 'T' of **rs35681** [R]

The *GHSR* gene encodes the growth hormone secretagogue receptor. Upon activation by the hunger hormone ghrelin, it signals the pituitary gland to start producing growth hormone [R].

Variants in this gene linked to an increased sensitivity of the receptor may increase the effectiveness of GHRP-2 treatment. Key variants include:

- 'A' of **rs509035** [R, R, R]
- 'T' of **rs572169** [R, R, R, R, R]
- 'C' of **rs562416** [R]
- 'A' of **rs6774762** [R]
- 'A' of **rs56271032** [R]

TYPICAL RESPONSE

Predisposed to a typical response to GHRP-2 based on 9 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GHRL	rs34911341	CC
GH1	rs5388	CC
GHR	rs6184	CC
CCDC152	rs17574650	AA
CCDC152	rs55681913	TT
GHR	rs78095808	CC
CCDC152	rs62372052	AA
IGF1	rs6214	CC
IGF1	rs5742692	AA
ASCL1	rs56006730	GG
WASHC3	rs78929770	CC
IGF1	rs855203	AA
WASHC3	rs5742704	TT
TNFSF10	rs509035	GG
TNFSF10	rs572169	CC
GHSR	rs562416	AA
TNFSF10	rs519384	TT
TNFSF10	rs79204749	AA
TNFSF10	rs115898535	CC
GHRL	rs35682	GA
GHRL	rs35680	CT
GHRL	rs35683	AC
GHRL	rs35681	TC
CD79B	rs3020619	AG
FTSJ3	rs7209608	AC
GHR	rs6873545	CT
GHR	rs4273617	GA
CCDC152	rs12654242	GA
OXCT1	rs16872401	TC
IGF1	rs833718	GA

- ‘A’ of **rs519384** [R]
- ‘C’ of **rs79204749** [R]
- ‘C’ of **rs16845548** [R]
- ‘A’ of **rs13061442** [R]
- ‘A’ of **rs115898535** [R]
- ‘A’ of **rs13085429** [R]
- ‘C’ of **rs558572** [R]
- ‘T’ of **rs530893** [R]

The *GH1* gene encodes the growth hormone protein. This hormone is produced in the growth-stimulating somatotropic cells of the pituitary gland, and is necessary for the normal growth of the body's bones and tissues [R].

Certain variants in the *GH1* gene may increase the amount of growth hormone secreted, potentially enhancing the effectiveness of GHRP-2. Some of these variants include

- ‘G’ of **rs3020619** [R, R]
- ‘A’ of **rs7209608** [R, R]
- ‘T’ of **rs5388** [R, R]
- ‘G’ of **rs3020619** [R, R]

The *GHR* gene encodes the growth hormone receptor, found in the outer membrane of cells and most abundant in the liver [R].

Specific genetic variants in this gene may lead to altered receptor sensitivity and impact the efficacy of GHRP-2 therapy. Some key variants associated with increased sensitivity to growth hormone, leading to a potentially higher response to GHRP-2, include:

- ‘C’ of **rs6873545** [R, R, R, R]
- ‘A’ of **rs6184** (Pro579Thr) [R, R]
- ‘A’ of **rs6180** (Ile544Leu) [R, R, R]
- ‘G’ of **rs2910875** [R]
- ‘C’ of **rs17574650** [R]
- ‘C’ of **rs7736209** [R]
- ‘T’ of **rs2972781** [R]
- ‘G’ of **rs4273617** [R]
- ‘C’ of **rs55681913** [R, R]
- ‘T’ of **rs78095808** [R]
- ‘G’ of **rs62372052** [R, R]
- ‘G’ of **rs12654242** [R]
- ‘G’ of **rs2972770** [R]
- ‘A’ of **rs10066141** [R]
- ‘C’ of **rs2973018** [R]
- ‘C’ of **rs16872401** [R]

GENE	SNP	GENOTYPE
TNFSF10	rs13061442	AC
WASHC3	rs978458	TT
GHR	rs7736209	CC
GHSR	rs6774762	AA
WASHC3	rs6219	TT
IGF1	rs6218	AA
GHR	rs6180	AA
TNFSF10	rs56271032	AA
GHSR	rs558572	CC
GHRL	rs55821288	CC
GHSR	rs530893	TT
GHRL	rs4684677	TT
CCDC152	rs2973018	CC
CCDC152	rs2972781	TT
CCDC152	rs2972770	GG
GHR	rs2910875	GG
TNFSF10	rs16845548	CC
GHSR	rs13085429	AA
ASCL1	rs12830406	AA
PAH	rs10507151	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Finally, the *IGF1* gene encodes insulin-like growth factor 1, a protein that has similar functions to insulin and is involved in controlling growth and development in an organism. IGF1 is produced in the liver in response to growth hormone [R].

Variants in *IGF1* can affect IGF-1 levels, thereby influencing the therapeutic outcomes of GHRP-2. Those that may lead to a higher response to this peptide include:

- 'T' of **rs6214** [R]
- 'C' of **rs1019731** [R]
- 'G' of **rs5742692** [R]
- 'A' of **rs56006730** [R]
- 'T' of **rs978458** [R]
- 'A' of **rs78929770** [R]
- 'C' of **rs855203** [R]
- 'G' of **rs833718** [R]
- 'A' of **rs12830406** [R]
- 'G' of **rs10507151** [R]
- 'T' of **rs6219** [R]
- 'A' of **rs6218** [R]
- 'C' of **rs5742704** [R]

Response To GHRP-6 (Hypothesis)

Genetic variation in growth hormone signaling may influence individual response to GHRP-6, affecting GH release, muscle development, fat oxidation, and appetite modulation.

The *GHRL* gene encodes the ghrelin-obestatin preproprotein, which is cleaved to yield two peptides: ghrelin and obestatin. The “hunger hormone” ghrelin stimulates appetite, promotes eating, and increases fat storage. Recent reports suggest multiple metabolic roles for obestatin, including regulating adipocyte function and glucose metabolism [R, R, R].

The following *GHRL* variants have been associated with lower ghrelin levels. Carriers may benefit more from GHRP-6 administration to compensate for the absence of this hormone:

- ‘T’ of **rs34911341** [R, R]
- ‘T’ of **rs4684677** [R, R]
- ‘C’ of **rs55821288** [R]
- ‘G’ of **rs35682** [R]
- ‘C’ of **rs35680** [R]
- ‘A’ of **rs35683** [R]
- ‘T’ of **rs35681** [R]

The *GHSR* gene encodes the growth hormone secretagogue receptor. Upon activation by the hunger hormone ghrelin, it signals the pituitary gland to start producing growth hormone [R].

Variants in this gene linked to an increased sensitivity of the receptor may increase the effectiveness of GHRP-6 treatment. Key variants include:

- ‘A’ of **rs509035** [R, R, R]
- ‘T’ of **rs572169** [R, R, R, R, R]
- ‘C’ of **rs562416** [R]
- ‘A’ of **rs6774762** [R]
- ‘A’ of **rs56271032** [R]
- ‘A’ of **rs519384** [R]
- ‘C’ of **rs79204749** [R]
- ‘C’ of **rs16845548** [R]
- ‘A’ of **rs13061442** [R]
- ‘A’ of **rs115898535** [R]

TYPICAL RESPONSE

Predisposed to a typical response to GHRP-6 based on 9 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GHRL	rs34911341	CC
GH1	rs5388	CC
GHR	rs6184	CC
CCDC152	rs17574650	AA
CCDC152	rs55681913	TT
GHR	rs78095808	CC
CCDC152	rs62372052	AA
IGF1	rs6214	CC
IGF1	rs5742692	AA
ASCL1	rs56006730	GG
WASHC3	rs78929770	CC
IGF1	rs855203	AA
WASHC3	rs5742704	TT
TNFSF10	rs509035	GG
TNFSF10	rs572169	CC
GHSR	rs562416	AA
TNFSF10	rs519384	TT
TNFSF10	rs79204749	AA
TNFSF10	rs115898535	CC
GHRL	rs35682	GA
GHRL	rs35680	CT
GHRL	rs35683	AC
GHRL	rs35681	TC
CD79B	rs3020619	AG
FTSJ3	rs7209608	AC
GHR	rs6873545	CT
GHR	rs4273617	GA
CCDC152	rs12654242	GA
OXCT1	rs16872401	TC
IGF1	rs833718	GA

- ‘A’ of **rs13085429** [R]
- ‘C’ of **rs558572** [R]
- ‘T’ of **rs530893** [R]

The *GH1* gene encodes the growth hormone protein. This hormone is produced in the growth-stimulating somatotropic cells of the pituitary gland, and is necessary for the normal growth of the body's bones and tissues [R].

Certain variants in the *GH1* gene may increase the amount of growth hormone secreted, potentially enhancing the effectiveness of GHRP-6. Some of these variants include

- ‘G’ of **rs3020619** [R, R]
- ‘A’ of **rs7209608** [R, R]
- ‘T’ of **rs5388** [R, R]
- ‘G’ of **rs3020619** [R, R]

The *GHR* gene encodes the growth hormone receptor, found in the outer membrane of cells and most abundant in the liver [R].

Specific genetic variants in this gene may lead to altered receptor sensitivity and impact the efficacy of GHRP-6 therapy. Some key variants associated with increased sensitivity to growth hormone, leading to a potentially higher response to GHRP-6, include:

- ‘C’ of **rs6873545** [R, R, R, R]
- ‘A’ of **rs6184** (Pro579Thr) [R, R]
- ‘A’ of **rs6180** (Ile544Leu) [R, R, R]
- ‘G’ of **rs2910875** [R]
- ‘C’ of **rs17574650** [R]
- ‘C’ of **rs7736209** [R]
- ‘T’ of **rs2972781** [R]
- ‘G’ of **rs4273617** [R]
- ‘C’ of **rs55681913** [R, R]
- ‘T’ of **rs78095808** [R]
- ‘G’ of **rs62372052** [R, R]
- ‘G’ of **rs12654242** [R]
- ‘G’ of **rs2972770** [R]
- ‘A’ of **rs10066141** [R]
- ‘C’ of **rs2973018** [R]
- ‘C’ of **rs16872401** [R]

Finally, the *IGF1* gene encodes insulin-like growth factor 1, a protein that has similar functions to insulin and is involved in controlling growth and development in an organism. IGF1 is produced in the liver in response to growth hormone [R].

GENE	SNP	GENOTYPE
TNFSF10	rs13061442	AC
WASHC3	rs978458	TT
GHR	rs7736209	CC
GHSR	rs6774762	AA
WASHC3	rs6219	TT
IGF1	rs6218	AA
GHR	rs6180	AA
TNFSF10	rs56271032	AA
GHSR	rs558572	CC
GHRL	rs55821288	CC
GHSR	rs530893	TT
GHRL	rs4684677	TT
CCDC152	rs2973018	CC
CCDC152	rs2972781	TT
CCDC152	rs2972770	GG
GHR	rs2910875	GG
TNFSF10	rs16845548	CC
GHSR	rs13085429	AA
ASCL1	rs12830406	AA
PAH	rs10507151	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Variants in *IGF1* can affect IGF-1 levels, thereby influencing the therapeutic outcomes of GHRP-6. Those that may lead to a higher response to this peptide include:

- 'T' of **rs6214** [R]
- 'C' of **rs1019731** [R]
- 'G' of **rs5742692** [R]
- 'A' of **rs56006730** [R]
- 'T' of **rs978458** [R]
- 'A' of **rs78929770** [R]
- 'C' of **rs855203** [R]
- 'G' of **rs833718** [R]
- 'A' of **rs12830406** [R]
- 'G' of **rs10507151** [R]
- 'T' of **rs6219** [R]
- 'A' of **rs6218** [R]
- 'C' of **rs5742704** [R]

Response To CJC-1295 (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of CJC-1295.*

CJC-1295 works by mimicking the action of GHRH, which in turn stimulates the release of growth hormone from the pituitary gland. However, individual responses to CJC-1295 can vary based on genetic factors that influence growth hormone signaling and receptor activity. Several genes and genetic variants are associated with the body's response to this peptide.

The *GHRHR* gene encodes the growth hormone-releasing hormone receptor. This receptor, located on the growth-stimulating somatotropic cells in the pituitary gland, binds to the growth hormone-releasing hormone to trigger the production of growth hormone and its release from the pituitary gland [R].

Variants in this gene linked to an increased sensitivity of the receptor may increase the effectiveness of CJC-1295 treatment. Key variants include:

- 'C' of **rs2228078** [R, R]
- 'G' of **rs11763787** [R]
- 'T' of **rs10278927** [R]
- 'T' of **rs10271008** [R]
- 'A' of **rs881499** [R]
- 'T' of **rs10280233** [R]

The *GH1* gene encodes the growth hormone protein. This hormone is produced in the growth-stimulating somatotropic cells of the pituitary gland, and is necessary for the normal growth of the body's bones and tissues [R].

Certain variations in the *GH1* gene may increase the amount of growth hormone secreted, potentially enhancing the effectiveness of CJC-1295. Some of these variants include

- 'G' of **rs3020619** [R, R]
- 'A' of **rs7209608** [R, R]
- 'T' of **rs5388** [R, R]
- 'G' of **rs3020619** [R, R]

TYPICAL RESPONSE

Predisposed to a typical response to CJC-1295 based on 10 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GHRHR	rs2228078	TT
GHRHR	rs10278927	CC
ADCYAP1R1	rs10271008	CC
GHRHR	rs10280233	CC
GH1	rs5388	CC
GHR	rs6184	CC
CCDC152	rs17574650	AA
CCDC152	rs55681913	TT
GHR	rs78095808	CC
CCDC152	rs62372052	AA
IGF1	rs6214	CC
IGF1	rs5742692	AA
ASCL1	rs56006730	GG
WASHC3	rs78929770	CC
IGF1	rs855203	AA
WASHC3	rs5742704	TT
CD79B	rs3020619	AG
FTSJ3	rs7209608	AC
GHR	rs6873545	CT
GHR	rs4273617	GA
CCDC152	rs12654242	GA
OXCT1	rs16872401	TC
IGF1	rs833718	GA
WASHC3	rs978458	TT
AQP1	rs881499	AA
GHR	rs7736209	CC
WASHC3	rs6219	TT
IGF1	rs6218	AA
GHR	rs6180	AA
CCDC152	rs2973018	CC

The *GHR* gene encodes the growth hormone receptor, found in the outer membrane of cells and most abundant in the liver [R].

Specific genetic variants in this gene may lead to altered receptor sensitivity and impact the efficacy of CJC-1295 therapy. Some key variants associated with increased sensitivity to growth hormone, leading to a potentially higher response to CJC-1295, include:

- 'C' of **rs6873545** [R, R, R, R]
- 'A' of **rs6184** (Pro579Thr) [R, R]
- 'A' of **rs6180** (Ile544Leu) [R, R, R]
- 'G' of **rs2910875** [R]
- 'C' of **rs17574650** [R]
- 'C' of **rs7736209** [R]
- 'T' of **rs2972781** [R]
- 'G' of **rs4273617** [R]
- 'C' of **rs55681913** [R, R]
- 'T' of **rs78095808** [R]
- 'G' of **rs2910875** [R]
- 'G' of **rs62372052** [R]
- 'G' of **rs12654242** [R]
- 'G' of **rs2972770** [R]
- 'A' of **rs10066141** [R]
- 'G' of **rs62372052** [R]
- 'C' of **rs2973018** [R]
- 'C' of **rs16872401** [R]

Finally, the *IGF1* gene encodes insulin-like growth factor 1, a protein that has similar functions to insulin and is involved in controlling growth and development in an organism. IGF1 is produced in the liver in response to growth hormone [R].

Variants in *IGF1* can affect IGF-1 levels, thereby influencing the therapeutic outcomes of CJC-1295. Those that may lead to higher response to this peptide include:

- 'T' of **rs6214** [R]
- 'C' of **rs1019731** [R]
- 'G' of **rs5742692** [R]
- 'A' of **rs56006730** [R]
- 'T' of **rs978458** [R]
- 'A' of **rs78929770** [R]
- 'C' of **rs855203** [R]
- 'G' of **rs833718** [R]
- 'A' of **rs12830406** [R]
- 'G' of **rs10507151** [R]
- 'A' of **rs5742692** [R]

GENE	SNP	GENOTYPE
CCDC152	rs2972781	TT
CCDC152	rs2972770	GG
GHR	rs2910875	GG
ASCL1	rs12830406	AA
ADCYAP1R1	rs11763787	GG
PAH	rs10507151	GG
IGF1	rs1019731	CC
CCDC152	rs10066141	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

- ‘T’ of **rs6219** [R]
- ‘A’ of **rs6218** [R]
- ‘C’ of **rs5742704** [R

Response To Thymosin Beta-4 (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Thymosin Beta-4.*

Because TB-4 interfaces with angiogenesis, ECM remodeling, and inflammation/oxidative stress, variants across these pathways can plausibly modify outcomes. Direct pharmacogenomic studies for TB-4 are limited, meaning the markers below are mechanism-informed candidates.

The *VEGFA* gene codes for vascular endothelial growth factor A (VEGF-A). VEGF-A supports blood vessel formation and wound healing. It does this by stimulating the growth and division of cells that line blood vessels [R].

Key genetic variants that could boost TB4's efficacy through their link to higher VEGF levels include

- 'C' of **rs833070** [R]
- 'G' of **rs11965885** [R, R]
- 'C' of **rs699947** [R]

The *MMP1* gene codes for matrix metalloproteinase 1, also known as interstitial collagenase. This enzyme plays a significant role in collagen breakdown; it degrades type I and III collagen, the primary types found in skin. While MMP1 activity is essential for normal skin remodeling and repair, excessive activity can accelerate collagen degradation, leading to premature aging [R].

Variants with decreased MMP1 activity may affect how well the TB4 peptide can accelerate healing. These include:

- 'T' of **rs1799750** [R]
- 'A' of **rs470747** [R]
- 'G' of **rs139018071** [R]
- 'T' of **rs470558** [R]
- 'C' of **rs17879749** [R]

The *MMP9* gene codes for matrix metalloproteinase 9, also known as 92 kDa type IV collagenase, 92 kDa gelatinase, or

TYPICAL RESPONSE

Predisposed to a typical response to Thymosin beta-1 based on 9 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MMP1	rs139018071	AA
MMP1	rs470558	CC
IGF1	rs6214	CC
IGF1	rs5742692	AA
ASCL1	rs56006730	GG
WASHC3	rs78929770	CC
IGF1	rs855203	AA
WASHC3	rs5742704	TT
TMEM91	rs12461895	AA
TGFB1	rs73045269	CC
TNF	rs1800629	GG
TNF	rs1799964	TT
IL10	rs1800871	GG
IL19	rs1800872	GG
STEAP1B	rs75045751	GG
GPX1	rs1050450	GG
VEGFA	rs833070	TC
VEGFA	rs699947	AC
MMP9	rs17576	AG
PLTP	rs3918249	TC
PLTP	rs6094237	AT
IGF1	rs833718	GA
TNF	rs1799724	TC
IL19	rs1800896	CT
IL19	rs1800890	TA
IL19	rs3024505	AG
IL6	rs1524107	CT
CAT	rs1001179	TC
CAT	rs7943316	TA
WASHC3	rs978458	TT

gelatinase B. This enzyme plays a significant role in collagen breakdown; it degrades type IV and V collagens. While MMP9 activity is essential for embryonic development, reproduction, and tissue remodeling, excessive activity can accelerate collagen degradation, leading to arthritis and metastasis [R].

The following *MMP9* variants may lead to better tissue repair outcomes when using TB4:

- 'A' of **rs17576** [R]
- 'T' of **rs3918249** [R]
- 'A' of **rs6094237** [R]
- 'G' of **rs2250889** [R]

The *TGFB1* gene codes for transforming growth factor beta 1 (TGF-beta1). TGF-beta1 is an immune messenger (cytokine) that plays a role in many cellular processes, including cell growth & division, specialization, movement, and death. By doing so, it controls bone, cartilage, and blood vessel formation, muscle and fat development, wound healing, and inflammation [R].
Certain alleles linked to higher TGFB1 expression may amplify collagen deposition and tissue regeneration. These include:

- 'C' of **rs12461895** [R]
- 'G' of **rs1800472** [R]
- 'T' of **rs73045269** [R]

The *IGF1* gene encodes insulin-like growth factor 1, a protein that has similar functions to insulin and is involved in controlling growth and development in an organism. IGF1 is produced in the liver in response to growth hormone [R].

Variants in *IGF1* can affect IGF-1 levels, thereby influencing the therapeutic outcomes of TB4. Those that may lead to higher response to this peptide include:

- 'T' of **rs6214** [R]
- 'C' of **rs1019731** [R]
- 'G' of **rs5742692** [R]
- 'A' of **rs56006730** [R]
- 'T' of **rs978458** [R]
- 'A' of **rs78929770** [R]
- 'C' of **rs855203** [R]
- 'G' of **rs833718** [R]
- 'A' of **rs12830406** [R]
- 'G' of **rs10507151** [R]
- 'T' of **rs6219** [R]
- 'A' of **rs6218** [R]
- 'C' of **rs5742704** [R]

GENE	SNP	GENOTYPE
CAT	rs769217	CC
WASHC3	rs6219	TT
IGF1	rs6218	AA
SOD2	rs4880	GG
MMP1	rs470747	AA
MMP9	rs2250889	CC
IL6	rs1800795	GG
TGFB1	rs1800472	GG
MMP1	rs1799750	TT
MMP1	rs17879749	CC
ASCL1	rs12830406	AA
VEGFA	rs11965885	GG
PAH	rs10507151	GG
IGF1	rs1019731	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Interleukin-6 (IL-6) is a cytokine encoded by the *IL6* gene that has either proinflammatory or anti-inflammatory properties, depending on the circumstances and the tissue that secretes it. It has complicated mechanisms of effect and interacts with many other cytokines and inflammatory signals [R, R].

By far, the most well-researched *IL6* polymorphism is **rs1800795** (also known as the “-174G>C” polymorphism). The major 'G' allele of this SNP has been linked with higher levels of IL-6, while the 'C' allele has been associated with lower IL-6 levels. The major ‘G’ allele may lead to a more pronounced inflammatory response. Carriers may benefit more from TB4’s anti-inflammatory effects [R].

Other variants of this gene linked to an enhanced inflammatory response include:

- ‘C’ of **rs1524107** [R]
- ‘A’ of **rs75045751** [R]

The *TNF* gene encodes a protein called tumor necrosis factor-alpha (TNF-alpha or cachexin). TNF-alpha plays a central role in the immune response and inflammation, and it has been implicated in a wide variety of inflammatory disorders [R].

The **rs1800629** polymorphism (also known as *TNF* -308) is one of the most researched SNPs in the *TNF* gene. **The ‘A’ allele is associated with 6-7 times higher levels of TNF-alpha.** This may result in a better response to TB4 [R].

Other variants that may increase TNF levels include:

- ‘C’ of **rs1799964** [R]
- ‘C’ of **rs1799724** [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 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].

The *SOD2* gene has many described polymorphisms. Among them, **rs4880** has received the spotlight in SOD2 research. Its minor allele ‘G’ is associated with decreased activity and worse protection against oxidative stress. Carriers may respond better to TB4’s antioxidant and cytoprotective actions [R].

The *CAT* gene encodes subunits of catalase, a key antioxidant enzyme in the body's defense against oxidative stress. Four identical subunits, each attached to an iron-containing molecule called a heme group, form the functional catalase enzyme [R].

The best-characterized *CAT* polymorphism is **rs1001179**, commonly referred to as -262G>A. Its minor 'T' allele may increase *CAT* expression but decrease it in people with conditions such as type 2 diabetes or chronic hepatitis C. Other well-known CAT variants with increased activity include 'T' of **rs769217** and 'A' of **rs7943316.** They may decrease the antioxidant effects of TB4 [R, R, R, R, R].

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].

One study found a direct link between a common *GPX1* variant and human longevity. The heterozygous **genotype 'AG' at rs1050450 was significantly more common in the very elderly** than in the general population. Other studies have strongly suggested that the **'G' allele at rs1050450 confers higher GPx activity**. Carriers may benefit less from TB4 [R, R, R].

Response To BPC-157 (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of BPC-157.*

BPC-157 works by interacting with a variety of signaling pathways involved in tissue repair, blood vessel formation, and inflammation modulation. Its effectiveness in promoting healing may vary based on individual genetic factors, especially those that influence tissue repair, inflammation, and growth factor activity.

The *VEGFA* gene codes for vascular endothelial growth factor A (VEGF-A). VEGF-A supports blood vessel formation and wound healing. It does this by stimulating the growth and division of cells that line blood vessels [R].

Key genetic variants that could influence BPC-157's efficacy through their link to higher VEGF levels include

- 'C' of **rs833070** [R]
- 'G' of **rs11965885** [R, R]
- 'C' of **rs699947** [R]

The *MMP1* gene codes for matrix metalloproteinase 1, also known as interstitial collagenase. This enzyme plays a significant role in collagen breakdown; it degrades type I and III collagen, the primary types found in skin. While MMP1 activity is essential for normal skin remodeling and repair, excessive activity can accelerate collagen degradation, leading to premature aging [R].

Variants with decreased MMP1 activity may affect how well the BPC-157 peptide can accelerate healing. These include:

- 'T' of **rs1799750** [R]
- 'A' of **rs470747** [R]
- 'G' of **rs139018071** [R]
- 'T' of **rs470558** [R]
- 'C' of **rs17879749** [R]

BETTER RESPONSE

Predisposed to a better BPC-157 response based on 26 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MMP1	rs139018071	AA
MMP1	rs470558	CC
IGF1	rs6214	CC
IGF1	rs5742692	AA
ASCL1	rs56006730	GG
WASHC3	rs78929770	CC
IGF1	rs855203	AA
WASHC3	rs5742704	TT
TMEM91	rs12461895	AA
TGFB1	rs73045269	CC
VEGFA	rs833070	TC
VEGFA	rs699947	AC
MMP9	rs17576	AG
PLTP	rs3918249	TC
PLTP	rs6094237	AT
IGF1	rs833718	GA
WASHC3	rs978458	TT
WASHC3	rs6219	TT
IGF1	rs6218	AA
MMP1	rs470747	AA
MMP9	rs2250889	CC
TGFB1	rs1800472	GG
MMP1	rs1799750	TT
MMP1	rs17879749	CC
ASCL1	rs12830406	AA
VEGFA	rs11965885	GG
PAH	rs10507151	GG
IGF1	rs1019731	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

The *MMP9* gene codes for matrix metalloproteinase 9, also known as 92 kDa type IV collagenase, 92 kDa gelatinase, or gelatinase B. This enzyme plays a significant role in collagen breakdown; it degrades type IV and V collagens. While MMP9 activity is essential for embryonic development, reproduction, and tissue remodeling, excessive activity can accelerate collagen degradation, leading to arthritis and metastasis [R].

The following *MMP9* variants may lead to better tissue repair outcomes when using BPC-157:

- 'A' of **rs17576** [R]
- 'T' of **rs3918249** [R]
- 'A' of **rs6094237** [R]
- 'G' of **rs2250889** [R]

The *TGFB1* gene codes for transforming growth factor beta 1 (TGF-beta1). TGF-beta1 is an immune messenger (cytokine) that plays a role in many cellular processes, including cell growth & division, specialization, movement, and death. By doing so, it controls bone, cartilage, and blood vessel formation, muscle and fat development, wound healing, and inflammation [R].

Certain alleles linked to higher TGFB1 expression may amplify collagen deposition and tissue regeneration. These include:

- 'C' of **rs12461895** [R]
- 'G' of **rs1800472** [R]
- 'T' of **rs73045269** [R]

Finally, the *IGF1* gene encodes insulin-like growth factor 1, a protein that has similar functions to insulin and is involved in controlling growth and development in an organism. IGF1 is produced in the liver in response to growth hormone [R].

Variants in *IGF1* can affect IGF-1 levels, thereby influencing the therapeutic outcomes of BPC-157. Those that may lead to higher response to this peptide include:

- 'T' of **rs6214** [R]
- 'C' of **rs1019731** [R]
- 'G' of **rs5742692** [R]
- 'A' of **rs56006730** [R]
- 'T' of **rs978458** [R]
- 'A' of **rs78929770** [R]
- 'C' of **rs855203** [R]
- 'G' of **rs833718** [R]
- 'A' of **rs12830406** [R]
- 'G' of **rs10507151** [R]

- ‘T’ of **rs6219** [R]
- ‘A’ of **rs6218** [R]
- ‘C’ of **rs5742704** [R]

Response To TB-500 (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of TB-500.*

TB-500 exerts its effects by influencing cellular pathways involved in tissue regeneration, cell migration, and angiogenesis. Individual responses to TB-500 may vary based on genetic differences that affect growth factors, inflammatory pathways, and extracellular matrix remodeling.

The *VEGFA* gene codes for vascular endothelial growth factor A (VEGF-A). VEGF-A supports blood vessel formation and wound healing. It does this by stimulating the growth and division of cells that line blood vessels [R].

Key genetic variants that could influence TB-500's efficacy through their link to higher VEGF levels include

- 'C' of **rs833070** [R]
- 'G' of **rs11965885** [R, R]
- 'C' of **rs699947** [R]

The *MMP1* gene codes for matrix metalloproteinase 1, also known as interstitial collagenase. This enzyme plays a significant role in collagen breakdown; it degrades type I and III collagen, the primary types found in skin. While MMP1 activity is essential for normal skin remodeling and repair, excessive activity can accelerate collagen degradation, leading to premature aging [R].

Variants with decreased MMP1 activity may affect how well the TB-500 peptide can accelerate healing. These include:

- 'T' of **rs1799750** [R]
- 'A' of **rs470747** [R]
- 'G' of **rs139018071** [R]
- 'T' of **rs470558** [R]
- 'C' of **rs17879749** [R]

The *MMP9* gene codes for matrix metalloproteinase 9, also known as 92 kDa type IV collagenase, 92 kDa gelatinase, or

BETTER RESPONSE

Predisposed to a better response to TB-500 based on 26 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MMP1	rs139018071	AA
MMP1	rs470558	CC
IGF1	rs6214	CC
IGF1	rs5742692	AA
ASCL1	rs56006730	GG
WASHC3	rs78929770	CC
IGF1	rs855203	AA
WASHC3	rs5742704	TT
TMEM91	rs12461895	AA
TGFB1	rs73045269	CC
VEGFA	rs833070	TC
VEGFA	rs699947	AC
MMP9	rs17576	AG
PLTP	rs3918249	TC
PLTP	rs6094237	AT
IGF1	rs833718	GA
WASHC3	rs978458	TT
WASHC3	rs6219	TT
IGF1	rs6218	AA
MMP1	rs470747	AA
MMP9	rs2250889	CC
TGFB1	rs1800472	GG
MMP1	rs1799750	TT
MMP1	rs17879749	CC
ASCL1	rs12830406	AA
VEGFA	rs11965885	GG
PAH	rs10507151	GG
IGF1	rs1019731	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

gelatinase B. This enzyme plays a significant role in collagen breakdown; it degrades type IV and V collagens. While MMP9 activity is essential for embryonic development, reproduction, and tissue remodeling, excessive activity can accelerate collagen degradation, leading to arthritis and metastasis [R].

The following *MMP9* variants may lead to better tissue repair outcomes when using TB-500:

- 'A' of **rs17576** [R]
- 'T' of **rs3918249** [R]
- 'A' of **rs6094237** [R]
- 'G' of **rs2250889** [R]

The *TGFB1* gene codes for transforming growth factor beta 1 (TGF-beta1). TGF-beta1 is an immune messenger (cytokine) that plays a role in many cellular processes, including cell growth & division, specialization, movement, and death. By doing so, it controls bone, cartilage, and blood vessel formation, muscle and fat development, wound healing, and inflammation [R].
Certain alleles linked to higher TGFB1 expression may amplify collagen deposition and tissue regeneration. These include:

- 'C' of **rs12461895** [R]
- 'G' of **rs1800472** [R]
- 'T' of **rs73045269** [R]

Finally, the *IGF1* gene encodes insulin-like growth factor 1, a protein that has similar functions to insulin and is involved in controlling growth and development in an organism. IGF1 is produced in the liver in response to growth hormone [R].

Variants in *IGF1* can affect IGF-1 levels, thereby influencing the therapeutic outcomes of TB-500. Those that may lead to higher response to this peptide include:

- 'T' of **rs6214** [R]
- 'C' of **rs1019731** [R]
- 'G' of **rs5742692** [R]
- 'A' of **rs56006730** [R]
- 'T' of **rs978458** [R]
- 'A' of **rs78929770** [R]
- 'C' of **rs855203** [R]
- 'G' of **rs833718** [R]
- 'A' of **rs12830406** [R]
- 'G' of **rs10507151** [R]
- 'T' of **rs6219** [R]
- 'A' of **rs6218** [R]
- 'C' of **rs5742704** [R]

Response To Ipamorelin (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Ipamorelin.*

Ipamorelin acts through the stimulation of the GHS-R1a receptor in the pituitary gland, leading to GH release and subsequent IGF-1 production. While research hasn't directly linked specific variants to Ipamorelin response, the effectiveness of this peptide may vary depending on variants influencing GH secretion, receptor sensitivity, and IGF-1 production.

The *GHSR* gene encodes the ghrelin receptor, which binds ghrelin and stimulates the pituitary gland to start releasing growth hormone.

Variants that may increase receptor sensitivity or expression, potentially enhancing Ipamorelin's effect, include:

- 'A' of **rs509035**
- 'T' of **rs572169**
- 'A' of **rs6774762**
- 'A' of **rs562416**
- 'A' of **rs519384**
- 'C' of **rs79204749**
- 'A' of **rs13085429**
- 'C' of **rs558572**

The *GH1* gene encodes the growth hormone protein. This hormone is produced in the growth-stimulating somatotropic cells of the pituitary gland, and is necessary for the normal growth of the body's bones and tissues [R].

Certain variations in the *GH1* gene may increase the amount of growth hormone secreted, potentially enhancing the effectiveness of Ipamorelin. Some of these variants include

- 'G' of **rs3020619** [R, R]
- 'A' of **rs7209608** [R, R]
- 'T' of **rs5388** [R, R]
- 'G' of **rs3020619** [R, R]

BETTER RESPONSE

Predisposed to a better response to Ipamorelin based on 39 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TNFSF10	rs509035	GG
TNFSF10	rs572169	CC
TNFSF10	rs519384	TT
TNFSF10	rs79204749	AA
GH1	rs5388	CC
GHR	rs6184	CC
CCDC152	rs17574650	AA
CCDC152	rs55681913	TT
GHR	rs78095808	CC
CCDC152	rs62372052	AA
IGF1	rs6214	CC
IGF1	rs5742692	AA
ASCL1	rs56006730	GG
WASHC3	rs78929770	CC
IGF1	rs855203	AA
WASHC3	rs5742704	TT
FTSJ3	rs7209608	AC
GHR	rs6873545	CT
GHR	rs4273617	GA
CCDC152	rs12654242	GA
OXCT1	rs16872401	TC
IGF1	rs833718	GA
WASHC3	rs978458	TT
GHR	rs7736209	CC
GHSR	rs6774762	AA
WASHC3	rs6219	TT
IGF1	rs6218	AA
GHR	rs6180	AA
GHSR	rs562416	AA
GHSR	rs558572	CC

The *GHR* gene encodes the growth hormone receptor, found in the outer membrane of cells and most abundant in the liver [R].

Specific genetic variants in this gene may lead to altered receptor sensitivity and impact the efficacy of ipamorelin therapy. Some key variants associated with increased sensitivity to growth hormone, leading to a potentially higher response to Ipamorelin, include:

- 'C' of **rs6873545** [R, R, R, R]
- 'A' of **rs6184** (Pro579Thr) [R, R]
- 'A' of **rs6180** (Ile544Leu) [R, R, R]
- 'G' of **rs2910875** [R]
- 'C' of **rs17574650** [R]
- 'C' of **rs7736209** [R]
- 'T' of **rs2972781** [R]
- 'G' of **rs4273617** [R]
- 'C' of **rs55681913** [R, R]
- 'T' of **rs78095808** [R]
- 'G' of **rs62372052** [R, R]
- 'G' of **rs12654242** [R]
- 'G' of **rs2972770** [R]
- 'A' of **rs10066141** [R]
- 'C' of **rs2973018** [R]
- 'C' of **rs16872401** [R]

Finally, the *IGF1* gene encodes insulin-like growth factor 1, a protein that has similar functions to insulin and is involved in controlling growth and development in an organism. IGF1 is produced in the liver in response to growth hormone [R].

Variants in *IGF1* can affect IGF-1 levels, thereby influencing the therapeutic outcomes of Ipamorelin. Those that may lead to higher response to this peptide include:

- 'T' of **rs6214** [R]
- 'C' of **rs1019731** [R]
- 'G' of **rs5742692** [R]
- 'A' of **rs56006730** [R]
- 'T' of **rs978458** [R]
- 'A' of **rs78929770** [R]
- 'C' of **rs855203** [R]
- 'G' of **rs833718** [R]
- 'A' of **rs12830406** [R]
- 'G' of **rs10507151** [R]
- 'T' of **rs6219** [R]
- 'A' of **rs6218** [R]
- 'C' of **rs5742704** [R]

GENE	SNP	GENOTYPE
CCDC152	rs2973018	CC
CCDC152	rs2972781	TT
CCDC152	rs2972770	GG
GHR	rs2910875	GG
GHSR	rs13085429	AA
ASCL1	rs12830406	AA
PAH	rs10507151	GG
IGF1	rs1019731	CC
CCDC152	rs10066141	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Response To Sermorelin (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Sermorelin.*

Genetic variation in growth hormone signaling, metabolism, and receptor pathways may influence individual response to Sermorelin. This may affect GH release, muscle development, fat oxidation, and appetite modulation.

The *GHRHR* gene encodes the growth hormone-releasing hormone receptor. This receptor, located on the growth-stimulating somatotropic cells in the pituitary gland, binds to the growth hormone-releasing hormone to trigger the production of growth hormone and its release from the pituitary gland [R].

Variants in this gene linked to an increased sensitivity of the receptor may increase the effectiveness of Sermorelin treatment. Key variants include:

- 'C' of **rs2228078** [R, R]
- 'G' of **rs11763787** [R]
- 'T' of **rs10278927** [R]
- 'T' of **rs10271008** [R]
- 'A' of **rs881499** [R]
- 'T' of **rs10280233** [R]

The *GH1* gene encodes the growth hormone protein. This hormone is produced in the growth-stimulating somatotropic cells of the pituitary gland, and is necessary for the normal growth of the body's bones and tissues [R].

Certain variations in the *GH1* gene may increase the amount of growth hormone secreted, potentially enhancing the effectiveness of Sermorelin. Some of these variants include

- 'G' of **rs3020619** [R, R]
- 'A' of **rs7209608** [R, R]
- 'T' of **rs5388** [R, R]
- 'G' of **rs3020619** [R, R]

BETTER RESPONSE

Predisposed to a better response to Sermorelin based on 38 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GHRHR	rs2228078	TT
GHRHR	rs10278927	CC
ADCYAP1R1	rs10271008	CC
GHRHR	rs10280233	CC
GH1	rs5388	CC
GHR	rs6184	CC
CCDC152	rs17574650	AA
CCDC152	rs55681913	TT
GHR	rs78095808	CC
CCDC152	rs62372052	AA
IGF1	rs6214	CC
IGF1	rs5742692	AA
ASCL1	rs56006730	GG
WASHC3	rs78929770	CC
IGF1	rs855203	AA
WASHC3	rs5742704	TT
CD79B	rs3020619	AG
FTSJ3	rs7209608	AC
GHR	rs6873545	CT
GHR	rs4273617	GA
CCDC152	rs12654242	GA
OXCT1	rs16872401	TC
IGF1	rs833718	GA
WASHC3	rs978458	TT
AQP1	rs881499	AA
GHR	rs7736209	CC
WASHC3	rs6219	TT
IGF1	rs6218	AA
GHR	rs6180	AA
CCDC152	rs2973018	CC

The *GHR* gene encodes the growth hormone receptor, found in the outer membrane of cells and most abundant in the liver [R].

Specific genetic variants in this gene may lead to altered receptor sensitivity and impact the efficacy of Sermorelin therapy. Some key variants associated with increased sensitivity to growth hormone, leading to a potentially higher response to Sermorelin, include:

- 'C' of **rs6873545** [R, R, R, R]
- 'A' of **rs6184** (Pro579Thr) [R, R]
- 'A' of **rs6180** (Ile544Leu) [R, R, R]
- 'G' of **rs2910875** [R]
- 'C' of **rs17574650** [R]
- 'C' of **rs7736209** [R]
- 'T' of **rs2972781** [R]
- 'G' of **rs4273617** [R]
- 'C' of **rs55681913** [R, R]
- 'T' of **rs78095808** [R]
- 'G' of **rs62372052** [R, R]
- 'G' of **rs12654242** [R]
- 'G' of **rs2972770** [R]
- 'A' of **rs10066141** [R]
- 'C' of **rs2973018** [R]
- 'C' of **rs16872401** [R]

Finally, the *IGF1* gene encodes insulin-like growth factor 1, a protein that has similar functions to insulin and is involved in controlling growth and development in an organism. IGF1 is produced in the liver in response to growth hormone [R].

Variants in *IGF1* can affect IGF-1 levels, thereby influencing the therapeutic outcomes of Sermorelin. Those that may lead to higher response to this peptide include:

- 'T' of **rs6214** [R]
- 'C' of **rs1019731** [R]
- 'G' of **rs5742692** [R]
- 'A' of **rs56006730** [R]
- 'T' of **rs978458** [R]
- 'A' of **rs78929770** [R]
- 'C' of **rs855203** [R]
- 'G' of **rs833718** [R]
- 'A' of **rs12830406** [R]
- 'G' of **rs10507151** [R]
- 'T' of **rs6219** [R]
- 'A' of **rs6218** [R]
- 'C' of **rs5742704** [R]

GENE	SNP	GENOTYPE
CCDC152	rs2972781	TT
CCDC152	rs2972770	GG
GHR	rs2910875	GG
ASCL1	rs12830406	AA
ADCYAP1R1	rs11763787	GG
PAH	rs10507151	GG
IGF1	rs1019731	CC
CCDC152	rs10066141	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Weight Management & Energy Metabolism

Your metabolism, appetite control, and energy balance are tightly regulated by hormones, neurotransmitters, and mitochondrial function. This section examines peptides that influence fat metabolism, glucose regulation, and cellular energy production — including Semaglutide, Tesofensine, AOD-9604, MOTS-c, and more. These compounds can enhance metabolic efficiency, improve insulin sensitivity, and support weight management by optimizing how your body uses and stores energy. Understanding their effects can help guide strategies for maintaining a healthy body composition and sustaining long-term vitality.

WORSE RESPONSE

Response to GLP-1 (Ozempic)

Likely worse response to GLP-1 (Ozempic)

TYPICAL RESPONSE

Response to Retatrutide (Hypothesis)

Predisposed to a typical response to Retatrutide

TYPICAL RESPONSE

Response to Tirzepatide (Hypothesis)

Predisposed to a typical response to Tirzepatide

TYPICAL RESPONSE

Response to 5-Amino-1MQ (Hypothesis)

Predisposed to a typical response to 5-amino-1MQ

BETTER RESPONSE

Response to AOD-9604 (Hypothesis)

Predisposed to a better response to AOD-9604

BETTER RESPONSE

Response to MOTS-c (Hypothesis)

Predisposed to a better MOTS-c response

BETTER RESPONSE

Response to SS-31 (Hypothesis)

Predisposed to a better SS-31 response

Response To GLP-1 (Ozempic)

The following factors may affect the effectiveness of Ozempic at lowering HbA1c levels and promoting weight loss:

- Dosage and adherence
- Age and sex: Metabolic rates and body composition, which can vary with age and between genders, might influence how Ozempic affects blood sugar levels and weight.
- Diet and exercise: Lifestyle modifications, such as a balanced diet and regular physical activity, significantly enhance Ozempic's efficacy in lowering blood sugar and promoting weight loss.
  Body weight: Initial body weight can affect how noticeable the weight loss effects of Ozempic are. Those with higher starting weights may see more dramatic results.
  Diabetes progression: Those with a longer history of diabetes may have more beta-cell dysfunction and thus may not respond as well
- Drug interactions: Ozempic can interact with other medications, which may enhance or reduce its effectiveness. For example, certain medications like insulin or sulfonylureas might require dosage adjustments to prevent hypoglycemia.
- **Genetics**

A study of 4571 adults with type 2 diabetes identified several gene variants associated with a better response to GLP-1 receptor agonists such as Ozempic (measured as a greater decrease in HbA1c levels) [R].

Please note: Semaglutide (Ozempic) can have significant side effects, particularly when used off-label. Currently, its only approved indication is for the treatment of type 2 diabetes. Always consult your doctor before starting or stopping any medication.

WORSE RESPONSE

Likely worse response to GLP-1 (Ozempic) based on 16 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
PTPRN2	rs2286414	CC
B3GNTL1	rs4986076	GG
LPP	rs75941546	GG
TINCR	rs10561032	TT
TAFA5	rs5767119	CC
TCERG1L	rs2298192	AA
PTPN12	rs10224036	AA
CMYA5	rs11746176	CC
NR2E3	rs11072298	CC
SLC7A11	rs7687008	TT
ADAMTS18	rs56354900	TT
SLC6A3	rs40182	AA
LMX1A	rs61800555	GA
SAYSD1	rs2268640	AG
TUBA3E	rs1969320	GG
GLP1R	rs6923761	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Response To Retatrutide (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Retatrutide.*

Individual variation in response to Tirzepatide may be influenced by genes involved in incretin signaling, insulin secretion, glucose metabolism, and appetite regulation.

The *GLP1R* gene encodes a receptor for GLP-1, a hormone that plays a pivotal role in regulating blood sugar levels. GLP-1 is produced in the gut in response to food intake and lowers blood sugar by enhancing insulin, lowering glucagon, slowing stomach emptying, and reducing appetite [R, R, R, R].

The minor 'A' allele of **rs6923761** (Gly168Ser), which presumably encodes an overactivated version of the protein, has been associated with better anthropometric parameters (such as weight, BMI, and fat mass) and has been associated with greater weight loss but reduced metabolic benefits in response to GLP1R inhibitors such as liraglutide and semaglutide [R, R, R, R, R, R, R, R].

Other variants of this gene associated with better response to GLP1R inhibitors include:
'T' of **rs10305420** [R, R, R]
'G' of **rs3765467** [R]

The *GIPR* gene encodes a protein that plays an important role in insulin production in the body by acting as a receptor for the GIP incretin protein. When glucose levels become high, such as after a meal, GIP is secreted from special intestinal cells called K cells. This newly secreted GIP binds to GIPR located on the pancreas, which causes insulin secretion [R, R].

The main variants in the *GIPR* gene are **rs2287019** and **rs10423928**. They are almost always inherited together, meaning that you will most likely have either both or none of them. The minor alleles, rs2287019–**T** and rs10423928–**A**, may **reduce insulin secretion** after a meal. This

TYPICAL RESPONSE

Predisposed to a typical response to Retatrutide based on 9 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GLP1R	rs6923761	GG
CDKN1C	rs2237892	CC
CDKN1C	rs2237897	CC
GLP1R	rs10305420	CT
TCF7L2	rs7903146	CT
TCF7L2	rs12255372	GT
ADIPOQ	rs2241766	GT
GLP1R	rs3765467	GG
FTO	rs9939609	TT
KCNJ11	rs5219	CC
KCNJ11	rs5215	TT
GIPR	rs2287019	CC
PPARG	rs1801282	CC
FABP2	rs1799883	CC
MC4R	rs17782313	TT
RFC4	rs17300539	GG
ST6GAL1	rs1501299	GG
MC4R	rs12970134	GG
GIPR	rs10423928	TT

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

may impair blood sugar control and contribute to diabetes, and may worsen the effectiveness of Retatrutide [R, R].

The *TCF7L2* gene, previously known as TCF4, codes for a vital transcription factor. It controls the expression of other genes in digestive organs such as the gut and pancreas, with a particular role in glucose metabolism and insulin secretion [R, R].

The two main *TCF7L2* variants are **rs7903146 and rs12255372**. They are almost always inherited together, which means they act as a single genetic factor. **Their "T" alleles have one of the strongest known links with type 2 diabetes**, possibly weakening Retatrutide's effect [R, R].

The *KCNJ11* and *KCNQ1* genes are involved in the formation of distinct channels found on the surface of pancreatic cells. These passageways allow positively charged potassium ions to pass from the inside of pancreatic cells to the outside, thus leaving a negative charge on the inner surface of the cells [R, R, R].

The following variants of *KCNJ11* and *KCNQ1* may decrease the production or activity of potassium channels, leading to overactivity of pancreatic cells. This can result in excess release of insulin and low blood sugar levels. In the long run, high insulin levels can result in decreased insulin receptor sensitivity and worse response to Retatrutide [R, R, R]:

- 'C' of **rs5215**
- 'T' of **rs5219**
- 'C' of **rs2237892**
- 'C' of **rs2237897**

The *PPARG* gene encodes a protein called PPAR-γ (peroxisome proliferator-activated receptor-gamma). PPAR-γ affects metabolic health and response to diet, acting as a master regulator between nutrient intake, weight control, fat burning, and insulin sensitivity [R, R].

Out of the different SNPs in the *PPARG* gene, researchers have mostly focused on **rs1801282** (*Pro12Ala*). Its 'G' allele changes one amino acid in the PPAR-γ structure, reducing its ability to activate target genes. This variant has been associated with an increased risk of obesity and may reduce the fat-burning effects of Retatrutide [R, R].

FTO is one of the best-studied genes when it comes to body weight and obesity, hence the name: *fat mass and obesity-associated gene*. It's the first discovered genetic link to obesity

and continues to be the gene with the largest known effect on body weight to this day [R, R, R].

A SNP in this gene, **rs9939609**, has shown a robust association with obesity across different ages and ethnic groups. Carriers of the minor 'A' allele tend to gain more weight and have higher rates of obesity. They may also show worse response to Retatrutide [R, R, R, R].

The *MC4R* gene encodes the melanocortin 4 (MC4) receptor, which binds alpha-melanocyte-stimulating hormone or a-MSH. The primary location of this receptor is the brain, more precisely the hypothalamus, where it controls food intake, metabolism, reproductive behavior, and more [R].

The most studied SNP near the *MC4R* gene is **rs17782313**. The **"C" allele** is linked to obesity and overeating, possibly because it reduces *MC4R* expression or activity [R, R, R, R, R].

Another *MC4R* variant potentially linked to decreased activity, and thus reduced effectiveness of Retatrutide, is 'A' of **rs12970134** [R, R, R].

The *ADIPOQ* gene encodes the protein hormone adiponectin. This hormone is the most abundant gene product of fat cells and acts as a messenger molecule in other tissues, especially in the muscles and liver [R, R].

The following variants have been associated with obesity, insulin resistance, type 2 diabetes, and metabolic syndrome [R, R, R, R]:

- 'T' of **rs1501299**
- 'A' of **rs17300539**
- 'G' of **rs2241766**

Finally, the *FABP2* gene encodes a protein called fatty acid binding protein 2 (FABP2 or I-FABP) that is found throughout the whole intestine and may help with the body's energy balance. FABP2 absorbs and transports long-chain fatty acids, and helps produce triglyceride-rich lipoproteins. It also senses and adjusts how many fat molecules are available for energy production [R, R].

The best-researched *FABP2* polymorphism is **rs1799883**, also called Ala54Thr. Its minor 'T' allele encodes a protein with an amino acid substitution that increases its affinity for long-chain fatty acids. As a result, the mutated version of this protein increases intestinal fatty acid absorption while reducing insulin

sensitivity. This variant, shown to worsen the effectiveness of a low-glycemic-index diet, may also impair response to Retatrutide [R, R].

Response To Tirzepatide (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Tirzepatide.*

Individual variation in response to Tirzepatide may be influenced by genes involved in incretin signaling, insulin secretion, glucose metabolism, and appetite regulation.

The *GLP1R* gene encodes a receptor for GLP-1, a hormone that plays a pivotal role in regulating blood sugar levels. GLP-1 is produced in the gut in response to food intake and lowers blood sugar by enhancing insulin, lowering glucagon, slowing stomach emptying, and reducing appetite [R, R, R, R].

The minor 'A' allele of **rs6923761** (Gly168Ser), which presumably encodes an overactivated version of the protein, has been associated with better anthropometric parameters (such as weight, BMI, and fat mass) and has been associated with greater weight loss but reduced metabolic benefits in response to GLP1R inhibitors such as liraglutide and semaglutide [R, R, R, R, R, R, R, R].

Other variants of this gene associated with better response to GLP1R inhibitors include:
'T' of **rs10305420** [R, R, R]
'G' of **rs3765467** [R]

The *GIPR* gene encodes a protein that plays an important role in insulin production in the body by acting as a receptor for the GIP incretin protein. When glucose levels become high, such as after a meal, GIP is secreted from special intestinal cells called K cells. This newly secreted GIP binds to GIPR located on the pancreas, which causes insulin secretion [R, R].

The main variants in the *GIPR* gene are **rs2287019** and **rs10423928**. They are almost always inherited together, meaning that you will most likely have either both or none of them. The minor alleles, rs2287019–**T** and rs10423928–**A**, may **reduce insulin secretion** after a meal. This

TYPICAL RESPONSE

Predisposed to a typical response to Tirzepatide based on 9 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GLP1R	rs6923761	GG
CDKN1C	rs2237892	CC
CDKN1C	rs2237897	CC
GLP1R	rs10305420	CT
TCF7L2	rs7903146	CT
TCF7L2	rs12255372	GT
ADIPOQ	rs2241766	GT
GLP1R	rs3765467	GG
FTO	rs9939609	TT
KCNJ11	rs5219	CC
KCNJ11	rs5215	TT
GIPR	rs2287019	CC
PPARG	rs1801282	CC
FABP2	rs1799883	CC
MC4R	rs17782313	TT
RFC4	rs17300539	GG
ST6GAL1	rs1501299	GG
MC4R	rs12970134	GG
GIPR	rs10423928	TT

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

may impair blood sugar control and contribute to diabetes, and may worsen the effectiveness of Tirzepatide [R, R].

The *TCF7L2* gene, previously known as TCF4, codes for a vital transcription factor. It controls the expression of other genes in digestive organs such as the gut and pancreas, with a particular role in glucose metabolism and insulin secretion [R, R].

The two main *TCF7L2* variants are **rs7903146 and rs12255372**. They are almost always inherited together, which means they act as a single genetic factor. **Their "T" alleles have one of the strongest known links with type 2 diabetes**, possibly weakening Tirzepatide's effect [R, R].

The *KCNJ11* and *KCNQ1* genes are involved in the formation of distinct channels found on the surface of pancreatic cells. These passageways allow positively charged potassium ions to pass from the inside of pancreatic cells to the outside, thus leaving a negative charge on the inner surface of the cells [R, R, R].

The following variants of *KCNJ11* and *KCNQ1* may decrease the production or activity of potassium channels, leading to overactivity of pancreatic cells. This can result in excess release of insulin and low blood sugar levels. In the long run, high insulin levels can result in decreased insulin receptor sensitivity and worse response to Tirzepatide [R, R, R]:

- 'C' of **rs5215**
- 'T' of **rs5219**
- 'C' of **rs2237892**
- 'C' of **rs2237897**

The *PPARG* gene encodes a protein called PPAR-γ (peroxisome proliferator-activated receptor-gamma). PPAR-γ affects metabolic health and response to diet, acting as a master regulator between nutrient intake, weight control, fat burning, and insulin sensitivity [R, R].

Out of the different SNPs in the *PPARG* gene, researchers have mostly focused on **rs1801282** (*Pro12Ala*). Its 'G' allele changes one amino acid in the PPAR-γ structure, reducing its ability to activate target genes. This variant has been associated with an increased risk of obesity and may reduce the fat-burning effects of Tirzepatide [R, R].

FTO is one of the best-studied genes when it comes to body weight and obesity, hence the name: *fat mass and obesity-associated gene*. It's the first discovered genetic link to obesity

and continues to be the gene with the largest known effect on body weight to this day [R, R, R].

A SNP in this gene, **rs9939609**, has shown a robust association with obesity across different ages and ethnic groups. Carriers of the minor 'A' allele tend to gain more weight and have higher rates of obesity. They may also show worse response to Tirzepatide [R, R, R, R].

The *MC4R* gene encodes the melanocortin 4 (MC4) receptor, which binds alpha-melanocyte-stimulating hormone or a-MSH. The primary location of this receptor is the brain, more precisely the hypothalamus, where it controls food intake, metabolism, reproductive behavior, and more [R].

The most studied SNP near the *MC4R* gene is **rs17782313**. The **"C" allele** is linked to obesity and overeating, possibly because it reduces *MC4R* expression or activity [R, R, R, R, R].

Another *MC4R* variant potentially linked to decreased activity, and thus reduced effectiveness of Tirzepatide, is 'A' of **rs12970134** [R, R, R].

The *ADIPOQ* gene encodes the protein hormone adiponectin. This hormone is the most abundant gene product of fat cells and acts as a messenger molecule in other tissues, especially in the muscles and liver [R, R].

The following variants have been associated with obesity, insulin resistance, type 2 diabetes, and metabolic syndrome [R, R, R, R]:

- 'T' of **rs1501299**
- 'A' of **rs17300539**
- 'G' of **rs2241766**

Finally, the *FABP2* gene encodes a protein called fatty acid binding protein 2 (FABP2 or I-FABP) that is found throughout the whole intestine and may help with the body's energy balance. FABP2 absorbs and transports long-chain fatty acids, and helps produce triglyceride-rich lipoproteins. It also senses and adjusts how many fat molecules are available for energy production [R, R].

The best-researched *FABP2* polymorphism is **rs1799883**, also called Ala54Thr. Its minor 'T' allele encodes a protein with an amino acid substitution that increases its affinity for long-chain fatty acids. As a result, the mutated version of this protein increases intestinal fatty acid absorption while reducing insulin

sensitivity. This variant, shown to worsen the effectiveness of a low-glycemic-index diet, may also impair response to Tirzepatide [R, R].

Response To 5-Amino-1MQ (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of 5-amino-1MQ.*

Response to 5-amino-1MQ may be influenced by variants in *NNMT* and related metabolic genes, as well as genes involved in NAD+ metabolism and mitochondrial function. These variants can modify the drug's effect on metabolism, energy efficiency, and aging-related pathways.

The *NNMT* gene encodes an enzyme called nicotinamide N-methyltransferase that metabolizes drugs and other xenobiotics in the liver by N-methylation using SAM-e as a methyl donor [R].

The best-researched *NNMT* polymorphism is **rs694539**. Its minor 'T' allele, which presumably increases NNMT levels, has been associated with an elevated homocysteine levels and an increased risk of conditions such as congenital heart disease, acute lymphoblastic leukemia, bipolar disorder, migraines, and schizophrenia. Because 5-amino-1MQ blocks NNMT, carriers of this variant may show a weaker response to the peptide [R, R, R, R, R, R].

The *NAMPT* gene encodes an enzyme called nicotinamide phosphoribosyltransferase that catalyzes the condensation of nicotinamide with 5-phosphoribosyl-1-pyrophosphate to yield nicotinamide mononucleotide, one step in the biosynthesis of NAD+ [R].

The following variants have been associated with an increased NAMPT activity and higher NAD+ levels, potentially boosting the effects of 5-amino-1MQ:

- 'T' of **rs1319501** [R]
- 'C' of **rs3801266** [R, R]

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].

TYPICAL RESPONSE

Predisposed to a typical response to 5-amino-1MQ based on 13 genetic variants we looked at

79th

PERCENTILE

Your risk is **greater than 79% of the population** and **lower than 21% of the population.**

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
NAMPT	rs3801266	TT
SIRT1	rs7895833	AA
SIRT3	rs11555236	CC
SIRT3	rs4980329	CC
SIRT1	rs12778366	TC
SIRT1	rs3758391	CT
SIRT1	rs7896005	GA
SIRT3	rs3782118	CT
ZBTB16	rs694539	CC
NAMPT	rs1319501	TT
IFITM2	rs28365927	GG
SIRT1	rs2273773	TT
RIC8A	rs185277566	CC
SIRT3	rs11246020	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

A handful of studies have shown that people with certain *SIRT1* variants linked to increased activity of these genes are more likely to live long lives. Carriers may also respond better to 5-amino-1MQ. Some of these variants include:

- 'C' of **rs12778366** [R, R]
- 'G' of **rs7895833** [R, R]
- 'T' of **rs3758391** [R, R, R]
- 'A' of **rs7896005** [R, R, R]
- 'C' of **rs2273773** [R, R, R]

The *SIRT3* gene encodes another sirtuin protein [R, R, R].

Variants with increased *SIRT3* expression or activity have been linked to increased longevity and may enhance the effects of 5-amino-1MQ. Some of them include:

- 'A' of **rs11555236** [R, R, R]
- 'T' of **rs4980329** [R, R, R]
- 'C' of **rs11246020** [R, R, R, R]
- 'G' of **rs28365927** [R, R]
- 'C' of **rs185277566** [R, R]
- 'T' of **rs3782118** [R]

Response To AOD-9604 (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of AOD-9604.*

The effectiveness of AOD-9604 in promoting fat loss and supporting metabolism may be influenced by genetic variants affecting fat metabolism, growth hormone signaling, and receptor sensitivity.

The *GHR* gene encodes the growth hormone receptor, found in the outer membrane of cells and most abundant in the liver [R].

Specific genetic variants in this gene may lead to altered receptor sensitivity and impact the efficacy of AOD-9604 therapy. Some key variants associated with increased sensitivity to growth hormone, leading to a potentially higher response to AOD-9604, include:

- 'T' of **rs33972388** [R]
- 'A' of **rs1345825** [R]
- 'C' of **rs6873545** [R, R, R, R]
- 'A' of **rs6184** (Pro579Thr) [R, R]
- 'A' of **rs6180** (Ile544Leu) [R, R, R]
- 'G' of **rs2910875** [R]
- 'C' of **rs17574650** [R]
- 'C' of **rs7736209** [R]
- 'T' of **rs2972781** [R]
- 'G' of **rs4273617** [R]
- 'C' of **rs55681913** [R, R]
- 'T' of **rs78095808** [R]
- 'G' of **rs2910875** [R]
- 'G' of **rs62372052** [R]
- 'G' of **rs12654242** [R]
- 'G' of **rs2972770** [R]
- 'A' of **rs10066141** [R]
- 'G' of **rs62372052** [R]
- 'C' of **rs2973018** [R]
- 'C' of **rs16872401** [R]

The *ADRB3* gene encodes the beta-3 adrenergic receptor. This receptor binds catecholamines and activates the sympathetic

BETTER RESPONSE

Predisposed to a better response to AOD-9604 based on 22 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
GHR	rs6184	CC
CCDC152	rs17574650	AA
CCDC152	rs55681913	TT
GHR	rs78095808	CC
CCDC152	rs62372052	AA
CCDC152	rs1345825	AC
GHR	rs6873545	CT
GHR	rs4273617	GA
CCDC152	rs12654242	GA
OXCT1	rs16872401	TC
LEPR	rs1137101	AG
UCP1	rs1800592	TC
GHR	rs7736209	CC
GHR	rs6180	AA
/	rs33972388	TT
CCDC152	rs2973018	CC
CCDC152	rs2972781	TT
CCDC152	rs2972770	GG
GHR	rs2910875	GG
CCDC152	rs10066141	AA
FTO	rs9939609	TT
ADRB3	rs4994	AA
PPARG	rs1801282	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

nervous system by increasing cAMP levels [R].

Over 100 studies have examined the relationship between one *ADRB3* variant—**rs4994** or Trp64Arg— and body-weight measures. The A>G switch at rs4994 changes one amino acid in the beta-3 receptor structure. The “mutant” receptor had a reduced ability to produce cAMP and burn fat in test tubes. This variant may reduce the fat-burning effects of AOD-9604 [R, R, R].

The *LEPR* gene encodes the leptin receptor and is a prime candidate gene for susceptibility to leptin resistance. Most researchers argue that people with fewer or less sensitive leptin receptors would be more likely to develop resistance and be overweight [R, R].

Many *LEPR* variants are currently under investigation for their possible link to leptin resistance and obesity. The most important one is **rs1137101**. The ‘G’ variant likely reduces the number or activity of leptin receptors, potentially contributing to leptin resistance, and may worsen response to AOD-9604 [R, R].

FTO is one of the best-studied genes when it comes to body weight and obesity, hence the name: *fat mass and obesity-associated gene*. It’s the first discovered genetic link to obesity and continues to be the gene with the largest known effect on body weight to this day [R, R, R].

A SNP in this gene, **rs9939609**, has shown a robust association with obesity across different ages and ethnic groups. Carriers of the minor 'A' allele tend to gain more weight and have higher rates of obesity. They may also show worse response to AOD-9604 [R, R, R, R].

The *PPARG* gene encodes a protein called PPAR-γ (peroxisome proliferator-activated receptor-gamma). PPAR-γ affects metabolic health and response to diet, acting as a master regulator between nutrient intake, weight control, fat burning, and insulin sensitivity [R, R].

Out of the different SNPs in the *PPARG* gene, researchers have mostly focused on **rs1801282** (*Pro12Ala*). Its ‘G’ allele changes one amino acid in the PPAR-γ structure, reducing its ability to activate target genes. This variant has been associated with an increased risk of obesity, and may reduce the fat-burning effects of AOD-9604 [R, R].

Finally, 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].

One of the best-studied SNPs in the *UCP1* gene is **rs1800592** (also known as the “-3826 A>G” polymorphism). Its '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). Carriers of this variant may respond better to AOD-9604 [R, R].

Response To MOTS-C (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of MOTS-c.*

MOTS-c exerts its effects primarily via mitochondrial and nuclear signaling pathways, including AMPK activation, insulin signaling, and mitochondrial metabolism genes. Variants in these genes may influence individual responsiveness.

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].

A handful of studies have shown that people with certain *SIRT1* variants linked to increased activity of these genes are more likely to live long lives. Carriers may also respond better to MOTS-c. Some of these variants include:

- 'C' of **rs12778366** [R, R]
- 'G' of **rs7895833** [R, R]
- 'T' of **rs3758391** [R, R, R]
- 'A' of **rs7896005** [R, R, R]
- 'C' of **rs2273773** [R, R, 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 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].

The *SOD2* gene has many described polymorphisms. Among them, rs4880 has received the spotlight in SOD2 research. Its minor allele 'G' is associated with decreased activity and worse protection against oxidative stress. This variant may also worsen response to MOTS-c [R].

BETTER RESPONSE

Predisposed to a better MOTS-c response based on 24 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SIRT1	rs7895833	AA
SOD2	rs4880	GG
CAT	rs769217	CC
NFE2L2	rs35652124	TT
NFE2L2	rs6726395	AA
NFE2L2	rs1806649	CC
SIRT1	rs12778366	TC
SIRT1	rs3758391	CT
SIRT1	rs7896005	GA
CAT	rs1001179	TC
CAT	rs7943316	TA
PPARGC1A	rs8192678	TC
IKZF4	rs10876864	GA
SUOX	rs1131017	CG
RPS26	rs705702	AA
NFE2L2	rs6721961	GG
AGPS	rs2364723	GG
SIRT1	rs2273773	TT
AGPS	rs1962142	GG
NQO1	rs1800566	GG
HNRNPA3	rs13001694	GG
NQO1	rs1131341	GG
GPX1	rs1050450	GG
AGPS	rs10497511	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

The *CAT* gene encodes subunits of catalase, a key antioxidant enzyme in the body's defense against oxidative stress. Four identical subunits, each attached to an iron-containing molecule called a heme group, form the functional catalase enzyme [R].

The best-characterized *CAT* polymorphism is **rs1001179**, commonly referred to as -262G>A. Its minor 'T' allele may increase *CAT* expression but decrease it in people with conditions such as type 2 diabetes or chronic hepatitis C. Other well-known CAT variants with increased activity include 'T' of **rs769217** and 'A' of **rs7943316.** They may enhance the effects of MOTS-c [R, R, R, R, R].

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].

The best-researched *PPARGC1A* polymorphism is **rs8192678**. Its minor 'T' allele decreases *PPARGC1A* expression and PGC-1α levels in the muscles, potentially worsening response to MOTS-c [R, R, R].

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].

Research has identified several variations in the NFE2L2 gene that can reduce the expression and activity of NRF2. Researchers have claimed that reduced NRF2 impairs the body's ability to detox and defend itself from oxidative stress, which can ultimately lead to diminished response to MOTS-c. Some of these variants include [R, R]:

- 'T' of **rs35652124**
- 'T' of **rs6721961**
- 'A' of **rs6726395**
- 'C' of **rs2364723**
- 'G' of **rs10497511**
- 'A' of **rs13001694**
- 'C' of **rs1806649**
- 'A' of **rs1962142**

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 detox quinones [R, R].

Certain *NQO1* gene variants may impair the body's ability to detox by reducing the activity of this enzyme. Based on this mechanism, they may also reduce responsiveness to MOTS-c [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].

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

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].

One study found a direct link between a common *GPX1* variant and human longevity. The heterozygous **genotype 'AG' at rs1050450 was significantly more common in the very elderly** than in the general population. Other studies have strongly suggested that the **'G' allele at rs1050450 confers higher GPx activity**. Carriers may respond better to MOTS-c [R, R, R].

The *SUOX* gene encodes an enzyme called sulfite oxidase, which is found in the intermembrane space of the mitochondria. SUOX plays a key role in the transsulfuration pathway of detoxification, where it's involved in the final breakdown step of sulfur-containing amino acids such as cysteine and methionine. SUOX converts sulfite to sulfate, a less toxic compound that can be excreted by the body, which is the last step in the breakdown of these amino acids [R].

The best-researched *SUOX* polymorphism is **rs705702**. Its minor 'G' allele has been associated with increased oxidative stress and homocysteine buildup due to reduced SUOX activity, which may reduce the effects of MOTS-c. Other variants with reduced activity include 'G' of **rs10876864** and 'G' of **rs1131017** [R, R, R, R, R, R, R, R, R, R, R, R].

Response To SS-31 (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of SS-31.*

SS-31's effectiveness depends on mitochondrial integrity and ROS-handling capacity. Genetic variation in genes involved in mitochondrial function and oxidative stress response may influence responsiveness.

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].

A handful of studies have shown that people with certain *SIRT1* variants linked to increased activity of these genes are more likely to live long lives. Carriers may also respond better to SS-31. Some of these variants include:

- 'C' of **rs12778366** [R, R]
- 'G' of **rs7895833** [R, R]
- 'T' of **rs3758391** [R, R, R]
- 'A' of **rs7896005** [R, R, R]
- 'C' of **rs2273773** [R, R, 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 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].

The *SOD2* gene has many described polymorphisms. Among them, rs4880 has received the spotlight in SOD2 research. Its minor allele 'G' is associated with decreased activity and worse protection against oxidative stress. This variant may also worsen response to SS-31 [R].

BETTER RESPONSE

Predisposed to a better SS-31 response based on 24 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SIRT1	rs7895833	AA
SOD2	rs4880	GG
CAT	rs769217	CC
NFE2L2	rs35652124	TT
NFE2L2	rs6726395	AA
NFE2L2	rs1806649	CC
SIRT1	rs12778366	TC
SIRT1	rs3758391	CT
SIRT1	rs7896005	GA
CAT	rs1001179	TC
CAT	rs7943316	TA
PPARGC1A	rs8192678	TC
IKZF4	rs10876864	GA
SUOX	rs1131017	CG
RPS26	rs705702	AA
NFE2L2	rs6721961	GG
AGPS	rs2364723	GG
SIRT1	rs2273773	TT
AGPS	rs1962142	GG
NQO1	rs1800566	GG
HNRNPA3	rs13001694	GG
NQO1	rs1131341	GG
GPX1	rs1050450	GG
AGPS	rs10497511	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

The *CAT* gene encodes subunits of catalase, a key antioxidant enzyme in the body's defense against oxidative stress. Four identical subunits, each attached to an iron-containing molecule called a heme group, form the functional catalase enzyme [R].

The best-characterized *CAT* polymorphism is **rs1001179**, commonly referred to as -262G>A. Its minor 'T' allele may increase *CAT* expression but decrease it in people with conditions such as type 2 diabetes or chronic hepatitis C. Other well-known CAT variants with increased activity include 'T' of **rs769217** and 'A' of **rs7943316.** They may enhance the effects of SS-31 [R, R, R, R, R].

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].

The best-researched *PPARGC1A* polymorphism is **rs8192678**. Its minor 'T' allele decreases *PPARGC1A* expression and PGC-1α levels in the muscles, potentially worsening response to SS-31 [R, R, R].

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].

Research has identified several variations in the NFE2L2 gene that can reduce the expression and activity of NRF2. Researchers have claimed that reduced NRF2 impairs the body's ability to detox and defend itself from oxidative stress, which can ultimately lead to diminished response to SS-31. Some of these variants include [R, R]:

- 'T' of **rs35652124**
- 'T' of **rs6721961**
- 'A' of **rs6726395**
- 'C' of **rs2364723**
- 'G' of **rs10497511**
- 'A' of **rs13001694**
- 'C' of **rs1806649**
- 'A' of **rs1962142**

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 detox quinones [R, R].

Certain *NQO1* gene variants may impair the body's ability to detox by reducing the activity of this enzyme. Based on this mechanism, they may also reduce responsiveness to SS-31 [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].

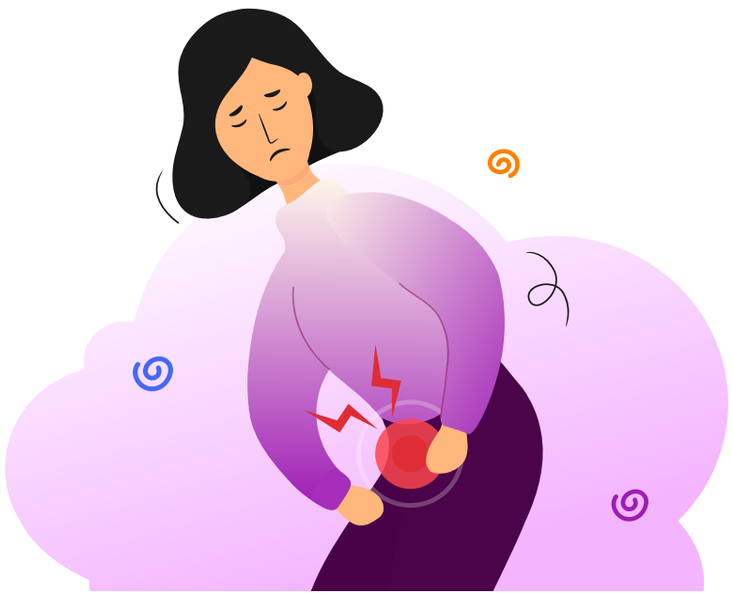
Another major one is **NQO1*3**, which corresponds to **rs1131341-A**. It leads to a slower conversion of quinone into its safer metabolites [R].

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].

One study found a direct link between a common *GPX1* variant and human longevity. The heterozygous **genotype 'AG' at rs1050450 was significantly more common in the very elderly** than in the general population. Other studies have strongly suggested that the **'G' allele at rs1050450 confers higher GPx activity**. Carriers may respond better to SS-31 [R, R, R].

The *SUOX* gene encodes an enzyme called sulfite oxidase which is found in the intermembrane space of the mitochondria. SUOX plays a key role in the transsulfuration pathway of detoxification, where it's involved in the final breakdown step of sulfur-containing amino acids such as cysteine and methionine. SUOX converts sulfite to sulfate, a less toxic compound that can be excreted by the body, which is the last step in the breakdown of these amino acids [R].

The best-researched *SUOX* polymorphism is **rs705702**. Its minor 'G' allele has been associated with increased oxidative stress and homocysteine buildup due to reduced SUOX activity, which may reduce the effects of SS-31. Other variants with reduced activity include 'G' of **rs10876864** and 'G' of **rs1131017** [R, R, R, R, R, R, R, R, R, R, R, R].



Inflammation & Immune Modulation

Your immune system's ability to defend against pathogens while maintaining balance is essential for overall health and resilience. This section explores peptides that help regulate inflammation, enhance immune coordination, and protect cellular integrity — including Thymosin Alpha-1, Thymogen, KPV, VIP, and Larazotide. These compounds support immune signaling, promote tissue healing, and help modulate overactive or suppressed immune responses. Understanding how they work can offer insights into improving recovery from illness, reducing chronic inflammation, and supporting long-term immune balance.

WORSE RESPONSE

Response to KPV (Hypothesis)

Predisposed to a worse response to KPV

WORSE RESPONSE

Response to Larazotide (Hypothesis)

Predisposed to a worse response to Larazotide

TYPICAL RESPONSE

Response to Thymosin Alpha-1 (Hypothesis)

Predisposed to a typical response to Thymosin alpha-1

TYPICAL RESPONSE

Response to Thymogen (Hypothesis)

Predisposed to a typical response to Thymogen

TYPICAL RESPONSE

Response to Thymalin (Hypothesis)

Predisposed to a typical response to Thyamlin

TYPICAL RESPONSE

Response to ARA 290 (Hypothesis)

Predisposed to a typical response to ARA 290

TYPICAL RESPONSE

Response to LL-37 (Hypothesis)

Predisposed to a typical response to LL-37

TYPICAL RESPONSE

Response to VIP (Hypothesis)

Predisposed to a typical response to VIP

Response To KPV (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of KPV.*

The response to KPV therapy may vary based on an individual's genetic makeup, particularly genes involved in inflammation regulation, immune response, and tissue repair. As a peptide with immune-modulating properties, KPV's effects can be influenced by genetic variants that regulate cytokine production, immune cell recruitment, and wound healing processes.

The *TNF* gene encodes a protein called tumor necrosis factor-alpha (TNF-alpha or cachexin). TNF-alpha plays a central role in the immune response and inflammation, and it has been implicated in a wide variety of inflammatory disorders [R].

The **rs1800629** polymorphism (also known as *TNF*-308) is one of the most researched SNPs in the *TNF* gene. **The 'A' allele is associated with 6-7 times higher levels of TNF-alpha.** This may result in a stronger anti-inflammatory response when treated with KPV [R].

Other variants that may increase TNF levels include:

- 'C' of **rs1799964** [R]
- 'C' of **rs1799724** [R]

The *TLR4* gene codes for toll-like receptor 4. This protein jump-starts the innate immune response, which is a mechanism our bodies have for fighting pathogens we have never encountered before. TLR4 recognizes foreign compounds on the surface of invading pathogens (usually bacteria) and activates the immune response. As part of this process, it increases inflammation through NF-κB and cytokines like IL-1 and IL-12 [R, R].

The best-researched TLR4 variant is **rs4986791**. Its minor 'T' allele is believed to increase TLR4 activation based on its pro-inflammatory effects, possibly increasing the effectiveness of KPV against skin infections and inflammation [R].

WORSE RESPONSE

Predisposed to a worse response to KPV based on 15 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TNF	rs1800629	GG
TLR4	rs4986791	CC
TMEM91	rs12461895	AA
TGFB1	rs73045269	CC
TNF	rs1799964	TT
TLR7	rs3853839	C
IL19	rs1800896	CT
IL19	rs1800890	TA
IL19	rs3024505	AG
TNF	rs1799724	TC
IL19	rs1800872	GG
IL10	rs1800871	GG
TGFB1	rs1800472	GG
TLR7	rs179010	C
TLR7	rs179008	A

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

The *TLR7* gene codes for toll-like receptor 7. This protein is also part of the innate immune system and detects single-stranded RNA viruses, including coronaviruses and influenza A. It's primarily found in immune cells like B-cells and dendritic cells, and when activated, it triggers the body's initial immune response, including type I interferon production [R].

Variants with increased TLR7 activity, potentially improving response to KPV, include:

- 'A' of **rs179008** [R]
- 'G' of **rs3853839** [R]
- 'T' of **rs179010** [R, R, R]

The *IL10* gene codes for interleukin-10 (also abbreviated as IL-10), a cytokine with a complex relationship with inflammation. Most of the time, IL-10 is **anti-inflammatory** and suppresses the activity of Th1 cells, Th2 cells, neutrophils, macrophages, and natural killer cells [R, R, R].

Variants of this gene linked to a more anti-inflammatory profile may enhance the tissue-healing effects of KPV. These include [R, R, R, R, R, R, R]:

- 'C' of **rs1800896**
- 'G' of **rs1800871**
- 'T' of **rs1800890**
- 'G' of **rs3024505**
- 'G' of **rs1800872**

Finally, the *TGFB1* gene codes for transforming growth factor beta 1 (TGF-beta1). TGF-beta1 is an immune messenger (cytokine) that plays a role in many cellular processes, including cell growth & division, specialization, movement, and death. By doing so, it controls bone, cartilage, and blood vessel formation, muscle and fat development, wound healing, and inflammation [R].

Certain alleles linked to higher TGFB1 expression may amplify the immunomodulatory effects of KPV. These include:

- 'C' of **rs12461895** [R]
- 'G' of **rs1800472** [R]
- 'T' of **rs73045269** [R]

Response To Larazotide (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Larazotide.*

Larazotide's effectiveness depends on the regulation of intestinal tight junctions, zonulin signaling, and immune activation pathways. Variants in genes that influence these pathways may modulate response to Larazotide.

The *HLA* system has three classes. Specifically, *HLA-DQA1* belongs to class II. The *HLA-DQA1* gene codes for an alpha subunit of the HLA-DQ receptor. The *HLA-DQB1* gene codes for a beta subunit of the HLA-DQ receptor. Together, the two subunits can form a so-called DQ(αβ) heterodimer, which allows white blood cells to present antigens to other cells of the immune system [R, R].

Two alleles — DQA1*0501 and DQB1*0201 — form the DQ2.5 haplotype, which codes for the DQ2.5 receptor on white blood cells. The DQ2.5 receptor binds gluten and presents it to T-helper cells, initiating widespread gut inflammation. The 'T' variant of **rs2187668** serves as a genetic marker for this haplotype. Carriers are more likely to have celiac disease, potentially benfiting from Lazarotide [R, R, R].

The *HP* gene encodes haptoglobin, a protein that binds haptoglobin and carries it back to the liver to be broken down and packaged for removal from the body [R, R].

Zonulin is structurally identical to pre-haptoglobin-2. Variants with increased haptoglobin production, such as 'C' of **rs192626533** and 'A' of **rs12924886**, may correlate with higher intestinal permeability and potentially greater benefit from Larazotide [R].

The *CLDN2* gene encodes claudin 2. Claudins are major integral membrane proteins localized exclusively at tight junctions. The claudin encoded by CLDN2 is mainly expressed in the intestines [R].

WORSE RESPONSE

Predisposed to a worse response to Larazotide based on 14 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
HLA-DQA1	rs2187668	CC
MAGI1	rs17417230	AA
MAGI2	rs6962966	AA
NOD2	rs17221417	CC
NOD2	rs2066844	CC
NOD2	rs2066845	GG
NOD2	rs5743289	CC
TNF	rs1800629	GG
TNF	rs1799964	TT
TNF	rs1799724	TC
ZNF821	rs192626533	CC
PHTF1	rs1343126	TT
HP	rs12924886	AA
MORC4	rs12014762	C

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

The ‘C’ allele of **rs12014762** has been associated with an increased risk of Crohn’s disease, and potentially greater benefits from Larazotide [R].

MAGI genes encode members of the membrane-associated guanylate kinase homologue (MAGUK) family. MAGUK proteins participate in the assembly of multiprotein complexes on the inner surface of the plasma membrane at regions of cell-cell contact [R, R, R].

The following variants in *MAGI* genes have been associated with an increased risk of gut inflammation, potentially enhancing responsiveness to Larazotide:

- ‘C’ of **rs17417230** (*MAGI1*) [R]
- ‘A’ of **rs6962966** (*MAGI2*) [R]
- ‘T’ of **rs1343126** (*MAGI3*) [R]

The *NOD2* gene encodes a pattern recognition receptor that senses proteins belonging to bacteria and activates the immune response. It responds to both potential pathogens and beneficial microbes, keeping up a low level of immune activity at all times and regulating the balance between our bodies and our microbiome [R, R, R].

The strongest effects come from **rs2066844**-T, **rs2066845**-C, and **rs2066847**-C. The unlucky few with the homozygous minor genotype at just one of these variants are up to 35 times more likely to develop Crohn’s disease. Fortunately, the harmful alleles at these SNPs are very rare. One of them (rs2066844) has also been associated with an increased risk of asthma. Other SNPs associated with Crohn’s disease include **rs17221417**-G and **rs5743289**-T. Carriers may benefit more from Larazotide [R, R].

The *TNF* gene encodes a protein called tumor necrosis factor-alpha (TNF-alpha or cachexin). TNF-alpha plays a central role in the immune response and inflammation, and it has been implicated in a wide variety of inflammatory disorders [R].

The **rs1800629** polymorphism (also known as *TNF*-308) is one of the most researched SNPs in the *TNF* gene. **The ‘A’ allele is associated with 6-7 times higher levels of TNF-alpha.** This variant has been associated with an increased risk and severity of IBD. Other variants associated with IBD are ‘C’ at **rs1799964** and ‘C’ at **rs1799724**. Carriers may benefit more from Larzotide [R, R, R, R, R, R, R, R, R].

Response To Thymosin Alpha-1 (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Thymosin alpha-1.*

The effectiveness of TA1 may vary depending on an individual's genetic makeup, particularly in relation to genes that influence immune system function and inflammation regulation. Several genetic factors have been implicated in modulating the response to TA1 therapy, particularly those involved in T-cell activation, cytokine production, and immune system regulation.

Interleukin-6 (IL-6) is a cytokine encoded by the *IL6* gene that has either proinflammatory or anti-inflammatory properties, depending on the circumstances and the tissue that secretes it. It has complicated mechanisms of effect and interacts with many other cytokines and inflammatory signals [R, R].

By far, the most well-researched *IL6* polymorphism is **rs1800795** (also known as the "-174G>C" polymorphism). The major 'G' allele of this SNP has been linked with higher levels of IL-6, while the 'C' allele has been associated with lower IL-6 levels. The major 'G' allele may lead to a more pronounced inflammatory response to TA1, potentially enhancing its immune-stimulating effects [R].

Other variants of this gene linked to an enhanced inflammatory response include:

- 'C' of **rs1524107** [R]
- 'A' of **rs75045751** [R]

The *TNF* gene encodes a protein called tumor necrosis factor-alpha (TNF-alpha or cachexin). TNF-alpha plays a central role in the immune response and inflammation, and it has been implicated in a wide variety of inflammatory disorders [R].

The **rs1800629** polymorphism (also known as *TNF* -308) is one of the most researched SNPs in the *TNF* gene. **The 'A' allele is associated with 6-7 times higher levels of TNF-alpha.** This may result in a stronger immune response when treated with TA1 [R].

TYPICAL RESPONSE

Predisposed to a typical response to Thymosin alpha-1 based on 11 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TNF	rs1800629	GG
STEAP1B	rs75045751	GG
TNF	rs1799964	TT
HLA-DQA2	rs2395185	TG
HLA-DRB5	rs3763313	AA
HLA-DQA1	rs2187668	CC
IL6	rs1524107	CT
TNF	rs1799724	TC
/	rs9271366	AG
HLA-DRB1	rs2516049	CT
IL6	rs1800795	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Other variants that may increase TNF levels include:

- ‘C’ of **rs1799964** [R]
- ‘C’ of **rs1799724** [R]

The *HLA-DRB1* gene encodes the beta chain of the *human leukocyte antigen (HLA)* system, which merges with the alpha chain to form the HLA-DR receptor. Hundreds of DRB1 variants can change the structure and activity of this receptor and thus impact the immune response. Some of them have been associated with autoimmune conditions [R, R, R, R].

Variants linked to a higher expression of the *HLA-DRB1* and *DQA1* genes may enhance the immune-modulating effects of TA1. These include [R, R]:

- ‘G’ of **rs2395185**
- ‘G’ of **rs9271366**
- ‘C’ of **rs3763313**
- ‘T’ of **rs2187668**
- ‘C’ of **rs2516049**

Response To Thymogen (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Thymogen.*

Because Thymogen acts on immune balance and oxidative stress control, individual variability in these processes may determine its effectiveness.

Interleukin-6 (IL-6) is a cytokine encoded by the *IL6* gene that has either proinflammatory or anti-inflammatory properties, depending on the circumstances and the tissue that secretes it. It has complicated mechanisms of effect and interacts with many other cytokines and inflammatory signals [R, R].

By far, the best-researched *IL6* polymorphism is **rs1800795** (also known as the "-174G>C" polymorphism). The major 'G' allele of this SNP has been linked with higher levels of IL-6, while the 'C' allele has been associated with lower IL-6 levels. The major 'G' allele may lead to a more pronounced inflammatory response, potentially boosting the effects of Thymogen [R].

Other variants of this gene linked to greater inflammation include:

- 'C' of **rs1524107** [R]
- 'A' of **rs75045751** [R]

The *IL10* gene codes for interleukin-10 (also abbreviated as IL-10), a cytokine with a complex relationship with inflammation. Most of the time, IL-10 is **anti-inflammatory** and suppresses the activity of Th1 cells, Th2 cells, neutrophils, macrophages, and natural killer cells [R, R, R].

Variants of this gene linked to a more anti-inflammatory profile may lower the immunomodulating effects of Thymogen. These include [R, R, R, R, R, R, R]:

- 'C' of **rs1800896**
- 'G' of **rs1800871**
- 'T' of **rs1800890**
- 'G' of **rs3024505**

TYPICAL RESPONSE

Predisposed to a typical response to Thymogen based on 9 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TLR4	rs4986791	CC
IL10	rs1800871	GG
IL19	rs1800872	GG
TNF	rs1800629	GG
TNF	rs1799964	TT
HLA-DRB5	rs3763313	AA
HLA-DQA1	rs2187668	CC
SOD2	rs4880	GG
GPX1	rs1050450	GG
NFE2L2	rs6721961	GG
AGPS	rs2364723	GG
AGPS	rs10497511	AA
HNRNPA3	rs13001694	GG
AGPS	rs1962142	GG
NR3C1	rs2918419	TT
NR3C1	rs6196	AA
STEAP1B	rs75045751	GG
IL19	rs1800896	CT
IL19	rs1800890	TA
IL19	rs3024505	AG
TNF	rs1799724	TC
HLA-DQA2	rs2395185	TG
/	rs9271366	AG
HLA-DRB1	rs2516049	CT
CAT	rs1001179	TC
CAT	rs7943316	TA
NR3C1	rs852977	GA
NR3C1	rs1866388	GA
NR3C1	rs6188	AC
IL6	rs1524107	CT

- ‘G’ of **rs1800872**

The *TNF* gene encodes a protein called tumor necrosis factor-alpha (TNF-alpha or cachexin). TNF-alpha plays a central role in the immune response and inflammation, and it has been implicated in a wide variety of inflammatory disorders [R].

The **rs1800629** polymorphism (also known as *TNF* -308) is one of the most researched SNPs in the *TNF* gene. **The ‘A’ allele is associated with 6-7 times higher levels of TNF-alpha.** This may result in better restorative effect from Thymogen [R].

Other variants that may increase TNF levels include:

- ‘C’ of **rs1799964** [R]
- ‘C’ of **rs1799724** [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 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].

The *SOD2* gene has many described polymorphisms. Among them, **rs4880** has received the spotlight in SOD2 research. Its minor allele ‘G’ is associated with decreased activity and worse protection against oxidative stress. Carriers may respond better to Thymogen’s antioxidant and cytoprotective actions [R].

The *CAT* gene encodes subunits of catalase, a key antioxidant enzyme in the body’s defense against oxidative stress. Four identical subunits, each attached to an iron-containing molecule called a heme group, form the functional catalase enzyme [R].

The best-characterized *CAT* polymorphism is **rs1001179**, commonly referred to as -262G>A. Its minor ‘T’ allele may increase *CAT* expression but decrease it in people with conditions such as type 2 diabetes or chronic hepatitis C. Other well-known CAT variants with increased activity include ‘T’ of **rs769217** and ‘A’ of **rs7943316.** They may decrease the antioxidant effects of Thymogen [R, R, R, R, R].

The *GPX1* gene helps make glutathione peroxidase (GPx), one of the body’s key antioxidant enzymes. This enzyme converts

GENE	SNP	GENOTYPE
CAT	rs769217	CC
NFE2L2	rs6726395	AA
NR3C1	rs41423247	GG
NFE2L2	rs35652124	TT
NFE2L2	rs1806649	CC
IL6	rs1800795	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

hydrogen peroxide and glutathione into glutathione disulfide and water. By doing so, GPx helps reduce oxidative stress [R].

One study found a direct link between a common *GPX1* variant and human longevity. The heterozygous **genotype 'AG' at rs1050450 was significantly more common in the very elderly** than in the general population. Other studies have strongly suggested that the **'G' allele at rs1050450 confers higher GPx activity**. Carriers may benefit less from Thymogen [R, R, R].

The *TLR4* gene codes for toll-like receptor 4. This protein jump-starts the innate immune response, which is a mechanism our bodies have for fighting pathogens we have never encountered before. TLR4 recognizes foreign compounds on the surface of invading pathogens (usually bacteria) and activates the immune response. As part of this process, it increases inflammation through NF-κB and cytokines like IL-1 and IL-12 [R, R].

The best-researched TLR4 variant is **rs4986791**. Its minor 'T' allele is believed to increase TLR4 activation based on its pro-inflammatory effects, possibly increasing the effectiveness of Thymogen against infections and inflammation [R].

The *HLA-DRB1* gene encodes the beta chain of the *human leukocyte antigen (HLA)* system, which merges with the alpha chain to form the HLA-DR receptor. Hundreds of DRB1 variants can change the structure and activity of this receptor and thus impact the immune response. Some of them have been associated with autoimmune conditions [R, R, R, R].

Variants linked to a higher expression of the *HLA-DRB1* and *DQA1* genes may enhance the immune-modulating effects of Thymogen. These include [R, R]:

- 'G' of **rs2395185**
- 'G' of **rs9271366**
- 'C' of **rs3763313**
- 'T' of **rs2187668**
- 'C' of **rs2516049**

The *NR3C1* gene codes for the glucocorticoid receptor. Upon activation by glucocorticoids (such as the primary stress hormone cortisol), the glucocorticoid receptor is able to regulate the production of stress-related, inflammatory proteins [R].

Variants that lower the sensitivity or activity of the glucocorticoid receptor, thus resulting in dysregulated HPA axis activity, excess

cortisol release, and inflammation, have been associated with chronic fatigue syndrome and may worsen the response to Thymogen. They include:

- 'A' at **rs852977** [R, R, R]
- 'A' at **rs1866388** [R, R, R]
- 'C' at **rs6188** [R, R, R]
- 'T' at **rs2918419** [R, R, R]
- 'A' at **rs6196** [R, R, R]
- 'C' at **rs41423247** [R, R]

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].

Research has identified several variations in the NFE2L2 gene that can reduce the expression and activity of NRF2. Researchers have claimed that reduced NRF2 impairs the body's ability to detox and defend itself from oxidative stress. Some of these variants, which may be linked to enhanced benefits from Thymogen, include [R, R]:

- 'T' of **rs35652124**
- 'T' of **rs6721961**
- 'A' of **rs6726395**
- 'C' of **rs2364723**
- 'G' of **rs10497511**
- 'A' of **rs13001694**
- 'C' of **rs1806649**
- 'A' of **rs1962142**

Response To Thymalin (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Thymalin.*

Thymalin's effects on immunity, inflammation, oxidative balance, and cellular longevity mean that genetic variations in these pathways can alter its efficacy.

Interleukin-6 (IL-6) is a cytokine encoded by the *IL6* gene that has either proinflammatory or anti-inflammatory properties, depending on the circumstances and the tissue that secretes it. It has complicated mechanisms of effect and interacts with many other cytokines and inflammatory signals [R, R].

By far, the best-researched *IL6* polymorphism is **rs1800795** (also known as the "-174G>C" polymorphism). The major 'G' allele of this SNP has been linked with higher levels of IL-6, while the 'C' allele has been associated with lower IL-6 levels. The major 'G' allele may lead to a more pronounced inflammatory response, potentially boosting the effects of Thymalin [R].

Other variants of this gene linked to greater inflammation include:

- 'C' of **rs1524107** [R]
- 'A' of **rs75045751** [R]

The *IL10* gene codes for interleukin-10 (also abbreviated as IL-10), a cytokine with a complex relationship with inflammation. Most of the time, IL-10 is **anti-inflammatory** and suppresses the activity of Th1 cells, Th2 cells, neutrophils, macrophages, and natural killer cells [R, R, R].

Variants of this gene linked to a more anti-inflammatory profile may lower the immunomodulating effects of Thymalin. These include [R, R, R, R, R, R, R]:

- 'C' of **rs1800896**
- 'G' of **rs1800871**
- 'T' of **rs1800890**
- 'G' of **rs3024505**

TYPICAL RESPONSE

Predisposed to a typical response to Thyamlin based on 9 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TLR4	rs4986791	CC
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HLA-DQA1	rs2187668	CC
SOD2	rs4880	GG
GPX1	rs1050450	GG
NFE2L2	rs6721961	GG
AGPS	rs2364723	GG
AGPS	rs10497511	AA
HNRNPA3	rs13001694	GG
AGPS	rs1962142	GG
NR3C1	rs2918419	TT
NR3C1	rs6196	AA
STEAP1B	rs75045751	GG
SLC6A3	rs2736108	CC
TERT	rs4449583	CC
TERT	rs2075786	GG
FOXO3	rs12212067	TT
FOXO3	rs12202234	CC
FOXO3	rs17069665	AA
FOXO3	rs9398171	TT
FOXO3	rs3800230	TT
FOXO3	rs9400239	CC
FOXO3	rs479744	GG
FOXO3	rs4946936	CC
IL19	rs1800896	CT
IL19	rs1800890	TA

- ‘G’ of **rs1800872**

The *TNF* gene encodes a protein called tumor necrosis factor-alpha (TNF-alpha or cachexin). TNF-alpha plays a central role in the immune response and inflammation, and it has been implicated in a wide variety of inflammatory disorders [R].

The **rs1800629** polymorphism (also known as *TNF* -308) is one of the most researched SNPs in the *TNF* gene. **The ‘A’ allele is associated with 6-7 times higher levels of TNF-alpha.** This may result in better restorative effect from Thymalin [R].

Other variants that may increase TNF levels include:

- ‘C’ of **rs1799964** [R]
- ‘C’ of **rs1799724** [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 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].

The *SOD2* gene has many described polymorphisms. Among them, **rs4880** has received the spotlight in SOD2 research. Its minor allele ‘G’ is associated with decreased activity and worse protection against oxidative stress. Carriers may respond better to Thymalin’s antioxidant and cytoprotective actions [R].

The *CAT* gene encodes subunits of catalase, a key antioxidant enzyme in the body’s defense against oxidative stress. Four identical subunits, each attached to an iron-containing molecule called a heme group, form the functional catalase enzyme [R].

The best-characterized *CAT* polymorphism is **rs1001179**, commonly referred to as -262G>A. Its minor ‘T’ allele may increase *CAT* expression but decrease it in people with conditions such as type 2 diabetes or chronic hepatitis C. Other well-known CAT variants with increased activity include ‘T’ of **rs769217** and ‘A’ of **rs7943316.** They may decrease the antioxidant effects of Thymalin [R, R, R, R, R].

The *GPX1* gene helps make glutathione peroxidase (GPx), one of the body’s key antioxidant enzymes. This enzyme converts

GENE	SNP	GENOTYPE
IL19	rs3024505	AG
TNF	rs1799724	TC
HLA-DQA2	rs2395185	TG
/	rs9271366	AG
HLA-DRB1	rs2516049	CT
CAT	rs1001179	TC
CAT	rs7943316	TA
NR3C1	rs852977	GA
NR3C1	rs1866388	GA
NR3C1	rs6188	AC
IL6	rs1524107	CT
TERT	rs7705526	CA
TERT	rs2736100	AC
TERT	rs10069690	CT
TERT	rs13167280	AG
TERT	rs2242652	GA
SLC6A3	rs2735940	GA
TERT	rs2853672	AC
TERT	rs2853676	CT
TERT	rs2853677	AG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

hydrogen peroxide and glutathione into glutathione disulfide and water. By doing so, GPx helps reduce oxidative stress [R].

One study found a direct link between a common *GPX1* variant and human longevity. The heterozygous **genotype 'AG' at rs1050450 was significantly more common in the very elderly** than in the general population. Other studies have strongly suggested that the **'G' allele at rs1050450 confers higher GPx activity**. Carriers may benefit less from Thymalin [R, R, R].

The *TLR4* gene codes for toll-like receptor 4. This protein jump-starts the innate immune response, which is a mechanism our bodies have for fighting pathogens we have never encountered before. TLR4 recognizes foreign compounds on the surface of invading pathogens (usually bacteria) and activates the immune response. As part of this process, it increases inflammation through NF-κB and cytokines like IL-1 and IL-12 [R, R].

The best-researched TLR4 variant is **rs4986791**. Its minor 'T' allele is believed to increase TLR4 activation based on its pro-inflammatory effects, possibly increasing the effectiveness of Thymalin against infections and inflammation [R].

The *HLA-DRB1* gene encodes the beta chain of the *human leukocyte antigen (HLA)* system, which merges with the alpha chain to form the HLA-DR receptor. Hundreds of DRB1 variants can change the structure and activity of this receptor and thus impact the immune response. Some of them have been associated with autoimmune conditions [R, R, R, R].

Variants linked to a higher expression of the *HLA-DRB1* and *DQA1* genes may enhance the immune-modulating effects of Thymalin. These include [R, R]:

- 'G' of **rs2395185**
- 'G' of **rs9271366**
- 'C' of **rs3763313**
- 'T' of **rs2187668**
- 'C' of **rs2516049**

The *NR3C1* gene codes for the glucocorticoid receptor. Upon activation by glucocorticoids (such as the primary stress hormone cortisol), the glucocorticoid receptor is able to regulate the production of stress-related, inflammatory proteins [R].

Variants that lower the sensitivity or activity of the glucocorticoid receptor, thus resulting in dysregulated HPA axis activity, excess

cortisol release, and inflammation, have been associated with chronic fatigue syndrome and may worsen response to Thymalin. They include:

- 'A' at **rs852977** [R, R, R]
- 'A' at **rs1866388** [R, R, R]
- 'C' at **rs6188** [R, R, R]
- 'T' at **rs2918419** [R, R, R]
- 'A' at **rs6196** [R, R, R]
- 'C' at **rs41423247** [R, R]

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].

Research has identified several variations in the NFE2L2 gene that can reduce the expression and activity of NRF2. Researchers have claimed that reduced NRF2 impairs the body's ability to detox and defend itself from oxidative stress. Some of these variants, which may be linked to enhanced benefits from Thymalin, include [R, R]:

- 'T' of **rs35652124**
- 'T' of **rs6721961**
- 'A' of **rs6726395**
- 'C' of **rs2364723**
- 'G' of **rs10497511**
- 'A' of **rs13001694**
- 'C' of **rs1806649**
- 'A' of **rs1962142**

The *TERT* gene codes for hTERT, a part of the telomerase enzyme. Telomerase can protect DNA essential to the production of proteins (genes), by adding non-essential DNA (telomeres) to the ends of condensed DNA structures (chromosomes). This ultimately prevents genes from becoming damaged [R].

The *TERT* gene has a lot of known variations. Most people will probably have a mix of variations that increase and decrease relative telomere length, so it's important to look at as many SNPs as possible to see which way you lean. The following variants have been associated with an increased telomere length, potentially enhancing responsiveness to Thymalin [R, R, R, R, R, R]:

- 'T' at **rs2736108**
- 'A' at **rs7705526**

- ‘C’ at **rs2736100**
- ‘A’ at **rs7705526**
- ‘T’ at **rs4449583**
- ‘G’ at **rs33961405**
- ‘C’ at **rs10069690**
- ‘A’ at **rs13167280**
- ‘A’ at **rs2075786**
- ‘G’ at **rs2242652**
- ‘A’ at **rs2735940**
- ‘C’ at **rs2736098**
- ‘A’ at **rs2853669**
- ‘A’ at **rs2853672**
- ‘C’ at **rs2853676**
- ‘A’ at **rs2853677**
- ‘C’ at **rs4975605**

The *FOXO3* gene encodes a transcription factor involved in tumor suppression, immune function, DNA repair, and resistance to oxidative stress, which may explain its link with increased longevity [R, R, R].

A large body of research has linked certain variants in the *FOXO3* gene with longer lifespan, and potentially better response to Thymalin. They include [R, R, R, R, R, R, R]:

- ‘G’ at **rs12212067**
- ‘G’ at **rs768023**
- ‘C’ at **rs2253310**
- ‘A’ at **rs2802288**
- ‘G’ at **rs12202234**
- ‘G’ at **rs17069665**
- ‘C’ at **rs9398171**
- ‘G’ at **rs3800230**
- ‘C’ at **rs1935952**
- ‘T’ at **rs9400239**
- ‘T’ at **rs479744**
- ‘G’ at **rs2802292**
- ‘T’ at **rs4946936**
- ‘A’ at **rs6911407**

Response To ARA 290 (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of ARA 290.*

Response to ARA 290 may be influenced by variants in genes that regulate EPO signaling, cytokine response, and tissue-protective pathways. These variants may alter the peptide's cytoprotective, anti-inflammatory, and neuroprotective effects.

The *EPOR* gene encodes the erythropoietin receptor, which is activated upon binding by EPO. This stimulates a cascade of signals known as the JAK/STAT pathway to promote the development and maturation of red blood cells [R].

The following variants are associated with enhanced EPOR signaling, potentially boosting the effects of ARA 290:

- 'C' of **rs184452209** [R]
- 'A' of **rs370865377** [R]
- 'A' of **rs142094773** [R]

The *CSF2RB* gene encodes a subunit (the common beta chain) of the high-affinity receptor for IL-3, IL-5, and CSF. Together with EPOR, this receptor forms a tissue-protective receptor complex [R].

The following variants have been associated with higher levels of this protein, potentially improving response to ARA 290:

- 'A' of **rs200102736** [R]
- 'T' of **rs531862830** [R]
- 'T' of **rs549001237** [R]
- 'A' of **rs7292430** [R]
- 'T' of **rs76218233** [R]
- 'T' of **rs78549491** [R]
- 'A' of **rs114063448** [R]
- 'C' of **rs139703671** [R]
- 'T' of **rs141641176** [R]
- 'T' of **rs182342872** [R]
- 'G' of **rs184370538** [R]
- 'C' of **rs187683664** [R]

TYPICAL RESPONSE

Predisposed to a typical response to ARA 290 based on 35 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
EPOR	rs142094773	GG
CSF2RB	rs200102736	GG
CSF2RB	rs7292430	GG
CSF2RB	rs114063448	GG
CSF2RB	rs139703671	TT
NCF4	rs141641176	CC
CSF2RB	rs182342872	CC
NCF4	rs35435764	CC
NCF4	rs536391820	GG
C1QTNF6	rs571070978	AA
NCF4	rs142640148	AA
STAT3	rs744166	AA
STAT3	rs1053005	TT
CSF2RB	rs78549491	TC
CSF2RB	rs5756415	CG
CSF2RB	rs1534881	GA
STAT3	rs2293152	GC
JAK2	rs10758669	CA
NCF4	rs76218233	TT
NCF4	rs75218287	AA
NCF4	rs62229117	GG
NCF4	rs550784540	TT
NCF4	rs549001237	TT
NCF4	rs192765222	TT
CSF2RB	rs191574362	CC
CSF2RB	rs190658973	GG
CSF2RB	rs187683664	CC
CSF2RB	rs185893830	TT
/	rs184370538	GG
NCF4	rs144804886	CC

- ‘G’ of **rs190658973** [R]
- ‘C’ of **rs5756415** [R]
- ‘G’ of **rs1534881** [R]
- ‘C’ of **rs191574362** [R]
- ‘T’ of **rs192765222** [R]
- ‘T’ of **rs35435764** [R]
- ‘T’ of **rs536391820** [R]
- ‘T’ of **rs550784540** [R]
- ‘G’ of **rs571070978** [R]
- ‘G’ of **rs62229117** [R]
- ‘A’ of **rs75218287** [R]
- ‘C’ of **rs113609764** [R]
- ‘C’ of **rs117246948** [R]
- ‘G’ of **rs142640148** [R]
- ‘T’ of **rs143948973** [R]
- ‘C’ of **rs144804886** [R]
- ‘T’ of **rs185893830** [R]

GENE	SNP	GENOTYPE
NCF4	rs143948973	TT
IL2RB	rs117246948	CC
NCF4	rs113609764	CC
STAT3	rs4796793	CC
STAT3	rs3816769	TT

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

The *JAK2* gene encodes a protein called ‘Janus kinase 2’ (JAK2). Members of the JAK family activate STAT (‘Signal transducer and activator of transcription’) proteins in response to cytokines. JAK2 recognizes the signal of different cytokines, including IL-6, IL-11, IL-12, IL-22, and IL-23. The subsequent activation of STAT proteins turns “on” the expression of multiple genes, especially those involved in the growth, development, and activation of the immune system cells that fight off infections [R, R, R, R].

The most widely-studied *JAK2* polymorphism is **rs10758669**. **Its minor variant “C” increases the production and activity of the JAK2 protein**, leading to an enhanced immune response and inflammation. On the bright side, carriers may respond better to ARA 290 [R].

The *STAT3* gene codes for one member of a family of proteins called *signal transducer and activator of transcription*. Its main role is to control the expression of genes involved in the growth, development, and activation of immune system cells upon activation by proteins such as JAK. This process is triggered by cytokines such as IL-6, IL-10, IL-21, and IL-23 [R, R, R].

STAT3 variants linked to higher activity are typically linked to an increased risk of autoimmune and inflammatory diseases, but they may also enhance response to ARA 290. Some of these variants include:

- ‘G’ of **rs744166** [R]
- ‘G’ of **rs2293152** [R, R, R]

- ‘C’ of **rs4796793** [R]
- ‘T’ of **rs3816769** [R]
- ‘C’ of **rs1053005** [R, R]

Response To LL-37 (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of LL-37.*

Please note: *Individuals with weakened or compromised immune systems should exercise caution, as LL-37's strong immune-modulating and antimicrobial effects could potentially trigger unwanted immune responses or inflammation.*

TYPICAL RESPONSE

Predisposed to a typical response to LL-37 based on 10 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
ZNF589	rs9844566	CC
TLR4	rs4986791	CC
TLR7	rs3853839	C
IL19	rs1800896	CT
IL19	rs1800890	TA
IL19	rs3024505	AG
IL19	rs1800872	GG
IL10	rs1800871	GG
TLR7	rs179010	C
TLR7	rs179008	A

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

The effectiveness of LL-37 in immune defense and tissue repair can vary based on genetic factors that influence its expression and activity. Several genes regulate LL-37 production, and variations in these genes can impact the peptide's ability to defend against pathogens and promote healing.

The *CAMP* gene encodes the cathelicidin protein. This protein plays an important role in innate immunity defense against viruses. In addition to its antibacterial, antifungal, and antiviral activities, the encoded protein functions in cell chemotaxis, immune mediator induction, and inflammatory response regulation [R].

Carriers of variants resulting in decreased cathelicidin production may benefit more from taking LL-37. For instance, the 'G' allele of **rs9844566** has been associated with lower *CAMP* expression [R].

The *TLR4* gene codes for toll-like receptor 4. This protein jump-starts the innate immune response, which is a mechanism our bodies have for fighting pathogens we have never encountered before. TLR4 recognizes foreign compounds on the surface of invading pathogens (usually bacteria) and activates the immune response. As part of this process, it increases inflammation through NF-κB and cytokines like IL-1 and IL-12 [R, R].

The best-researched TLR4 variant is **rs4986791**. Its minor 'T' allele is believed to increase TLR4 activation based on its pro-inflammatory effects, possibly enhancing the effects of LL-37 [R].

The *TLR7* gene codes for toll-like receptor 7. This protein is also part of the innate immune system and detects single-stranded RNA viruses, including coronaviruses and influenza A. It's primarily found in immune cells like B-cells and dendritic cells, and when activated, it triggers the body's initial immune response, including type I interferon production [R].

Variants with increased TLR7 activity, potentially improving response to LL-37, include:

- 'A' of **rs179008** [R]
- 'G' of **rs3853839** [R]
- 'T' of **rs179010** [R, R, R]

The *IL10* gene codes for interleukin-10 (also abbreviated as IL-10), a cytokine with a complex relationship with inflammation. Most of the time, IL-10 is **anti-inflammatory** and suppresses the activity of Th1 cells, Th2 cells, neutrophils, macrophages, and natural killer cells [R, R, R].

Variants of this gene linked to a more anti-inflammatory profile may enhance the tissue-healing effects of LL-37. These include [R, R, R, R, R, R, R]:

- 'C' of **rs1800896**
- 'G' of **rs1800871**
- 'T' of **rs1800890**
- 'G' of **rs3024505**
- 'G' of **rs1800872**

Response To VIP (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of VIP.*

VIP exerts its effects by binding to its receptors VPAC1 and VPAC2, which tiggers cAMP-mediated signaling and downstream modulation of immune, cardiovascular, and neuronal pathways. Genetic variation in this peptide, these receptors, or downstream effectors such as BDNF may influence responsiveness to VIP therapy.

VIP is encoded by the *VIP* gene. Supplementation with exogenous VIP may be more effective in people carrying variants associated with decreased VIP production, such as [R]:

- 'T' of **rs688136** [R]
- 'T' of **rs35643203** [R]
- 'T' of **rs12201140** [R]

Alternatively, the following variants at other genes have been associated with lower VIP levels [R]:

- 'A' of **rs10754199** (*CHF*)
- 'T' of **rs1354034** (*ARHGEF3*)

The *VIPR1* gene encodes the VPAC1 receptor, which is predominantly expressed in immune cells, lungs, and gut [R].

Variants increasing the expression or activity of this receptor, such as the 'C' allele of **rs896**, may enhance the effectiveness of VIP therapy [R].

BDNF (brain-derived neurotrophic factor) is a component produced mainly in brain cells. It plays many key roles that support your brain's ability to grow and learn. More precisely, BDNF helps stimulate neurogenesis (the production of new nerve cells) and synaptic plasticity (growing new connections between brain cells) [R, R, R, R].

A crucial *BDNF* gene variant is **rs6265**, also known as "**Val66Met**". It may reduce BDNF production, storage, and release in brain cells. As a result, the **"T" ("Met") allele is linked**

TYPICAL RESPONSE

Predisposed to a typical response to VIP based on 10 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
VIP	rs35643203	CC
VIP	rs12201140	AA
CFHR3	rs10754199	GG
BDNF	rs59579819	TT
BDNF	rs80238569	AA
BDNF	rs75945125	TT
ARHGEF3	rs1354034	TC
BDNF	rs6265	TC
SEC22C	rs896	CC
VIP	rs688136	TT

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

to reduced cognitive function. Carriers of this allele may respond worse to VIP [R, R, R, R].

Other variants linked to lower BDNF levels and potentially worse response to VIP include:

- 'T' of **rs59579819** [R]
- 'A' of **rs80238569** [R]
- 'T' of **rs75945125** [R]

Sexual & Reproductive Health

A complex network of hormonal, neurological, and emotional pathways regulates desire, fertility, and bonding, ultimately controlling sexual and reproductive health. This section explores peptides that modulate these interconnected systems — including Bremelanotide, Kisspeptin, Oxytocin, and Melanotan II. These compounds can enhance libido, improve sexual function, and support hormonal balance, while also influencing mood and emotional connection. Understanding how these peptides work provides insight into optimizing reproductive wellness and promoting healthy intimacy.

TYPICAL RESPONSE

Response to Bremelanotide (Hypothesis)

Predisposed to a typical response to bremelanotide

TYPICAL RESPONSE

Response to Kisspeptin (Hypothesis)

Predisposed to a typical response to Kisspeptin

BETTER RESPONSE

Response to Oxytocin (Hypothesis)

Predisposed to a better response to oxytocin

BETTER RESPONSE

Response to Melanotan II (Hypothesis)

Predisposed to a better response to Melanotan II

Response To Bremelanotide (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of bremelanotide.*

The response to bremelanotide, like many other treatments, can be influenced by genetic factors that affect the melanocortin system, neurobiology, and sexual function. Bremelanotide primarily targets melanocortin receptors, particularly MC1R, MC3R, and MC4R, which are involved in sexual arousal and appetite regulation. Variants in these receptors and associated signaling pathways may alter an individual's response to the peptide.

The *MC1R* gene encodes the melanocortin 1 receptor, found on the surfaces of cells called melanocytes. Activation of this receptor by alpha-MSH controls which type of melanin is produced by melanocytes. MC1R is also active in cells other than melanocytes, including cells involved in the body's immune and inflammatory responses [R].

The minor 'T' allele of **rs1805007** likely reduces the production and activity of MC1R, potentially reducing the effectiveness of bremelanotide [R].

The *MC3R* gene encodes the melanocortin 3 receptor, activated by alpha-MSH and other melanocortins. MC3R is primarily expressed in the brain and fat tissue; it controls feeding behavior, fat burning, energy balance, and more [R, R].

Scientists have identified two major SNPs in the *MC3R* gene with a potential impact on body-weight measures: **rs3746619** (Thr6Lys) and **rs3827103** (Val81Ile). They are almost always inherited together, and the studies often refer to them in pairs. Their minor 'A' alleles may reduce MC3R activity, potentially leading to worse response to bremelanotide [R, R, R, R].

The *MC4R* gene encodes the melanocortin 4 (MC4) receptor, which binds alpha-melanocyte-stimulating hormone or a-MSH. The primary location of this receptor is the brain, more precisely

TYPICAL RESPONSE

Predisposed to a typical response to bremelanotide based on 8 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
POMC	rs6713532	TT
POMC	rs934778	AA
MC3R	rs3827103	GG
MC3R	rs3746619	CC
MC1R	rs1805007	CC
MC4R	rs17782313	TT
MC4R	rs12970134	GG
POMC	rs1042571	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

the hypothalamus, where it controls food intake, metabolism, reproductive behavior, and more [R].

The most studied SNP near the *MC4R* gene is **rs17782313**. The **"C" allele** is linked to obesity and overeating, possibly because it reduces *MC4R* expression or activity [R, R, R, R, R].

Another *MC4R* variant potentially linked to decreased activity, and thus reduced effectiveness of bremelanotide, is 'A' of **rs12970134** [R, R, R].

The *POMC* gene encodes proopiomelanocortin, a protein that is cut into multiple other, smaller proteins with specialized functions. More specifically, POMC is divided into adrenocorticotropic hormone (ACTH) and three melanocyte-stimulating hormones (α-, β-, and γ-MSH). POMC helps maintain homeostasis by, among other things, suppressing appetite when we have eaten enough food [R, R].

Three SNPs so far have been associated with higher weight and body fat to varying degrees, likely because they reduce POMC levels. They include [R, R, R, R]:

- 'T' of **rs6713532**
- 'A' of **rs1042571**
- 'G' of **rs934778**

Due to their association with reduced POMC levels, they may also reduce bremelanotide effectiveness.

Response To Kisspeptin (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Kisspeptin.*

Genetic variation in KISS1, KISS1R, and reproductive hormone pathways may influence individual response to Kisspeptin, affecting hormone levels, fertility, and sexual function.

The *KISS1* gene 'KiSS-1 metastasis suppressor' encodes kisspeptin, a peptide that plays a critical role in regulating reproduction. It controls the onset of puberty by stimulating gonadotropin-releasing hormone (GnRH)-induced gonadotropin secretion, which regulates the activation of GnRH neurons [R].

One of the main *KISS1* polymorphisms is **rs4889** (Pro110Arg). Its minor 'G' allele encodes a version of the protein with presumably worse binding affinity for GPR54. As a result, this variant has been associated with lower LH levels and higher PCOS risk. Carriers of this variant may benefit more from synthetic Kisseptin administration [R, R, R].

Another variant, 'A' of **rs372790354**, has also been associated with an increased risk of PCOS and lower kisseptin and LH levels, potentially making carriers more likely to benefit from Kisseptin [R, R, R].

The *KISS1R* gene encodes the KISS1 receptor, also known as GPR54. Upon activation by KIS11, the receptor triggers the release of sex hormones that regulate puberty onset and reproductive function [R].

Variants with impaired KISS1R signaling have been associated with delayed puberty and infertility, and may worsen the effectiveness of exogenous Kisspeptin. Some of these variants include:
'C' of **rs28939719** [R, R]
'C' of **rs104894703** [R, R, R]
'C' of **rs587777844** [R, R]

TYPICAL RESPONSE

Predisposed to a typical response to Kisspeptin based on 9 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
KISS1	rs372790354	GG
KISS1	rs4889	GC
UBA6	rs3756159	GA
LHB	rs3795052	CA
LHB	rs3795050	CA
GYS1	rs144948359	CT
LHB	rs3795047	TA
ESR1	rs2234693	CT
ESR1	rs9340799	GA
GNRH1	rs6185	CC
ARL14EP	rs11031006	GG
ARL14EP	rs11031005	TT
ARL14EP	rs10835638	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

The *GNRH1* gene encodes the gonadotropin-releasing hormone 1 peptide, a crucial regulator of the reproductive axis that transmits kisspeptin's signal to the pituitary [R].

One of the main GNRH1 polymorphisms is **rs6185**. Its 'G' allele encodes a less stable form of the protein that has been associated with an increased risk of PCOS, lower odds of having twins, and later menopause onset. Carriers may show a worse response to Kisspeptin [R, R, R].

Another variant, 'G' of **rs3756159**, has been associated with an increased risk of PCOS and lower baseline LH levels, also suggesting impaired GNRH1 function and worse response to Kisspeptin [R, R].

The *FSHB* gene encodes the beta subunit of follicle-stimulating hormone, a crucial hormone for reproduction that is released by the pituitary gland. FSH stimulates the growth and maturation of eggs in women and sperm in men [R, R, R].

The following variants have been associated with lower FSH levels, potentially reducing the effectiveness of Kisspeptin:

- 'C' of **rs11031005** [R, R]
- 'A' of **rs11031006** [R, R]
- 'T' of **rs10835638** [R]

Similarly, the *LHB* gene encodes the beta subunit of luteinizing hormone. In women, LH controls the menstrual cycle and stimulates ovulation. In men, it signals the testes to produce testosterone. Testosterone, in turn, stimulates sperm production [R, R, R].

Variants decreasing LH production may be associated with a worse response to Kisspeptin. Some of these variants include:

- 'C' of **rs3795052** [R]
- 'C' of **rs3795050** [R]
- 'C' of **rs144948359** [R]
- 'T' of **rs3795047** [R]

Finally, the *ESR1* gene encodes ERα, a nuclear hormone receptor that regulates the expression of genes involved in various physiological processes, including reproductive health, bone density, cardiovascular function, and cancer development. Estrogen binding to ERα influences gene expression, impacting cell growth, differentiation, and metabolism [R].

The two main *ESR1* variants are **rs2234693** (-397T>C or Pvull) and **rs9340799** (-351A>G or Xbal). They are often inherited together, meaning that you will likely carry either none or both. They increase ESR1 expression, which may lead to more pronounced effects of Kisspeptin [R].

Response To Oxytocin (Hypothesis)

Please note: *Some claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants other than those in OXTR have been found to modify the effectiveness of oxytocin.*

The *OXTR* gene is among the best-studied human genes. It helps make **oxytocin receptors** and thus has a crucial impact on the function of this hormone [R].

The most important *OXTR* gene variant is **rs53576**. In people with the "G" allele at this variant, oxytocin receptors seem to work better and bind more of this hormone. This may improve responsiveness to exogenous oxytocin [R].

Another well-researched polymorphism is **rs2254298**. Its minor 'A' allele may increase oxytocin sensitivity and amygdala size, also helping enhance the effects of exogenous oxytocin [R, R, R].

Another variant, 'T' at **rs1042778**, may decrease oxytocin levels. As a result, carriers may respond worse to oxytocin administration [R].

Similarly, the major 'T' allele of **rs13316193** may decrease the number of oxytocin receptors in the brain [R].

Finally, the rare 'A' allele of **rs2268494** may cause lower oxytocin levels or less efficient oxytocin receptors, potentially worsening response to oxytocin [R, R].

The *OXT* gene encodes oxytocin/neurophysin I prepropeptide, a precursor of the hormone oxytocin and its carrier protein, neurophysin I. Both proteins are packaged together into neurosecretory vesicles and transported to the nerve endings in the neurohypophysis, where oxytocin is either stored or secreted into the bloodstream [R].

The following OXT variants have been associated with higher levels of this protein, potentially increasing oxytocin levels [R, R, R, R].

BETTER RESPONSE

Predisposed to a better response to oxytocin based on 19 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
VPS16	rs12625893	GG
CD38	rs114713438	CC
CD38	rs188216762	CC
CD38	rs1004124	CC
CD38	rs75800426	CC
BST1	rs76730281	TT
BST1	rs77197599	TT
OXTR	rs2254298	GA
OXTR	rs1042778	GT
OXTR	rs13316193	CT
OXT	rs877172	GT
OXTR	rs53576	GG
OXTR	rs2268494	TT
FAM200B	rs3796863	TT
MRPS26	rs2740210	CC
CD38	rs188213195	CC
CD38	rs182844391	GG
FAM200B	rs150794852	CC
OXT	rs144102485	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

- ‘A’ of **rs12625893**
- ‘C’ of **rs144102485**
- ‘G’ of **rs877172**
- ‘C’ of **rs2740210**

The *CD38* gene encodes an enzyme that produces and metabolizes the messenger molecule cyclic adenosine 5'-diphosphate-ribose. Importantly, this gene is part of the pathway that releases oxytocin in the brain. More CD38 activity releases more oxytocin and enhances empathy and social communication [R, R, R].

Carriers of the ‘T’ allele at **rs3796863** in this gene release more oxytocin, which may further enhance the effectiveness of oxytocin administration [R].

Other variants of this gene linked ot higher CD38 levels, potentially boosting the effects of exogenous oxytocin, include [R]:

- ‘T’ of **rs114713438**
- ‘T’ of **rs188216762**
- ‘G’ of **rs1004124**
- ‘G’ of **rs182844391**
- ‘C’ of **rs188213195**
- ‘T’ of **rs75800426**
- ‘C’ of **rs76730281**
- ‘C’ of **rs77197599**
- ‘C’ of **rs150794852**

Response To Melanotan II (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Melanotan II.*

Individual response to MT-II is influenced by genetic variation in melanocortin receptors, pigmentation genes, and hormone signaling pathways.

Skin, hair, and eye color are primarily dependent on the relative amounts of eumelanin and pheomelanin, two forms of the melanin pigment group. Eumelanin gives off a brown-black color, while pheomelanin gives off a red-yellow color. Typically, greater amounts of eumelanin result in darker skin, hair, and eyes [R].

Eumelanin is normally produced in response to activation of the melanocortin 1 receptor (MC1R), found on the surfaces of cells called melanocytes. Activation of this receptor is mediated by α-MSH. When MC1R is inactivated or blocked, melanocytes instead stimulate the development of pheomelanin. Blocking of MC1R is mediated by ASIP [R].

A variant of *MC1R* has been associated with hair and skin color. Carriers of the minor 'T' allele of **rs1805007** are more likely to have red or blond hair and pale skin, and get sunburns and melanoma. They may also show diminished tanning response to MT-II. Other variants of this gene associated with lower eumelanin production include [R, R]:
'T' of **rs1805008**
'T' of **rs1805009**

The *ASIP* gene codes for agouti-signaling protein (ASIP), which promotes pheomelanin production and the development of lighter skin by physically blocking MC1R [R, R].

The following *ASIP* variants have been associated with melanoma. These variants may alter the production of ASIP, leading to dysregulated melanin production and consequent disease progression. Carriers of these varaints may also show blunted effects of MT-II [R, R]:

BETTER RESPONSE

Predisposed to a better response to Melanotan II based on 13 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
POMC	rs6713532	TT
HERC2	rs4778138	AA
TYR	rs1393350	AG
TYR	rs1126809	AG
ASIP	rs910873	GG
ASIP	rs6059655	GG
ASIP	rs1885120	GG
MC1R	rs1805009	GG
MC1R	rs1805008	CC
MC1R	rs1805007	CC
MC4R	rs17782313	TT
MC4R	rs12970134	GG
POMC	rs934778	AA
POMC	rs1042571	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

- 'A' of **rs910873**
- 'C' of **rs1885120**
- 'A' of **rs6059655**

The *TYR* gene codes for tyrosinase, an enzyme that helps produce melanin and other pigments in melanocytes [R, R].

Two variants have been associated with an increased production of tyrosinase, potentially increasing melanin production and boosting the tanning effects of MT-II. However, please note that these variants have also been associated with an increased risk of melanoma progression and worse response to immunotherapy for this disease. These variants are [R, R, R, R]:

- 'A' of **rs1393350**
- 'A' of **rs1126809**

The *OCA2* gene codes for the P protein. This protein is thought to be involved in the formation of melanin pigment within melanosomes (structures found in melanocyte cells). The P protein may specifically function to transport melanin precursor molecules into and out of melanosomes, balance enzymes that aid in the production of melanin (tyrosinase), and maintain the acidity of melanosomes at a level required for melanin production [R, R].

The 'A' allele of **rs4778138** has been associated with fairer skin and a higher risk of melanoma. This variant may ultimately alter the production of P protein, leading to dysregulated melanin production and consequent disease progression [R, R, R].

The *MC4R* gene encodes the melanocortin 4 (MC4) receptor, which binds alpha-melanocyte-stimulating hormone or α-MSH. The primary location of this receptor is the brain, more precisely the hypothalamus, where it controls food intake, metabolism, reproductive behavior, and more [R].

The most studied SNP near the *MC4R* gene is **rs17782313**. The **"C" allele** is linked to increased body weight and appetite. Another important variant linked to obesity and food cravings is the 'A' allele of **rs12970134**. These variants likely **reduce gene expression or receptor activity**, thus increasing food intake and hindering glucose and fat metabolism. Because MT-II is a synthetic analogue of α-MSH, carriers of these variants may experience diminished appetite suppression from MT-II [R, R, R, R, R].

Proopiomelanocortin, or *POMC*, is a protein that is cut into multiple other, smaller proteins with specialized functions. More specifically, POMC is divided into adrenocorticotropic hormone (ACTH) and three melanocyte-stimulating hormones (α-, β-, and γ-MSH). POMC helps maintain homeostasis by, among other things, suppressing appetite when we have eaten enough food [R].

Lower POMC is generally associated with higher appetite and potential for weight gain. Three SNPs so far have been associated with higher weight and body fat to varying degrees [R, R, R, R]:

- 'T' of **rs6713532**
- 'A' of **rs1042571**
- 'G' of **rs934778**

Due to the lower α-MSH production associated with these variants, they may worsen response to MT-II.

∞ Anti-Aging & Longevity

The aging process is deeply connected to how well your cells repair damage, maintain mitochondrial health, and regulate inflammation over time. This section highlights peptides that support cellular renewal, DNA integrity, and longevity — including Epitalon, GHK-Cu, FOXO4-DRI, and Humanin, among others. These compounds can promote telomere maintenance, enhance mitochondrial resilience, and help clear senescent cells that contribute to aging. Understanding their effects offers valuable insights into supporting healthy aging, vitality, and long-term cellular function.

TYPICAL RESPONSE

Response to Epitalon (Hypothesis)

Predisposed to a typical response to Epitalon

TYPICAL RESPONSE

Response to Vilon (Hypothesis)

Predisposed to a typical response to Vilon

TYPICAL RESPONSE

Response to FOXO4-DRI (Hypothesis)

Predisposed to a typical response to FOXO4-DRI

TYPICAL RESPONSE

Response to Humanin (Hypothesis)

Predisposed to a typical response to Humanin

TYPICAL RESPONSE

Response to Small Humanin-Like Peptides (Hypothesis)

Predisposed to a typical response to SHLPs

BETTER RESPONSE

Response to GHK-Cu (Hypothesis)

Predisposed to a better response to GHK-Cu

Response To Epitalon (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Epitalon.*

Epitalon's effects involve pathways related to telomere maintenance, circadian rhythm regulation, and cellular stress response. Genetic variation in key genes involved in these pathways may influence individual responsiveness.

The *TERT* gene codes for hTERT, a part of the telomerase enzyme. Telomerase can protect DNA essential to the production of proteins (genes), by adding non-essential DNA (telomeres) to the ends of condensed DNA structures (chromosomes). This ultimately prevents genes from becoming damaged [R].

The *TERT* gene has a lot of known variations. Most people will probably have a mix of variations that increase and decrease relative telomere length, so it's important to look at as many SNPs as possible to see which way you lean. The following variants have been associated with an increased telomere length, potentially enhancing responsiveness to Epitalon [R, R, R, R, R, R]:

- 'T' at **rs2736108**
- 'A' at **rs7705526**
- 'C' at **rs2736100**
- 'A' at **rs7705526**
- 'T' at **rs4449583**
- 'G' at **rs33961405**
- 'C' at **rs10069690**
- 'A' at **rs13167280**
- 'A' at **rs2075786**
- 'G' at **rs2242652**
- 'A' at **rs2735940**
- 'C' at **rs2736098**
- 'A' at **rs2853669**
- 'A' at **rs2853672**
- 'C' at **rs2853676**
- 'A' at **rs2853677**

TYPICAL RESPONSE

Predisposed to a typical response to Epitalon based on 44 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
SLC6A3	rs2736108	CC
TERT	rs4449583	CC
TERT	rs2075786	GG
SIRT1	rs7895833	AA
SIRT3	rs11555236	CC
SIRT3	rs4980329	CC
FOXO3	rs12212067	TT
FOXO3	rs12202234	CC
FOXO3	rs17069665	AA
FOXO3	rs9398171	TT
FOXO3	rs3800230	TT
FOXO3	rs9400239	CC
FOXO3	rs479744	GG
FOXO3	rs4946936	CC
MTNR1A	rs13140012	AA
TERT	rs7705526	CA
TERT	rs2736100	AC
TERT	rs10069690	CT
TERT	rs13167280	AG
TERT	rs2242652	GA
SLC6A3	rs2735940	GA
TERT	rs2853672	AC
TERT	rs2853676	CT
TERT	rs2853677	AG
SIRT1	rs12778366	TC
SIRT1	rs3758391	CT
SIRT1	rs7896005	GA
SIRT3	rs3782118	CT
MTNR1A	rs6553010	GA
ARMC2	rs768023	GG

- ‘C’ at **rs4975605**

The *SIRT1* and *SIRT3* genes encode sirtuin proteins. 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].

A handful of studies have shown that people with certain *SIRT1* and *SIRT3* variants linked to increased activity of these genes are more likely to live long lives. Carriers may also respond better to Epitalon. Some of these variants include:

- ‘C’ of **rs12778366** [R, R]
- ‘G’ of **rs7895833** [R, R]
- ‘T’ of **rs3758391** [R, R, R]
- ‘A’ of **rs7896005** [R, R, R]
- ‘C’ of **rs2273773** [R, R, R]
- ‘A’ of **rs11555236** [R, R, R]
- ‘T’ of **rs4980329** [R, R, R]
- ‘C’ of **rs11246020** [R, R, R, R]
- ‘G’ of **rs28365927** [R, R]
- ‘C’ of **rs185277566** [R, R]
- ‘T’ of **rs3782118** [R]

The *FOXO3* gene encodes a transcription factor involved in tumor suppression, immune function, DNA repair, and resistance to oxidative stress, which may explain its link with increased longevity [R, R, R].

A large body of research has linked certain variants in the FOXO3 gene with longer lifespan, and potentially better response to Epitalon. They include [R, R, R, R, R, R, R]:

- ‘G’ at **rs12212067**
- ‘G’ at **rs768023**
- ‘C’ at **rs2253310**
- ‘A’ at **rs2802288**
- ‘G’ at **rs12202234**
- ‘G’ at **rs17069665**
- ‘C’ at **rs9398171**
- ‘G’ at **rs3800230**
- ‘C’ at **rs1935952**
- ‘T’ at **rs9400239**
- ‘T’ at **rs479744**
- ‘G’ at **rs2802292**
- ‘T’ at **rs4946936**
- ‘A’ at **rs6911407**
- ‘C’ at **rs2764264**

GENE	SNP	GENOTYPE
MTNR1A	rs7665392	TT
ARMC2	rs6911407	AA
TERT	rs4975605	CC
TERT	rs33961405	GG
CLPTM1L	rs2853669	AA
IFITM2	rs28365927	GG
FOXO3	rs2802292	GG
FOXO3	rs2802288	AA
TERT	rs2736098	CC
FOXO3	rs2253310	CC
MTNR1A	rs2119882	TT
FOXO3	rs1935952	CC
RIC8A	rs185277566	CC
MTNR1A	rs12506228	CC
SIRT3	rs11246020	CC

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

The *MTNR1A* gene codes for melatonin receptor 1A (MT1). This receptor is activated by the hormone melatonin, best known to induce sleep [R, R].

Certain variants are associated with a reduced number of MT1 receptors in the brain, potentially worsening the response to Epitalon. Some of them include:

- 'C' of **rs2119882** [R]
- 'A' of **rs12506228** [R]
- 'A' of **rs13140012** [R]
- 'G' of **rs6553010** [R]
- 'G' of **rs7665392** [R]

Response To Vilon (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Vilon.*

Genetic variants in immune regulation, oxidative stress, inflammation, and tissue regeneration pathways may influence individual response to Vilon. They may affect healing, immune balance, and aging outcomes.

Interleukin-6 (IL-6) is a cytokine encoded by the *IL6* gene that has either proinflammatory or anti-inflammatory properties, depending on the circumstances and the tissue that secretes it. It has complicated mechanisms of effect and interacts with many other cytokines and inflammatory signals [R, R].

By far, the best-researched *IL6* polymorphism is **rs1800795** (also known as the "-174G>C" polymorphism). The major 'G' allele of this SNP has been linked with higher levels of IL-6, while the 'C' allele has been associated with lower IL-6 levels. The major 'G' allele may lead to a more pronounced inflammatory response, potentially boosting the effects of Vilon [R].

Other variants of this gene linked to greater inflammation include:

- 'C' of **rs1524107** [R]
- 'A' of **rs75045751** [R]

The *IL10* gene codes for interleukin-10 (also abbreviated as IL-10), a cytokine with a complex relationship with inflammation. Most of the time, IL-10 is **anti-inflammatory** and suppresses the activity of Th1 cells, Th2 cells, neutrophils, macrophages, and natural killer cells [R, R, R].

Variants of this gene linked to a more anti-inflammatory profile may lower the immunomodulating effects of Vilon. These include [R, R, R, R, R, R, R]:

- 'C' of **rs1800896**
- 'G' of **rs1800871**
- 'T' of **rs1800890**

TYPICAL RESPONSE

Predisposed to a typical response to Vilon based on 9 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
TLR4	rs4986791	CC
IL10	rs1800871	GG
IL19	rs1800872	GG
TNF	rs1800629	GG
TNF	rs1799964	TT
HLA-DRB5	rs3763313	AA
HLA-DQA1	rs2187668	CC
SOD2	rs4880	GG
GPX1	rs1050450	GG
NFE2L2	rs6721961	GG
AGPS	rs2364723	GG
AGPS	rs10497511	AA
HNRNPA3	rs13001694	GG
AGPS	rs1962142	GG
NR3C1	rs2918419	TT
NR3C1	rs6196	AA
STEAP1B	rs75045751	GG
IL19	rs1800896	CT
IL19	rs1800890	TA
IL19	rs3024505	AG
TNF	rs1799724	TC
HLA-DQA2	rs2395185	TG
/	rs9271366	AG
HLA-DRB1	rs2516049	CT
CAT	rs1001179	TC
CAT	rs7943316	TA
NR3C1	rs852977	GA
NR3C1	rs1866388	GA
NR3C1	rs6188	AC
IL6	rs1524107	CT

- ‘G’ of **rs3024505**
- ‘G’ of **rs1800872**

The *TNF* gene encodes a protein called tumor necrosis factor-alpha (TNF-alpha or cachexin). TNF-alpha plays a central role in the immune response and inflammation, and it has been implicated in a wide variety of inflammatory disorders [R].

The **rs1800629** polymorphism (also known as *TNF* -308) is one of the most researched SNPs in the *TNF* gene. **The ‘A’ allele is associated with 6-7 times higher levels of TNF-alpha.** This may result in better restorative effect from Vilon [R].

Other variants that may increase TNF levels include:

- ‘C’ of **rs1799964** [R]
- ‘C’ of **rs1799724** [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 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].

The *SOD2* gene has many described polymorphisms. Among them, **rs4880** has received the spotlight in SOD2 research. Its minor allele ‘G’ is associated with decreased activity and worse protection against oxidative stress. Carriers may respond better to Vilon’s antioxidant and cytoprotective actions [R].

The *CAT* gene encodes subunits of catalase, a key antioxidant enzyme in the body’s defense against oxidative stress. Four identical subunits, each attached to an iron-containing molecule called a heme group, form the functional catalase enzyme [R].

The best-characterized *CAT* polymorphism is **rs1001179**, commonly referred to as -262G>A. Its minor ‘T’ allele may increase *CAT* expression but decrease it in people with conditions such as type 2 diabetes or chronic hepatitis C. Other well-known CAT variants with increased activity include ‘T’ of **rs769217** and ‘A’ of **rs7943316.** They may decrease the antioxidant effects of Vilon [R, R, R, R, R].

The *GPX1* gene helps make glutathione peroxidase (GPx), one of the body’s key antioxidant enzymes. This enzyme converts

GENE	SNP	GENOTYPE
CAT	rs769217	CC
NFE2L2	rs6726395	AA
NR3C1	rs41423247	GG
NFE2L2	rs35652124	TT
NFE2L2	rs1806649	CC
IL6	rs1800795	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

hydrogen peroxide and glutathione into glutathione disulfide and water. By doing so, GPx helps reduce oxidative stress [R].

One study found a direct link between a common *GPX1* variant and human longevity. The heterozygous **genotype 'AG' at rs1050450 was significantly more common in the very elderly** than in the general population. Other studies have strongly suggested that the **'G' allele at rs1050450 confers higher GPx activity**. Carriers may benefit less from Vilon [R, R, R].

The *TLR4* gene codes for toll-like receptor 4. This protein jump-starts the innate immune response, which is a mechanism our bodies have for fighting pathogens we have never encountered before. TLR4 recognizes foreign compounds on the surface of invading pathogens (usually bacteria) and activates the immune response. As part of this process, it increases inflammation through NF-κB and cytokines like IL-1 and IL-12 [R, R].

The best-researched TLR4 variant is **rs4986791**. Its minor 'T' allele is believed to increase TLR4 activation based on its pro-inflammatory effects, possibly increasing the effectiveness of Vilon against infections and inflammation [R].

The *HLA-DRB1* gene encodes the beta chain of the *human leukocyte antigen (HLA)* system, which merges with the alpha chain to form the HLA-DR receptor. Hundreds of DRB1 variants can change the structure and activity of this receptor and thus impact the immune response. Some of them have been associated with autoimmune conditions [R, R, R, R].

Variants linked to a higher expression of the *HLA-DRB1* and *DQA1* genes may enhance the immune-modulating effects of Vilon. These include [R, R]:

- 'G' of **rs2395185**
- 'G' of **rs9271366**
- 'C' of **rs3763313**
- 'T' of **rs2187668**
- 'C' of **rs2516049**

The *NR3C1* gene codes for the glucocorticoid receptor. Upon activation by glucocorticoids (such as the primary stress hormone cortisol), the glucocorticoid receptor is able to regulate the production of stress-related, inflammatory proteins [R].

Variants that lower the sensitivity or activity of the glucocorticoid receptor, thus resulting in dysregulated HPA axis activity, excess

cortisol release, and inflammation, have been associated with chronic fatigue syndrome and may worsen response to Vilon. They include:

- 'A' at **rs852977** [R, R, R]
- 'A' at **rs1866388** [R, R, R]
- 'C' at **rs6188** [R, R, R]
- 'T' at **rs2918419** [R, R, R]
- 'A' at **rs6196** [R, R, R]
- 'C' at **rs41423247** [R, R]

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].

Research has identified several variations in the NFE2L2 gene that can reduce the expression and activity of NRF2. Researchers have claimed that reduced NRF2 impairs the body's ability to detox and defend itself from oxidative stress. Some of these variants, which may be linked to enhanced benefits from Vilon, include [R, R]:

- 'T' of **rs35652124**
- 'T' of **rs6721961**
- 'A' of **rs6726395**
- 'C' of **rs2364723**
- 'G' of **rs10497511**
- 'A' of **rs13001694**
- 'C' of **rs1806649**
- 'A' of **rs1962142**

The *TERT* gene codes for hTERT, a part of the telomerase enzyme. Telomerase can protect DNA essential to the production of proteins (genes), by adding non-essential DNA (telomeres) to the ends of condensed DNA structures (chromosomes). This ultimately prevents genes from becoming damaged [R].

The *TERT* gene has a lot of known variations. Most people will probably have a mix of variations that increase and decrease relative telomere length, so it's important to look at as many SNPs as possible to see which way you lean. The following variants have been associated with an increased telomere length, potentially enhancing responsiveness to Vilon [R, R, R, R, R, R]:

- 'T' at **rs2736108**
- 'A' at **rs7705526**

- ‘C’ at **rs2736100**
- ‘A’ at **rs7705526**
- ‘T’ at **rs4449583**
- ‘G’ at **rs33961405**
- ‘C’ at **rs10069690**
- ‘A’ at **rs13167280**
- ‘A’ at **rs2075786**
- ‘G’ at **rs2242652**
- ‘A’ at **rs2735940**
- ‘C’ at **rs2736098**
- ‘A’ at **rs2853669**
- ‘A’ at **rs2853672**
- ‘C’ at **rs2853676**
- ‘A’ at **rs2853677**
- ‘C’ at **rs4975605**

The *FOXO3* gene encodes a transcription factor involved in tumor suppression, immune function, DNA repair, and resistance to oxidative stress, which may explain its link with increased longevity [R, R, R].

A large body of research has linked certain variants in the *FOXO3* gene with longer lifespan, and potentially better response to Vilon. They include [R, R, R, R, R, R, R]:

- ‘G’ at **rs12212067**
- ‘G’ at **rs768023**
- ‘C’ at **rs2253310**
- ‘A’ at **rs2802288**
- ‘G’ at **rs12202234**
- ‘G’ at **rs17069665**
- ‘C’ at **rs9398171**
- ‘G’ at **rs3800230**
- ‘C’ at **rs1935952**
- ‘T’ at **rs9400239**
- ‘T’ at **rs479744**
- ‘G’ at **rs2802292**
- ‘T’ at **rs4946936**
- ‘A’ at **rs6911407**

Response To FOXO4-DRI (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of FOXO4-DRI.*

Genetic variation in mitochondrial function, oxidative stress, and apoptosis pathways may influence individual response to FOXO4-DRI, affecting energy metabolism, aging, and cell protection.

The *TP53* gene encodes the '*tumor protein 53*', often referred to as p53. p53 functions as a tumor suppressor protein that regulates the expression of genes involved in the 'cell cycle', DNA damage repair, and cell death [R, R].

The best-researched *TP53* variant is **rs1042522**, also called the 'Arg72Pro' polymorphism. Its minor 'G' variant, carried by almost 50% of the European population, encodes a protein with decreased activity and has been associated with an increased overall cancer risk. Because this variant favors cell cycle arrest over cell death, carriers may benefit more from FOXO4-DRI's apoptosis-restoring effect [R, R].

Other TP53 variants associated with lower activity and, presumably, better response to FOXO4-DRI include:
'C' of **rs12951053** [R, R, R, R]
'T' of **rs1625895** [R, R, R, R, R, R]

The *FOXO3* gene encodes a transcription factor involved in tumor suppression, immune function, DNA repair, and resistance to oxidative stress, which may explain its link with increased longevity [R, R, R].

A large body of research has linked certain variants in the *FOXO3* gene with longer lifespan, and, potentially, complementation of FOXO4-DRI's anti-aging actions. They include [R, R, R, R, R, R, R]:

- 'G' at **rs12212067**
- 'G' at **rs768023**
- 'C' at **rs2253310**

TYPICAL RESPONSE

Predisposed to a typical response to FOXO4-DRI based on 9 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
EFNB3	rs12951053	AA
TP53	rs1625895	CC
BID	rs183393610	GG
NFE2L2	rs6721961	GG
AGPS	rs2364723	GG
AGPS	rs10497511	AA
HNRNPA3	rs13001694	GG
AGPS	rs1962142	GG
FOXO3	rs12212067	TT
FOXO3	rs12202234	CC
FOXO3	rs17069665	AA
FOXO3	rs9398171	TT
FOXO3	rs3800230	TT
FOXO3	rs9400239	CC
FOXO3	rs479744	GG
FOXO3	rs4946936	CC
SLC6A3	rs2736108	CC
TERT	rs4449583	CC
TERT	rs2075786	GG
TERT	rs7705526	CA
TERT	rs2736100	AC
TERT	rs10069690	CT
TERT	rs13167280	AG
TERT	rs2242652	GA
SLC6A3	rs2735940	GA
TERT	rs2853672	AC
TERT	rs2853676	CT
TERT	rs2853677	AG
TP53	rs1042522	CC
ARMC2	rs768023	GG

- ‘A’ at **rs2802288**
- ‘G’ at **rs12202234**
- ‘G’ at **rs17069665**
- ‘C’ at **rs9398171**
- ‘G’ at **rs3800230**
- ‘C’ at **rs1935952**
- ‘T’ at **rs9400239**
- ‘T’ at **rs479744**
- ‘G’ at **rs2802292**
- ‘T’ at **rs4946936**
- ‘A’ at **rs6911407**

The *BID* gene encodes a protein called ‘BH3-interacting domain death agonist’ that regulates cell death by forming a complex with either its agonist BAX or its antagonist BCL2 [R].

The following *BID* variants have been associated with lower BID levels, which may favor the effects of FOXO4-DRI:

- ‘A’ of **rs183393610** [R]
- ‘A’ of **rs151268390** [R]

The *BCL2* gene encodes a protein that blocks the death of certain cells, such as lymphocytes [R].

The ‘C’ allele of **rs4987856** has been associated with higher BCL2 protein levels. Because this protein blocks cell death, carriers of this variant may respond better to FOXO4-DRI [R].

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].

Research has identified several variations in the NFE2L2 gene that can reduce the expression and activity of NRF2. Researchers have claimed that reduced NRF2 impairs the body’s ability to detox and defend itself from oxidative stress. Some of these variants, which may be linked to enhanced benefits from FOXO4-DRI, include [R, R]:

- ‘T’ of **rs35652124**
- ‘T’ of **rs6721961**
- ‘A’ of **rs6726395**
- ‘C’ of **rs2364723**
- ‘G’ of **rs10497511**
- ‘A’ of **rs13001694**
- ‘C’ of **rs1806649**
- ‘A’ of **rs1962142**

GENE	SNP	GENOTYPE
ARMC2	rs6911407	AA
NFE2L2	rs6726395	AA
BCL2	rs4987856	CC
TERT	rs4975605	CC
NFE2L2	rs35652124	TT
TERT	rs33961405	GG
CLPTM1L	rs2853669	AA
FOXO3	rs2802292	GG
FOXO3	rs2802288	AA
TERT	rs2736098	CC
FOXO3	rs2253310	CC
FOXO3	rs1935952	CC
NFE2L2	rs1806649	CC
BID	rs151268390	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Finally, the *TERT* gene codes for hTERT, a part of the telomerase enzyme. Telomerase can protect DNA essential to the production of proteins (genes), by adding non-essential DNA (telomeres) to the ends of condensed DNA structures (chromosomes). This ultimately prevents genes from becoming damaged [R].

The *TERT* gene has a lot of known variations. Most people will probably have a mix of variations that increase and decrease relative telomere length, so it's important to look at as many SNPs as possible to see which way you lean. The following variants have been associated with an increased telomere length, potentially enhancing responsiveness to FOXO4-DRI [R, R, R, R, R, R]:

- 'T' at **rs2736108**
- 'A' at **rs7705526**
- 'C' at **rs2736100**
- 'A' at **rs7705526**
- 'T' at **rs4449583**
- 'G' at **rs33961405**
- 'C' at **rs10069690**
- 'A' at **rs13167280**
- 'A' at **rs2075786**
- 'G' at **rs2242652**
- 'A' at **rs2735940**
- 'C' at **rs2736098**
- 'A' at **rs2853669**
- 'A' at **rs2853672**
- 'C' at **rs2853676**
- 'A' at **rs2853677**
- 'C' at **rs4975605**

Response To Humanin (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Humanin.*

Response to Humanin may be influenced by variants in genes that affect apoptosis, mitochondrial function, and insulin signaling. Genetic differences may modulate the peptide's protective and metabolic effects.

The *IGFBP3* gene encodes insulin-like growth factor binding protein 3, a protein that forms a complex with insulin-like growth factor acid-labile subunit (IGFALS) and either insulin-like growth factor 1 or 2. In this form, it circulates in the plasma, prolonging the half-life of IGFs and altering their interaction with cell surface receptors [R].

Variants associated with higher IGFBP3 levels or activity may enhance the beneficial effects of Humanin. Some of these variants include:

- 'G' of **rs2854746** [R, R]
- 'G' of **rs2854744** [R]
- 'G' of **rs145188037** [R]
- 'G' of **rs9282734** [R]
- 'C' of **rs199644320** [R]
- 'T' of **rs2949833** [R]
- 'T' of **rs788709** [R]
- 'A' of **rs11977526** [R]
- 'A' of **rs117561950** [R]
- 'A' of **rs117774089** [R]
- 'T' of **rs145646599** [R]
- 'T' of **rs1722116** [R]

The *BID* gene encodes a protein called 'BH3-interacting domain death agonist' that regulates cell death by forming a complex with either its agonist BAX or its antagonist BCL2 [R].

The following *BID* variants have been associated with lower BID levels, which may favor the effects of Humanin:

- 'A' of **rs183393610** [R]

TYPICAL RESPONSE

Predisposed to a typical response to Humanin based on 23 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
IGFBP3	rs9282734	TT
IGFBP3	rs199644320	AA
IGFBP3	rs788709	CC
IGFBP3	rs117561950	GG
IGFBP1	rs117774089	GG
IGFBP3	rs145646599	CC
BID	rs183393610	GG
IGFBP3	rs2854746	CG
IGFBP3	rs2854744	TG
IGFBP3	rs2949833	TC
IGFBP3	rs11977526	AG
IGFBP3	rs1722116	TA
PPARGC1A	rs8192678	TC
INSR	rs7252150	AG
IRS1	rs2943641	TC
IRS1	rs7578326	GA
INSR	rs75253922	TT
INSR	rs66496113	CC
IRS1	rs1801278	CC
INSR	rs1799815	GG
INSR	rs17175860	AA
BID	rs151268390	AA
IGFBP3	rs145188037	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

- ‘A’ of **rs151268390** [R]

The *BCL2* gene encodes a protein that blocks the death of certain cells, such as lymphocytes [R].

The ‘C’ allele of **rs4987856** has been associated with higher BCL2 protein levels. Because this protein blocks cell death, carriers of this variant may respond better to Humanin [R].

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].

The best-researched *PPARGC1A* polymorphism is **rs8192678**. Its minor ‘T’ allele decreases *PPARGC1A* expression and PGC-1α levels in the muscles, potentially worsening response to Humanin [R, R, R].

The *INSR* gene encodes a protein called an insulin receptor, which plays many roles in the body. It regulates blood sugar levels by controlling how much sugar is passed from the bloodstream into cells to be used as energy [R].

The following variants have been associated with higher insulin receptor levels and decreased blood sugar, potentially favoring the effects of Humanin:

- ‘C’ of **rs66496113** [R]
- ‘G’ of **rs1799815** [R]
- ‘A’ of **rs17175860** [R]
- ‘T’ of **rs75253922** [R]
- ‘G’ of **rs7252150** [R]

Insulin receptor substrate 1 (IRS1) is a key player in how our bodies process and use energy. It’s part of the system that helps cells respond to insulin, a hormone that regulates blood sugar levels.

Variants linked to better metabolic outcomes may boost IRS1 activity, improving insulin receptor activation. This stimulates the downstream signaling pathways essential for glucose uptake and lipid metabolism, directly impacting blood sugar and fat storage. Some examples of these variants include [R]:

- ‘T’ of **rs2943641** [R]
- ‘G’ of **rs7578326** [R, R]

- ‘C’ of **rs1801278** [R, R, R]

Response To Small Humanin-Like Peptides (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of Small Humanin-Like Peptides.*

Genetic variation in mitochondrial function, oxidative stress, and apoptosis pathways may influence individual response to SHLPs, affecting energy metabolism, aging, and cell protection.

The *BID* gene encodes a protein called 'BH3-interacting domain death agonist' that regulates cell death by forming a complex with either its agonist BAX or its antagonist BCL2 [R].

The following *BID* variants have been associated with lower BID levels, which may favor the effects of SHLPs:

- 'A' of **rs183393610** [R]
- 'A' of **rs151268390** [R]

The *BCL2* gene encodes a protein that blocks the death of certain cells, such as lymphocytes [R].

The 'C' allele of **rs4987856** has been associated with higher BCL2 protein levels. Because this protein blocks cell death, carriers of this variant may respond better to SHLPs [R].

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].

The best-researched *PPARGC1A* polymorphism is **rs8192678**. Its minor 'T' allele decreases *PPARGC1A* expression and PGC-1α levels in the muscles, potentially worsening response to SHLPs [R, R, 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 superoxide produced by the

TYPICAL RESPONSE

Predisposed to a typical response to SHLPs based on 9 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
BID	rs183393610	GG
NFE2L2	rs6721961	GG
AGPS	rs2364723	GG
AGPS	rs10497511	AA
HNRNPA3	rs13001694	GG
AGPS	rs1962142	GG
STEAP1B	rs75045751	GG
FOXO3	rs12212067	TT
FOXO3	rs12202234	CC
FOXO3	rs17069665	AA
FOXO3	rs9398171	TT
FOXO3	rs3800230	TT
FOXO3	rs9400239	CC
FOXO3	rs479744	GG
FOXO3	rs4946936	CC
PPARGC1A	rs8192678	TC
CAT	rs1001179	TC
CAT	rs7943316	TA
IL6	rs1524107	CT
TNF	rs1799724	TC
CAT	rs769217	CC
ARMC2	rs768023	GG
ARMC2	rs6911407	AA
NFE2L2	rs6726395	AA
BCL2	rs4987856	CC
SOD2	rs4880	GG
NFE2L2	rs35652124	TT
FOXO3	rs2802292	GG
FOXO3	rs2802288	AA
FOXO3	rs2253310	CC

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].

The *SOD2* gene has many described polymorphisms. Among them, **rs4880** has received the spotlight in SOD2 research. Its minor allele 'G' is associated with decreased activity and worse protection against oxidative stress. Carriers may respond better to the antioxidant and cytoprotective actions of SHLPs [R].

The *CAT* gene encodes subunits of catalase, a key antioxidant enzyme in the body's defense against oxidative stress. Four identical subunits, each attached to an iron-containing molecule called a heme group, form the functional catalase enzyme [R].

The best-characterized *CAT* polymorphism is **rs1001179**, commonly referred to as -262G>A. Its minor 'T' allele may increase *CAT* expression but decrease it in people with conditions such as type 2 diabetes or chronic hepatitis C. Other well-known CAT variants with increased activity include 'T' of **rs769217** and 'A' of **rs7943316.** They may decrease the antioxidant effects of SHLPs [R, R, R, R, R].

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].

One study found a direct link between a common *GPX1* variant and human longevity. The heterozygous **genotype 'AG' at rs1050450 was significantly more common in the very elderly** than in the general population. Other studies have strongly suggested that the **'G' allele at rs1050450 confers higher GPx activity**. Carriers may benefit less from SHLPs [R, R, R].

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].

Research has identified several variations in the NFE2L2 gene that can reduce the expression and activity of NRF2. Researchers have claimed that reduced NRF2 impairs the body's ability to detox and defend itself from oxidative stress.

GENE	SNP	GENOTYPE
FOXO3	rs1935952	CC
NFE2L2	rs1806649	CC
IL6	rs1800795	GG
TNF	rs1800629	GG
TNF	rs1799964	TT
BID	rs151268390	AA
GPX1	rs1050450	GG

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

Some of these variants, which may be linked to enhanced benefits from SHLPs, include [R, R]:

- 'T' of **rs35652124**
- 'T' of **rs6721961**
- 'A' of **rs6726395**
- 'C' of **rs2364723**
- 'G' of **rs10497511**
- 'A' of **rs13001694**
- 'C' of **rs1806649**
- 'A' of **rs1962142**

Interleukin-6 (IL-6) is a cytokine encoded by the *IL6* gene that has either proinflammatory or anti-inflammatory properties, depending on the circumstances and the tissue that secretes it. It has complicated mechanisms of effect and interacts with many other cytokines and inflammatory signals [R, R].

By far, the best-researched *IL6* polymorphism is **rs1800795** (also known as the "-174G>C" polymorphism). The major 'G' allele of this SNP has been linked with higher levels of IL-6, while the 'C' allele has been associated with lower IL-6 levels. The major 'G' allele may lead to a more pronounced inflammatory response. Carriers may benefit more from SHLPs [R].

Other variants of this gene linked to greater inflammation include:

- 'C' of **rs1524107** [R]
- 'A' of **rs75045751** [R]

The *TNF* gene encodes a protein called tumor necrosis factor-alpha (TNF-alpha or cachexin). TNF-alpha plays a central role in the immune response and inflammation, and it has been implicated in a wide variety of inflammatory disorders [R].

The **rs1800629** polymorphism (also known as *TNF* -308) is one of the most researched SNPs in the *TNF* gene. **The 'A' allele is associated with 6-7 times higher levels of TNF-alpha.** This may result in better anti-inflammatory effects from SHLPs [R].

Other variants that may increase TNF levels include:

- 'C' of **rs1799964** [R]
- 'C' of **rs1799724** [R]

Finally, the *FOXO3* gene encodes a transcription factor involved in tumor suppression, immune function, DNA repair, and

resistance to oxidative stress, which may explain its link with increased longevity [R, R, R].

A large body of research has linked certain variants in the FOXO3 gene with longer lifespan, and potentially better response to SHLPs. They include [R, R, R, R, R, R, R]:

- 'G' at **rs12212067**
- 'G' at **rs768023**
- 'C' at **rs2253310**
- 'A' at **rs2802288**
- 'G' at **rs12202234**
- 'G' at **rs17069665**
- 'C' at **rs9398171**
- 'G' at **rs3800230**
- 'C' at **rs1935952**
- 'T' at **rs9400239**
- 'T' at **rs479744**
- 'G' at **rs2802292**
- 'T' at **rs4946936**
- 'A' at **rs6911407**

Response To GHK-Cu (Hypothesis)

Please note: *The claims in this section haven't been verified by scientific research. They are a hypothesis based on the mechanism of this peptide. No genetic variants have been found to modify the effectiveness of GHK-Cu.*

The biological effects of GHK-Cu may be influenced by genetic variants that affect tissue repair, extracellular matrix production, copper metabolism, and inflammatory signaling.

The *MMP1* gene codes for matrix metalloproteinase 1, also known as interstitial collagenase. This enzyme plays a significant role in collagen breakdown; it degrades type I and III collagen, the primary types found in skin. While MMP1 activity is essential for normal skin remodeling and repair, excessive activity can accelerate collagen degradation, leading to premature aging [R].

Variants with decreased MMP1 activity may enhance how well the GHK-Cu peptide can accelerate healing. These include:

- 'T' of **rs1799750** [R]
- 'A' of **rs470747** [R]
- 'G' of **rs139018071** [R]
- 'T' of **rs470558** [R]
- 'C' of **rs17879749** [R]

The *MMP9* gene codes for matrix metalloproteinase 9, also known as 92 kDa type IV collagenase, 92 kDa gelatinase, or gelatinase B. This enzyme plays a significant role in collagen breakdown; it degrades type IV and V collagens. While MMP9 activity is essential for embryonic development, reproduction, and tissue remodeling, excessive activity can accelerate collagen degradation, leading to arthritis and metastasis [R].

The following *MMP9* variants may lead to better tissue repair outcomes when using GHK-Cu:

- 'A' of **rs17576** [R]
- 'T' of **rs3918249** [R]
- 'A' of **rs6094237** [R]
- 'G' of **rs2250889** [R]

BETTER RESPONSE

Predisposed to a better response to GHK-Cu based on 17 genetic variants we looked at

Your top variants that most likely impact your genetic predisposition:

GENE	SNP	GENOTYPE
MMP1	rs1799750	TT
MMP1	rs470747	AA
MMP1	rs17879749	CC
TNF	rs1800629	GG
TNF	rs1799964	TT
STEAP1B	rs75045751	GG
ATP7B	rs1801248	CC
NEK3	rs144942726	CC
MMP9	rs17576	AG
PLTP	rs3918249	TC
PLTP	rs6094237	AT
COL1A1	rs1800012	AC
COL5A1	rs12722	TC
TNF	rs1799724	TC
IL6	rs1524107	CT
MMP1	rs470558	CC
MMP9	rs2250889	CC
IL6	rs1800795	GG
MMP1	rs139018071	AA

The number of "risk" variants in this table doesn't necessarily reflect your overall result.

The *COL1A1* gene encodes part of a large molecule called type I collagen. Collagens are a family of proteins that strengthen and support many tissues in the body, including cartilage, bone, tendon, and skin. Type I collagen is the most abundant form of collagen in the human body. [R].

The best-researched *COL1A1* polymorphism is **rs1800012**. Its minor 'A' allele promotes the expression of an alternative version of the protein with increased pro-α1 chain levels, potentially increasing its mechanical strength and stability. Carriers may experience enhanced tissue regeneration from GHK-Cu [R].

The *COL5A1* gene encodes a component of type V collagen called the pro-α1(V) chain. Collagens are a family of proteins that strengthen and support many tissues in the body, including skin, ligaments, bones, tendons, and muscles [R].

The minor 'T' allele of **rs12722** has been associated with weaker ligaments, tendons, and muscles, as well as with an increased risk of soft tissue injuries. Carriers may respond worse to GHK-Cu due to their decreased production of collagen [R, R].

The *TNF* gene encodes a protein called tumor necrosis factor-alpha (TNF-alpha or cachexin). TNF-alpha plays a central role in the immune response and inflammation, and it has been implicated in a wide variety of inflammatory disorders [R].

The **rs1800629** polymorphism (also known as *TNF* -308) is one of the most researched SNPs in the *TNF* gene. **The 'A' allele is associated with 6-7 times higher levels of TNF-alpha.** This may require higher or prolonged exposure to GHK-Cu for optimal therapeutic effects [R].

Other variants that may increase TNF levels include:

- 'C' of **rs1799964** [R]
- 'C' of **rs1799724** [R]

Interleukin-6 (IL-6) is a cytokine encoded by the *IL6* gene that has either proinflammatory or anti-inflammatory properties, depending on the circumstances and the tissue that secretes it. It has complicated mechanisms of effect and interacts with many other cytokines and inflammatory signals [R, R].

By far, the best-researched *IL6* polymorphism is **rs1800795** (also known as the "-174G>C" polymorphism). The major 'G' allele of this SNP has been linked with higher levels of IL-6, while the 'C' allele has been associated with lower IL-6 levels. As was the

case with *TNF* variants resulting in higher production of the inflammatory cytokines, the ‘G’ allele may be associated with decreased response to GHK-Cu [R].

Other variants of this gene linked to an enhanced inflammatory response, and thus worse response to GHK-Cu, include:

- ‘C’ of **rs1524107** [R]
- ‘A’ of **rs75045751** [R]

Finally, the *ATP7B* gene encodes a protein called copper-transporting ATPase 2. It transports copper into and out of cells by using ATP. Mutations of this gene are associated with Wilson's disease, which occurs when excess copper is kept in the body and damages the organs [R].

Variants that reduce copper transport may limit the availability of active GHK-Cu, decreasing its regenerative and antioxidant effects. These include:

- ‘T’ of **rs1801248** [R, R]
- ‘T’ of **rs144942726** [R, R]