Our mission is to harness genetic data to craft personalized medical solutions, ensuring the most effective and safest treatments for patients

OZEMPIC AND OTHER GLP-1 AGONISTS

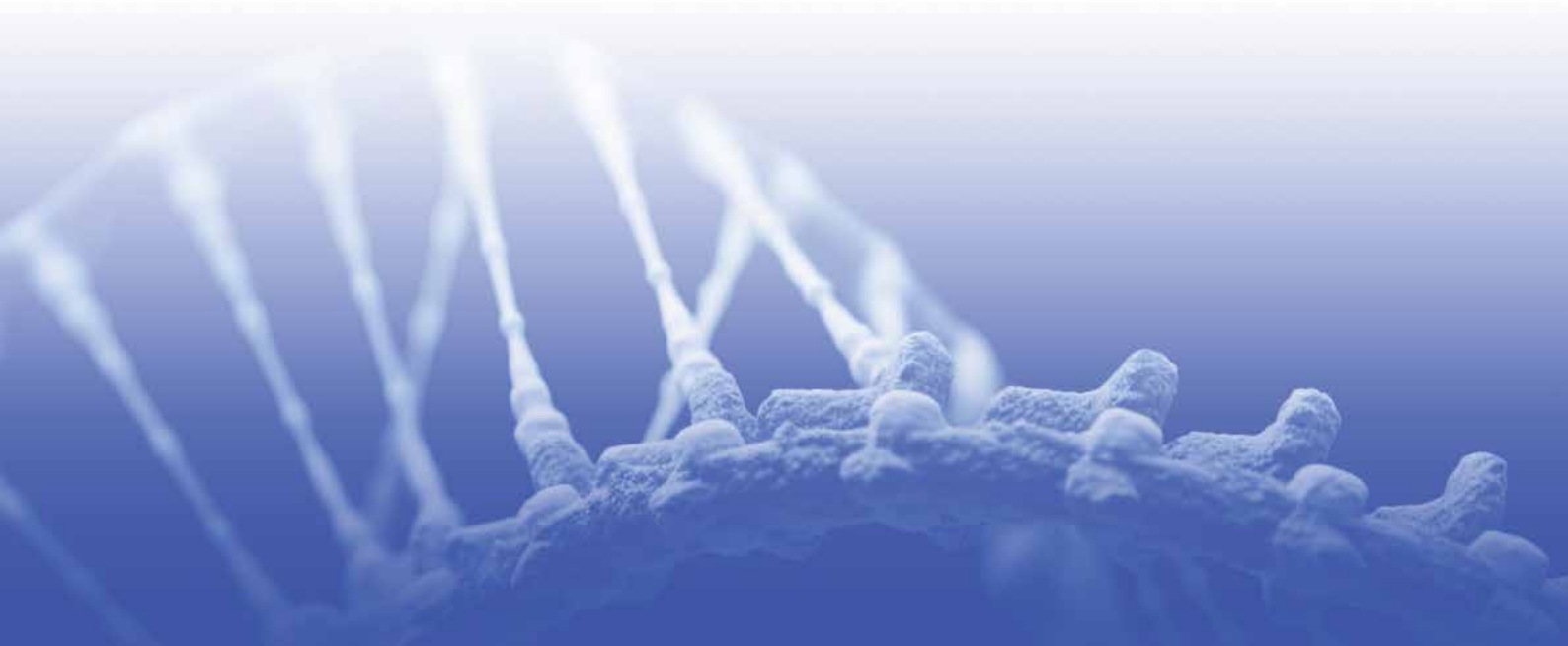


A company at the forefront of scientific research and innovation in the field of precision medicine

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SAMPLE REPORT



Our mission is to harness genetic data to craft personalized medical solutions, ensuring the most effective and safest treatments for patients

WHERE GENES MEET AI PRECISION

Pharmacogenetic analyses provide insights into specific DNA variations that can impact drug interactions by elucidating how particular DNA changes may affect enzyme efficiency in drug metabolism, while recognizing that other factors, such as age, body mass, liver and kidney health, concurrent medications, and lifestyle choices, also contribute.

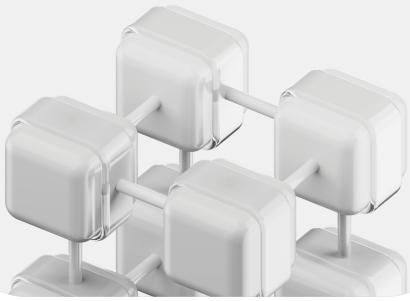
Our key advantages

SAMPLE REPORT

1 Links to the sources



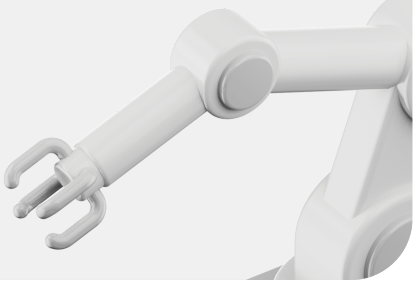
2 Impact of each individual gene



3 The results are presented in an easy-to-understand format

#Design

4 Detailed supplementary information about each substance



5 Comprehensive assessment of the influence of the entire genome on substances

6 Highly qualified team from leading universities

Stanford University

Berkeley UNIVERSITY OF CALIFORNIA

About this Report

This report compiles a detailed DNA analysis, highlighting key genetic variants that influence the efficacy, safety, metabolism, and receptor affinity of weight-loss medications. Leveraging PGxAI's expert interpretation, it delivers a comprehensive assessment of genetic responses for personalized weight-loss therapy. The insights provided, based on pharmacogenomic research and educational data for non-FDA-approved substances, aim to improve therapeutic outcomes by aligning weight-loss drugs use with the individual's genetic profile, ensuring safer and more effective experiences.

This report, generated by PGxAI analytical tool, is a cutting-edge resource providing comprehensive genetic insights to aid healthcare professionals in individualized treatment decisions. Despite the profound depth and breadth of genetic information it provides, this report has inherent limitations.

Limitations

The report does not encompass every possible genetic variant known to influence gene function and may not account for all individual biological variations.
Non-detection of a genetic variant does not negate the potential for altered phenotypes stemming from unidentified polymorphisms, drug interactions, co-existing medical conditions, and lifestyle factors.
Private genetic variants, pseudogenes, and other sequences homologous to the tested regions may impact the variant detection.

Methodology

The high-resolution array-based assays identify a wide array of common and rare alleles known to have clinical significance with an analytical sensitivity and specificity greater than 99%.

Disclaimer

The content of this report provides crucial genetic health information intended for use as an educational supplement, and should not supplant professional medical advice, diagnosis, or treatment. It is the purview of a qualified healthcare professional to guide patients regarding their medication usage. This report should be seen as one facet of a holistic patient assessment which should also include a thorough clinical history, evaluation of concurrent medical conditions and medications, family history, and other relevant factors. While the report can inform treatment options, the onus of determining the best course of patient treatment remains with the healthcare provider. Adherence to the report's recommendations does not guarantee a successful medical outcome.
The software involved in the generation of this report, including PGxAI, has not been reviewed by the U.S. Food and Drug Administration (FDA) or the European Medicines Agency (EMA). Its purpose is not to diagnose, treat, cure, or prevent any disease.

SAMPLE REPORT

Customer's Data

ID:

Processed:

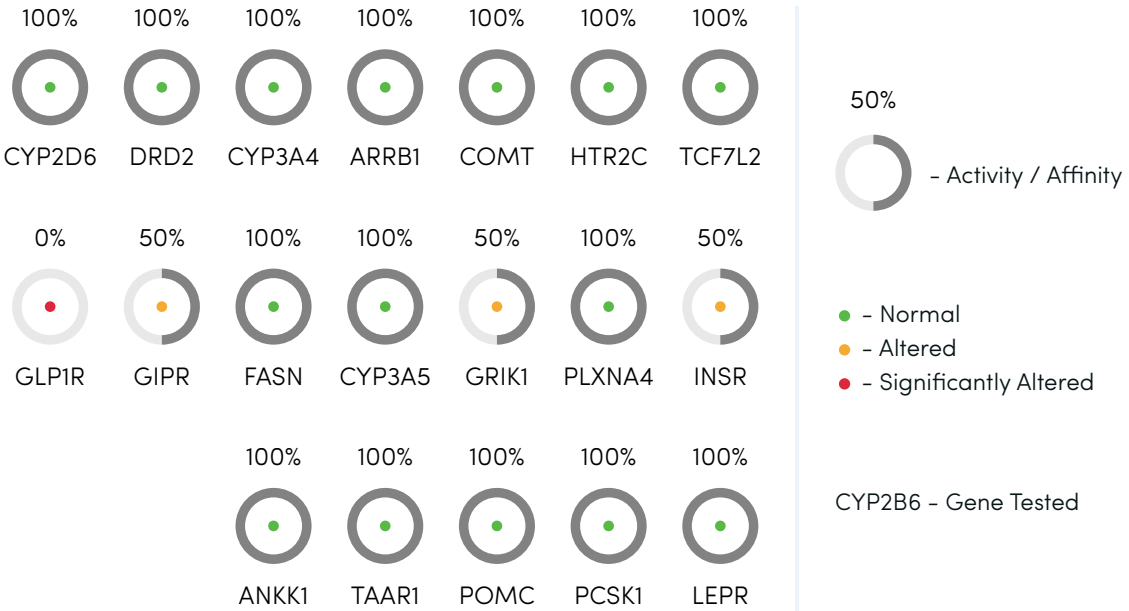
Individual Response

Anticipated response to weight-reduction substances based on this genetic analysis.



SAMPLE REPORT

Genetic Profile



Wegovy
Ozempic

Introduction

Semaglutide, marketed as Wegovy and Ozempic, is a GLP-1 receptor agonist that aids in weight loss and blood sugar control. It mimics the action of GLP-1, a hormone naturally produced in the gut following meals. By engaging with the appetite center in the brain, semaglutide decreases hunger and increases feelings of fullness, leading to reduced food intake and prolonged satiety. Additionally, it slows gastric emptying, further contributing to weight loss. Semaglutide also promotes insulin secretion and inhibits glucagon release, helping to manage blood sugar levels. Administered as a weekly subcutaneous injection, semaglutide, when combined with lifestyle modifications such as diet and exercise, can significantly enhance weight reduction and glycemic control. Its effectiveness may vary among individuals due to genetic factors. Variations in genes like GLP1R, ARRB1, and TCF7L2 can influence how well a person responds to the drug. This explains why some individuals might experience better weight loss outcomes with semaglutide than others.

Genetic Profile & Pharmacogenetic Implication [1, 2]

Overall

Lower Sensitivity

The genetic profile, including decreased affinity of GLP1R, normal activity of ARRB1, and normal function of TCF7L2, suggests a potentially lower sensitivity to semaglutide. The decreased affinity of GLP1R may result in reduced efficacy in terms of weight loss and glycemic control compared to typical clinical outcomes. While the ARRB1 and TCF7L2 genes indicate a standard response to the medication, the decreased affinity of GLP1R may lead to diminished GLP-1 receptor signaling. This configuration could result in less pronounced effects of semaglutide, potentially including reduced appetite suppression, less improvement in glycemic control, and possibly less significant weight loss. Individuals with this profile may experience a lower response to semaglutide treatment, particularly in terms of the drug's effects on appetite and glucose metabolism. However, the normal function of TCF7L2 may help maintain some degree of responsiveness to the medication's effects on insulin secretion and glucose homeostasis.

SAMPLE REPORT

GLP1R

Decreased Affinity 0%

Decreased affinity of the GLP-1 receptor may lead to reduced activation of GLP-1 signaling pathways. This is associated with a potentially diminished response to semaglutide, possibly affecting the expected effects on appetite suppression and blood glucose regulation.

ARRB1

Normal Activity 100%

Normal activity of β-arrestin-1 plays a key role in the desensitization and internalization of the GLP-1 receptor. This contributes to the regulation of semaglutide-induced signaling, influencing the duration and intensity of the drug's effects as expected in standard clinical outcomes.

TCF7L2

Normal Function 100%

Normal function of this transcription factor maintains its usual role in glucose homeostasis and insulin secretion. This is associated with typical sensitivity to semaglutide, affecting glycemic control and weight loss as expected in standard clinical outcomes.

Saxenda

Introduction

Liraglutide (Saxenda) is a GLP-1 receptor agonist medication that aids in weight loss. It works by mimicking the action of GLP-1, a natural hormone produced in the gut after eating. This drug targets the appetite center in the brain, creating a feeling of fullness and reducing hunger. As a result, you eat less and feel satisfied for longer. Additionally, liraglutide slows down stomach emptying, which also contributes to prolonged satiety. The medication also helps regulate blood sugar levels, which can be beneficial for people with diabetes or prediabetes. Administered as daily subcutaneous injections, liraglutide, when combined with proper diet and physical activity, can significantly assist in weight reduction. The effectiveness of liraglutide may vary among individuals, partly due to genetic factors. For instance, variations in the GLP1R and ARRB1 genes can influence how well a person responds to the drug. This explains why some people might achieve better weight loss results with liraglutide than others.

Genetic Profile & Pharmacogenetic Implication [6, 7]

Overall

Lower Sensitivity

The genetic profile, including decreased affinity of GLP1R, normal activity of ARRB1, and normal function of TCF7L2, suggests a potentially lower sensitivity to liraglutide. The decreased affinity of GLP1R may result in reduced efficacy in terms of weight loss and glycemic control compared to typical clinical outcomes. While the ARRB1 and TCF7L2 genes indicate a standard response to the medication, the decreased affinity of GLP1R may lead to diminished GLP-1 receptor signaling. This configuration could result in less pronounced effects of liraglutide, potentially including reduced appetite suppression, less improvement in glycemic control, and possibly less significant weight loss. Individuals with this profile may experience a lower response to liraglutide treatment, particularly in terms of the drug's effects on appetite and glucose metabolism. However, the normal function of TCF7L2 may help maintain some degree of responsiveness to the medication's effects on insulin secretion and glucose homeostasis.

SAMPLE REPORT

GLP1R

Decreased Affinity 0%

Decreased affinity of the GLP-1 receptor may lead to reduced activation of GLP-1 signaling pathways. This is associated with a potentially diminished response to liraglutide, possibly affecting the expected effects on appetite suppression and blood glucose regulation.

ARRB1

Normal Activity 100%

Normal activity of β-arrestin-1 plays a key role in the desensitization and internalization of the GLP-1 receptor. This contributes to the regulation of liraglutide-induced signaling, influencing the duration and intensity of the drug's effects as expected in standard clinical outcomes.

TCF7L2

Normal Function 100%

Normal function of this transcription factor maintains its usual role in glucose homeostasis and insulin secretion. This is associated with typical sensitivity to liraglutide, affecting glycemic control and weight loss as expected in standard clinical outcomes.

Xenical Alli

Introduction

Orlistat (Xenical, Alli) is a medication designed to aid in weight loss by inhibiting the enzyme responsible for breaking down dietary fats in the intestines. This inhibition leads to fewer calories being absorbed from food, promoting weight loss when combined with a reduced-calorie diet. Orlistat's action is primarily localized in the gut, where it blocks the absorption of dietary fats, leading to their excretion from the body. By reducing the amount of fat absorbed, orlistat effectively lowers the caloric intake, which is key for weight reduction, particularly in individuals with obesity or related health conditions. The medication is available over-the-counter and by prescription, with the latter often used for more significant weight loss. Orlistat is administered orally, and its effects are influenced by genetic factors like FASN, CYP3A4, and CYP3A5. These genes play crucial roles in fat synthesis and drug metabolism, impacting how well orlistat performs in the body. Variations in these genes can explain why some individuals might achieve better weight loss results with orlistat than others, leading to differences in the drug's effectiveness and potential side effects like gastrointestinal discomfort, oily spotting, and increased bowel movements. Understanding these genetic influences can help tailor orlistat treatment to individual needs, ensuring optimal efficacy and minimizing adverse reactions.

Genetic Profile & Pharmacogenetic Implication [8, 9, 10]

Overall

Standard Response

With normal activity of FASN, CYP3A4, and CYP3A5, orlistat's response is balanced and predictable, ensuring standard inhibition of fat absorption, which leads to weight loss. The substance's response is characterized by a standard reduction in fat absorption, effectively inhibiting the absorption of dietary fats, leading to reduced caloric intake and weight loss, along with the usual side effects like gastrointestinal discomfort, oily spotting, and increased bowel movements, which are part of orlistat's mechanism of action.

SAMPLE REPORT

FASN

Normal Activity 100%

Fatty acid synthase (FASN) is an enzyme that synthesizes fatty acids. Normal activity of FASN ensures typical synthesis of fatty acids, which is associated with a standard response to orlistat, allowing for the usual inhibition of fat absorption in the gut.

CYP3A4

Normal Metabolism 100%

This liver enzyme plays a key role in metabolizing various substances, including orlistat. Normal activity of CYP3A4 ensures typical drug metabolism, contributing to standard pharmacokinetics of orlistat.

CYP3A5

Normal Activity 100%

Similar to CYP3A4, normal activity of CYP3A5 is involved in drug metabolism, ensuring standard clearance of orlistat from the body.

Qsymia

Introduction

Phentermine Hydrochloride and Topiramate (Qsymia) is a combination medication aimed at promoting weight loss through dual mechanisms. Phentermine, a sympathomimetic amine, acts as a central nervous system stimulant, reducing hunger by increasing the release of norepinephrine, which signals satiety to the brain. Topiramate, an anticonvulsant, enhances this effect and also induces weight loss through additional pathways, including appetite suppression, increased energy expenditure, and the modulation of neurotransmitter activity. Administered as a daily oral medication, Qsymia offers a convenient and potentially effective solution for weight management. Its efficacy, however, can vary among individuals, influenced by genetic factors like GRIK1, PLXNA4, and INSR, which play crucial roles in appetite regulation, neuronal signaling, and insulin sensitivity. Variations in these genes can explain why some individuals might achieve better weight loss results with Qsymia than others, impacting both the drug's effectiveness and potential side effects.

Genetic Profile & Pharmacogenetic Implication [11, 12, 13]

Overall

Lower Sensitivity

The genetic profile characterized by decreased activity of GRIK1, normal activity of PLXNA4, and decreased function of INSR suggests very low sensitivity to Phentermine Hydrochloride and Topiramate (Qsymia). This configuration may result in significantly reduced efficacy in terms of appetite suppression, weight loss, and glycemic control compared to typical outcomes. Individuals with this genetic profile may experience minimal effects of Qsymia, potentially including negligible appetite suppression, minimal improvement in glycemic control, and possibly insignificant weight loss. The interplay between these genetic factors indicates a response that may be significantly less than standard clinical expectations, with the potential for both reduced efficacy and standard initial response in appetite suppression

SAMPLE REPORT

GRIK1

Decreased Activity 50%

Decreased activity of the GRIK1 gene may affect the function of ionotropic glutamate receptors, potentially reducing the regulation of appetite and satiety.

PLXNA4

Normal Activity 100%

Normal activity of this gene contributes to regular neuronal signaling and synaptic plasticity, potentially affecting the response to appetite suppression.

INSR

Decreased Function 50%

Decreased function of the insulin receptor gene may alter insulin signaling pathways, potentially reducing sensitivity to Qsymia's effects on glucose metabolism and appetite regulation.

Contrave

Introduction

Naltrexone and bupropion (Contrave) is a combination medication aimed at promoting weight loss through dual mechanisms. Contrave combines naltrexone, an opioid antagonist that reduces dopamine release associated with eating pleasure, with bupropion, an antidepressant that inhibits norepinephrine and dopamine reuptake, potentially reducing hunger and cravings. Administered as a daily oral extended-release tablet, Contrave, when combined with diet and exercise, can significantly aid in weight reduction. Its effectiveness may vary among individuals due to genetic factors. Variations in genes like ANKK1, DRD2, and INSR can influence how well a person responds to the drug, explaining why some might achieve better weight loss outcomes with Contrave than others, highlighting the importance of pharmacogenetics in personalized medicine for obesity.

Genetic Profile & Pharmacogenetic Implication [14]

Overall

Complex Sensitivity

The genetic profile, characterized by normal affinity of ANKK1, normal affinity of DRD2, and decreased function of INSR, suggests a complex response to naltrexone and bupropion (Contrave). This configuration presents a mixed picture of potential drug efficacy. While normal affinity of ANKK1 and DRD2 could contribute to typical effects on reward processing and appetite suppression, the decreased function of INSR may result in potentially reduced sensitivity to the drug's effects on insulin secretion, glucose regulation, and appetite control. Individuals with this profile may experience a balanced response to the drug in terms of reducing food cravings and appetite suppression but potentially reduced efficacy in terms of glycemic control and weight loss due to the altered insulin signaling pathways.

ANKK1

Normal Affinity — 100%

Normal affinity of the ANKK1 gene is associated with typical dopamine signaling, which can influence reward processing and potentially the effectiveness of naltrexone in reducing cravings.

SAMPLE REPORT

DRD2

Normal Affinity — 100%

Normal affinity of the DRD2 gene, which codes for the dopamine receptor D2, contributes to standard dopamine receptor function. This impacts the individual's sensitivity to the effects of bupropion, which acts as a dopamine and norepinephrine reuptake inhibitor, potentially enhancing its impact on weight management.

INSR

Decreased Function — 50%

Decreased function of the insulin receptor gene (INSR) may alter its usual role in insulin signaling pathways. This can influence the metabolic effects of bupropion, particularly in terms of weight loss and glycemic control.

Adipex-P
Lomaira

Introduction

Phentermine hydrochloride, marketed as Adipex-P and Lomaira, is a sympathomimetic amine used as an appetite suppressant for short-term weight loss. It stimulates the central nervous system, leading to the release of norepinephrine, which signals satiety to the brain. Administered as a daily oral medication, phentermine, when combined with lifestyle modifications like diet and exercise, can significantly aid in weight reduction. Its effectiveness may vary among individuals due to genetic factors. Variations in genes like TAAR1, COMT, and HTR2C can influence how well a person responds to the drug, explaining why some might achieve better weight loss outcomes with phentermine than others, highlighting the importance of pharmacogenetics in personalized medicine for obesity.

Genetic Profile & Pharmacogenetic Implication [15, 16]

Overall

● Standard Response

The genetic profile, characterized by normal function of TAAR1, normal function of COMT, and normal function of HTR2C, suggests a standard response to phentermine hydrochloride (Adipex-P, Lomaira). This configuration corresponds to typical efficacy in terms of appetite suppression, weight management, and mood enhancement compared to clinical expectations. Individuals with this profile are expected to experience a balanced and predictable response to the drug, with typical effects on appetite suppression, energy regulation, and mood modulation. The interplay between these genetic factors indicates that the response to phentermine will likely align with the typical outcomes observed in clinical studies, with potential benefits in terms of weight loss and appetite control.

TAAR1

● Normal Function 100%

Normal function of the trace amine-associated receptor 1 (TAAR1) contributes to typical responsiveness to phentermine, potentially influencing the drug's effects on appetite suppression and energy regulation.

SAMPLE REPORT

COMT

● Normal Activity 100%

Normal function of the catechol-O-methyltransferase (COMT) gene contributes to standard metabolism of catecholamines, which can impact the drug's effects on mood and alertness, potentially influencing the individual's response to phentermine.

HTR2C

● Normal Affinity 100%

Normal function of the 5-hydroxytryptamine receptor 2C (HTR2C) gene maintains typical responsiveness to phentermine's effects on appetite suppression and satiety.

Imcivree

Introduction

Setmelanotide, marketed as Imcivree, is a melanocortin 4 receptor (MC4R) agonist designed to treat obesity due to specific genetic conditions like proopiomelanocortin (POMC), proprotein convertase subtilisin/kexin type 1 (PCSK1), and leptin receptor (LEPR) deficiencies. By activating the MC4R in the brain's appetite-regulating centers, setmelanotide reduces food intake and increases energy expenditure, leading to significant weight loss. Administered as a daily subcutaneous injection, its effectiveness may vary among individuals due to genetic factors. Variations in genes like POMC, PCSK1, and LEPR directly influence how well a person responds to the drug, explaining why some might achieve better weight loss outcomes with setmelanotide than others.

Genetic Profile & Pharmacogenetic Implication [17, 18]

Overall

Standard Response

The genetic profile, characterized by normal function of POMC, PCSK1, and LEPR, suggests a standard response to setmelanotide (Imcivree). This configuration corresponds to typical efficacy in terms of appetite suppression, weight management, and hunger control compared to clinical expectations. Individuals with this profile are expected to experience a balanced and predictable response to the drug, with potential benefits in terms of weight loss, hunger reduction, and improved metabolic control due to the intact melanocortin-4 receptor (MC4R) signaling pathway. Since setmelanotide directly agonizes MC4R, the drug's effects on hunger regulation and weight management are expected to be similar to those observed in clinical studies for patients with normal genetic function of POMC, PCSK1, and LEPR.

POMC

Normal Function 100%

Normal function of the proopiomelanocortin (POMC) gene contributes to the regular production of hormones like melanocyte-stimulating hormone (MSH), supporting normal hunger regulation in the brain.

SAMPLE REPORT

PCSK1

Normal Function 100%

Normal function of the proprotein convertase subtilisin/kexin type 1 (PCSK1) gene ensures proper processing of POMC into its active hormone products, including MSH, thereby maintaining standard hunger control.

LEPR

Normal Function 100%

Normal function of the leptin receptor (LEPR) gene allows leptin to signal the brain to reduce food intake and increase energy expenditure, aiding in maintaining a healthy weight.

End Of Sample Report

This is a small part of a much larger report.