



DNA Wellness Report:

Weight Management Insights

For:

Alex Morgan

KIT ID: SAMPLE-DNA-0001

Report type: Wellness

Genetic variations: 41 SNPs

Date: 2026-09-24

Dear Alex Morgan

Thank you for choosing our genetic analysis service.

We are pleased to provide you with personalized information based on your genetic data. This report is designed to offer educational insights into selected genetic variants and their associations described in scientific literature.

Our goal is to present your results in a clear and informative format to support a better understanding of certain genetic characteristics related to general wellness. This information is intended for educational purposes only and is not intended to diagnose, treat, cure, or prevent any disease.

We hope your experience with our service has been clear, informative, and valuable. If you have any questions or need additional assistance, our team is available to help.

Thank you again for placing your trust in us.

Sincerely,

MAGISNAT OMICS LLC Team

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Introduction

GENETICS is important

DNA Wellness Report: Weight Management Insights

Weight management-related pathways include biological processes influenced by both lifestyle factors and genetic variation. This DNA report analyzes **41 selected genetic variants (SNPs)** that have been studied in relation to biological pathways associated with nutrient metabolism, energy balance, fat distribution, diet response, and body composition-related traits. Scientific literature suggests that genetic variation may be associated with differences in certain weight-related and body composition-related processes among individuals. The information in this report is provided for educational and general wellness purposes and is intended to offer context about genetic variation and weight-related biological pathways. **This report is not intended to diagnose, treat, cure, or prevent any disease.**

Traits



Diet Response Insights



Fat Distribution Insights

Understanding the report

How to read your genetic results

This report presents information about selected genetic variants identified through the analysis of specific genes and their variations, known as single nucleotide polymorphisms (SNPs).

Each result is displayed in a dedicated section that includes the gene name, a description of its biological role, the specific SNP analyzed, and the genotype identified (alleles).

The information provided in this report is based on findings from published scientific research describing associations between certain genetic variants and biological processes.

The content of this report is provided for educational and informational purposes only and is not intended to diagnose, treat, cure, or prevent any disease.

Report summary

Diet Response Insights

Gene:	SNP:	Alleles:	Page
MTHFR	rs1801133	G/A	6
MTHFR	rs1801131	T/G	7
FUT2	rs602662	G/G	7
GC	rs2282679	G/G	8
TMPRSS6	rs855791	A/A	8
ADIPOQ	rs266729	C/C	9
LEP	rs7799039	G/G	10
UCP3	rs1800849	A/A	10
PPARG	rs1801282	C/G	11
FTO	rs1558902	A/A	11
APOA2	rs5082	G/A	12
APOA5	rs662799	A/G	12
PPARGC1A	rs8192678	T/T	13
GHSR	rs490683	C/C	14
ADIPOQ	rs17300539	A/A	15
ADIPOQ	rs3774261	G/G	16
AMY1-AMY2	rs11185098	G/G	17

BMI-related traits

Gene:	SNP:	Alleles:	Page
TFAP2B	rs987237	A/A	18
TFAP2B	rs2272903	G/A	19

Fat Distribution Insights

Gene:	SNP:	Alleles:	Page
MAP2K5	rs2241423	A/G	20
MAP2K5	rs997295	G/T	21
FTO	rs8050136	C/C	21
FTO	rs3751812	T/T	22
ADIPOQ	rs182052	A/A	22
Intergenic (near TFAP2B)	rs734597	G/G	23
FTO	rs62033400	G/G	23
FTO	rs17817449	T/G	24
FTO	rs9936385	T/C	24
FTO	rs7202116	A/A	25
FTO	rs9930506	A/A	25
FTO	rs12149832	A/A	26
FTO	rs1121980	G/G	27

Genetic Data Results

Diet Response Insights

Diet response-related pathways include biological processes associated with fat metabolism, carbohydrate metabolism, and other diet-related traits. This section presents information about selected genetic variants that have been studied in relation to biological pathways associated with diet response. Scientific literature suggests that genetic variation may be associated with differences in certain diet-related processes among individuals. The information in this section is provided for educational and general wellness purposes and is intended to offer context about genetic variation and diet-related biological pathways.

Reference:

Singar, Saiful et al. "Personalized Nutrition: Tailoring Dietary Recommendations through Genetic Insights." *Nutrients* vol. 16,16 2673. 13 Aug.2024, doi:10.3390/nu16162673

Your results

Gene:

MTHFR

SNP:

rs1801133

Alleles:

G/A

Methylenetetrahydrofolate reductase.

Enzyme involved in folate metabolism that converts 5,10-methylenetetrahydrofolate to 5-methyltetrahydrofolate and contributes to homocysteine remethylation.

Possible moderately reduced MTHFR activity. [\[1-3\]](#)

The MTHFR gene encodes methylenetetrahydrofolate reductase, an enzyme involved in folate metabolism. Functional and dietary-intervention studies have explored whether rs1801133 is associated with differences in MTHFR activity and folate-related response.[\[1-3\]](#) Having one copy (heterozygosity) of the A allele, corresponding to the G/A genotype, is associated with moderately reduced MTHFR activity compared with the G/G genotype.[\[1\]\[2\]](#) In an 18-month dietary intervention, individuals with the G/G and G/A genotypes, considered together, showed greater increases in serum folate following a green-Mediterranean dietary intervention than individuals with the A/A genotype.[\[3\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

MTHFR

SNP:

rs1801131

Alleles:

T/G

Methylenetetrahydrofolate reductase.

Enzyme involved in folate metabolism that converts 5,10-methylenetetrahydrofolate to 5-methyltetrahydrofolate and contributes to homocysteine remethylation.

Possible modest reduction in MTHFR enzyme activity. [\[4-6\]](#)

The MTHFR gene encodes methylenetetrahydrofolate reductase, an enzyme involved in folate metabolism. Genetic and functional studies have explored whether rs1801131 is associated with differences in MTHFR activity and folate-related measures.[\[4-6\]](#) Having one copy (heterozygosity) of the G allele, corresponding to the T/G genotype, is associated with a possible modest reduction in MTHFR activity compared with the T/T genotype.[\[4-6\]](#) However, this variant alone has not been consistently associated with lower circulating folate or higher homocysteine levels.[\[4\]\[6\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

FUT2

SNP:

rs602662

Alleles:

G/G

Fucosyltransferase 2.

Enzyme that adds fucose to glycans on mucosal surfaces and contributes to secretor status.

Possible lower circulating vitamin B12 levels. [\[7\]\[8\]](#)

The FUT2 gene encodes fucosyltransferase 2, an enzyme involved in glycan fucosylation and secretor status. Genetic association studies have explored whether rs602662 is associated with circulating vitamin B12 levels.[\[7\]\[8\]](#) Having two copies (homozygosity) of the G allele, corresponding to the G/G genotype, is included within the G-allele-associated profile linked to relatively lower circulating vitamin B12 levels compared with the A/A genotype; however, available evidence does not establish a stronger effect for G/G compared with G/A.[\[7\]\[8\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

GC

SNP:

rs2282679

Alleles:

G/G

Vitamin D-binding protein.

Protein that binds vitamin D and its metabolites in plasma and transports them to target tissues.

Possible lower circulating 25-hydroxyvitamin D levels. [\[9\]\[10\]](#)

The GC gene encodes vitamin D-binding protein, which binds and transports vitamin D and its metabolites in the bloodstream. Genome-wide genetic association studies have explored whether rs2282679 is associated with circulating 25-hydroxyvitamin D [25(OH)D] levels. [\[9\]\[10\]](#) Having two copies (homozygosity) of the G allele, corresponding to the G/G genotype, is associated with relatively lower circulating 25(OH)D levels compared with the T/T genotype. [\[9\]\[10\]](#) This genetic result does not determine individual vitamin D levels, body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

TMPRSS6

SNP:

rs855791

Alleles:

A/A

Transmembrane protease serine 6 or Matriptase-2.

Protein playing a critical role in the regulation of iron homeostasis in the body.

Possible lower iron-related measures and reduced iron absorption. [\[11-13\]](#)

The TMPRSS6 gene encodes transmembrane serine protease 6, also known as matriptase-2, a protein involved in the regulation of hepcidin and iron absorption. Genetic, functional, and oral iron-absorption studies have explored whether rs855791 is associated with differences in iron-related measures. [\[11-13\]](#) Having two copies (homozygosity) of the A allele, corresponding to the A/A genotype, is associated with relatively lower iron-related measures compared with the G/G genotype. [\[11-13\]](#) In a study measuring oral iron absorption, the A/A genotype, was associated with lower serum iron and transferrin saturation and, after accounting for iron status, with reduced fractional iron absorption compared with the G/G genotype. [\[13\]](#) This genetic result does not determine individual iron status, body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

ADIPOQ

SNP:

rs266729

Alleles:

C/C

Adiponectin.

Hormone produced mainly by adipose tissue that contributes to the regulation of glucose and lipid metabolism, insulin sensitivity, and energy balance.

Possible more favorable improvements in adiponectin and insulin-related metabolic measures following a hypocaloric dietary intervention. [\[14-16\]](#)

The ADIPOQ gene encodes adiponectin, a hormone produced by adipose tissue that is involved in insulin sensitivity and metabolic regulation.[\[14\]](#)[\[15\]](#) A hypocaloric Mediterranean-type dietary intervention study explored whether rs266729 is associated with differences in adiponectin and insulin-related metabolic response.[\[16\]](#) Having two copies (homozygosity) of the C allele, corresponding to the C/C genotype, was associated with more favorable improvements in adiponectin, insulin and insulin-resistance-related measures compared with genotypes carrying the G allele.[\[16\]](#) Reductions in body weight and other adiposity measures occurred in both genotype groups.[\[16\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

LEP

SNP:

rs7799039

Alleles:

G/G

Leptin.

The LEP gene encodes leptin, a hormone produced mainly by adipose tissue that helps regulate appetite, energy balance, and body weight.

Possible relatively lower insulin and insulin-resistance-related measures. [\[17\]](#)

The LEP gene encodes leptin, a hormone produced mainly by adipose tissue that helps regulate appetite and energy balance. A meta-analysis of multiple human studies explored whether rs7799039 is associated with circulating leptin and insulin-related measures.[\[17\]](#) Having two copies (homozygosity) of the G allele, corresponding to the G/G genotype, was associated with relatively lower insulin and insulin-resistance-related measures compared with carriers of the A allele, and differences in circulating leptin levels were also reported between genotype groups.[\[17\]](#) This genetic result does not determine individual appetite, body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

UCP3

SNP:

rs1800849

Alleles:

A/A

Uncoupling Protein 3.

Protein present in mitochondria and involved in energy balance.

Possible less favorable weight-loss and metabolic response to hypocaloric dietary interventions. [\[18\]](#)

The UCP3 gene encodes uncoupling protein 3, a mitochondrial protein expressed mainly in skeletal muscle and involved in energy metabolism. A hypocaloric dietary-intervention study explored whether rs1800849 is associated with differences in weight-loss and metabolic response.[\[18\]](#) Having two copies (homozygosity) of the A allele, corresponding to the A/A genotype, is included within the A-allele-carrier group that showed less weight improvement and less favorable changes in selected metabolic measures compared with G/G; however, the study does not establish a stronger effect for A/A compared with A/G.[\[18\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

PPARG

SNP:

rs1801282

Alleles:

C/G

Peroxisome proliferator-activated receptor gamma.

Receptor that regulates fatty acid deposition and glucose metabolism.

Possible less favorable weight-loss and metabolic response to a hypocaloric dietary intervention. [\[19-21\]](#)

The PPARG gene encodes peroxisome proliferator-activated receptor gamma, a protein involved in adipocyte function, lipid metabolism, and glucose-related processes.[\[20-21\]](#) A hypocaloric dietary-intervention study explored whether rs1801282 is associated with differences in weight-loss and metabolic response.[\[19\]](#) Having one copy (heterozygosity) of the G allele, corresponding to the C/G genotype, is included within the G-allele-carrier group that showed less favorable reductions in body weight, fat mass, waist circumference and selected metabolic measures compared with C/C.[\[19-21\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

FTO

SNP:

rs1558902

Alleles:

A/A

FTO alpha-ketoglutarate dependent dioxygenase.

Enzyme involved in RNA modification and cellular processes related to energy balance and metabolism.

Possible greater weight loss and reductions in several body-fat measures in response to a high-protein diet. [\[22\]](#)

The FTO gene encodes an enzyme involved in RNA modification and cellular processes related to energy balance and metabolism. A dietary-intervention study explored whether rs1558902 is associated with differences in response to weight-loss diets.[\[22\]](#) Having two copies (homozygosity) of the A allele, corresponding to the A/A genotype, was associated with greater weight loss and greater reductions in several body-fat measures among individuals following a high-protein weight-loss diet.[\[22\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

APOA2

SNP:

rs5082

Alleles:

G/A

Apolipoprotein A-II.

Component of blood lipoproteins that plays a crucial role in lipid transport in the body.

No specific saturated-fat-related BMI association established for this genotype. [\[23-25\]](#)

The APOA2 gene encodes apolipoprotein A-II, a protein involved in lipid transport and metabolism. Observational studies across several populations have explored whether rs5082 interacts with saturated-fat intake in relation to BMI and body-weight-related measures.[\[23-25\]](#) Having one copy (heterozygosity) of the G allele, corresponding to the G/A genotype, does not have the G/G genotype, in which the higher-BMI association under relatively high saturated-fat intake was primarily observed, and available evidence does not establish the same saturated-fat-related BMI association for G/A.[\[23-25\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

APOA5

SNP:

rs662799

Alleles:

A/G

Apolipoprotein A-V.

Component of blood lipoproteins that plays a crucial role in lipid transport in the body.

Possible intermediate fasting triglyceride levels. [\[26-28\]](#)

The APOA5 gene encodes apolipoprotein A-V, a protein involved in the regulation of triglyceride metabolism. Genetic association studies have explored whether rs662799 is associated with fasting triglyceride levels.[\[26-28\]](#) Having one copy (heterozygosity) of the G allele, corresponding to the A/G genotype, is associated with triglyceride levels that are generally intermediate between those observed in G/G and A/A individuals.[\[26\]\[28\]](#) This genetic result does not determine individual triglyceride levels, body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

PPARGC1A

SNP:

rs8192678

Alleles:

T/T

Peroxisome proliferator-activated receptor gamma coactivator 1 alpha.

Transcriptional coactivator regulating the expression of genes involved in energy metabolism, mitochondrial biogenesis, and adaptive thermogenesis.

Possible differences in metabolic response to energy-restricted dietary interventions. [\[31\]](#)[\[32\]](#)

The PPARGC1A gene encodes a transcriptional coactivator involved in energy metabolism, mitochondrial function and adaptive thermogenesis. Dietary-intervention studies have explored whether rs8192678 is associated with metabolic response to energy-restricted diets.[\[31\]](#)[\[32\]](#) Having two copies (homozygosity) of the T allele, corresponding to the T/T genotype, is included within the T-allele profile associated with greater reductions in total and LDL cholesterol than C/C during a low-fat energy-restricted diet, and T/T individuals also showed improvement in insulin and insulin-resistance-related measures in a separate low-calorie intervention.[\[31\]](#)[\[32\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

GHSR

SNP:

rs490683

Alleles:

C/C

Growth hormone secretagogue receptor.

Receptor that binds ghrelin, a hormone involved in the regulation of appetite, hunger, and growth hormone release.

Possible differences in weight-loss response during lifestyle and hypocaloric dietary interventions. [\[33\]](#)[\[34\]](#)

The GHSR gene encodes the growth hormone secretagogue receptor, which mediates ghrelin signaling involved in appetite and energy balance. Lifestyle and hypocaloric dietary-intervention studies have explored whether rs490683 is associated with differences in weight-loss and metabolic response.[\[33\]](#)[\[34\]](#) Having two copies (homozygosity) of the C allele, corresponding to the C/C genotype, was associated with a more favorable weight-loss response than G-allele carriers and, in a recent hypocaloric intervention, with greater improvements in fat mass, waist circumference and selected glucose- and lipid-related metabolic measures.[\[33\]](#)[\[34\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

ADIPOQ

SNP:

rs17300539

Alleles:

A/A**Adiponectin.**

Hormone produced mainly by adipose tissue that contributes to the regulation of glucose and lipid metabolism, insulin sensitivity, and energy balance.

Possible association with an A-allele-related adiponectin and metabolic profile; an A/A-specific weight-loss response has not been established. [\[35-37\]](#)

The ADIPOQ gene encodes adiponectin, a hormone produced by adipose tissue that contributes to insulin sensitivity and glucose and lipid metabolism. Observational and lifestyle-intervention studies have explored whether rs17300539 is associated with adiponectin-related metabolic traits and weight-loss response. [\[35-37\]](#) Having two copies (homozygosity) of the A allele, corresponding to the A/A genotype, is included within the A-allele profile associated with higher adiponectin levels and differences in metabolic and body-weight-related measures. [\[35\]](#) [\[36\]](#) In one hypocaloric diet and physical exercise intervention, a higher mean weight reduction was observed among only two A/A participants, so an A/A-specific association with weight-loss response has not been established. [\[37\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

ADIPOQ

SNP:

rs3774261

Alleles:

G/G

Adiponectin.

Hormone produced mainly by adipose tissue that contributes to the regulation of glucose and lipid metabolism, insulin sensitivity, and energy balance.

Possible differences in metabolic response during hypocaloric Mediterranean-pattern dietary interventions.
[\[38\]](#)[\[39\]](#)

The ADIPOQ gene encodes adiponectin, a hormone produced by adipose tissue that contributes to insulin sensitivity and glucose and lipid metabolism. Hypocaloric Mediterranean-pattern dietary-intervention studies have explored whether rs3774261 is associated with metabolic response.[\[38\]](#)[\[39\]](#) Having two copies (homozygosity) of the G allele, corresponding to the G/G genotype, is included within the G-allele-carrier group that showed less pronounced improvements in adiponectin, lipid profile, and CRP compared with A/A; however, available evidence does not establish a progressively less favorable response for G/G compared with A/G, and weight loss itself was similar across genotype groups.[\[38\]](#)[\[39\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Carbohydrate Metabolism

Gene:

AMY1-AMY2

SNP:

rs11185098

Alleles:

G/G

Alpha-Amylase.

Proteins involved in the first steps of digestion of carbohydrates in saliva.

No specific A-allele-associated weight-loss advantage established for this genotype. [\[61\]](#)

The AMY1-AMY2 gene region includes genes involved in starch digestion. A 2-year calorie-restricted dietary-intervention study explored whether rs11185098 is associated with differences in weight-management response.[\[61\]](#) Having two copies (homozygosity) of the G allele, corresponding to the G/G genotype, does not carry the A allele associated with greater reductions in body weight and waist circumference, and no specific A-allele-associated weight-loss advantage was established for this genotype.[\[61\]](#) This genetic result does not determine an individual's response, which is influenced by multiple genetic, dietary, lifestyle, and environmental factors. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Your notes

BMI-related traits

Diet Response

Your results

Gene:

TFAP2B

SNP:

rs987237

Alleles:

A/A

Transcription factor AP-2 beta.

Transcription factor regulating genes that control cell growth, differentiation, and apoptosis (programmed cell death).

Possible more favorable weight-maintenance response to a high-protein diet. [\[15\]](#)[\[29\]](#)

The TFAP2B gene encodes transcription factor AP-2 beta, a protein involved in the regulation of gene expression and pathways related to adipocyte function and energy balance.[\[15\]](#) A dietary-intervention trial explored whether rs987237 is associated with weight maintenance after initial weight loss.[\[29\]](#) Having two copies (homozygosity) of the A allele, corresponding to the A/A genotype, was associated with more favorable weight maintenance when following a high-protein diet, whereas this benefit was not established in individuals carrying the G allele.[\[29\]](#) This genetic result does not determine individual body weight or the ability to maintain weight loss. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

TFAP2B

SNP:

rs2272903

Alleles:

G/A

Transcription factor AP-2 beta.

Transcription factor regulating genes that control cell growth, differentiation, and apoptosis (programmed cell death).

Possible modest genetic tendency toward higher BMI. [\[30\]](#)

The TFAP2B gene encodes transcription factor AP-2 beta, a protein involved in the regulation of gene expression and pathways related to adipocyte function and energy balance. A large genetic association study explored whether rs2272903 is associated with BMI and reported a small increase in BMI for each copy of the G allele.[\[30\]](#) Having one copy (heterozygosity) of the G allele, corresponding to the G/A genotype, is consistent with carrying one copy of the BMI-associated allele and a possible modest BMI-related genetic tendency for this variant.[\[30\]](#) This genetic result does not determine individual body weight or the ability to maintain weight loss. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Fat Distribution Insights

Fat distribution-related pathways include biological processes associated with body fat distribution and body composition. This section presents information about selected genetic variants that have been studied in relation to biological pathways associated with fat distribution. Scientific literature suggests that genetic variation may be associated with differences in certain body composition-related processes among individuals. The information in this section is provided for educational and general wellness purposes and is intended to offer context about genetic variation and fat distribution-related biological pathways.

Reference:

Agrawal, Saaket, et al. "Inherited Basis of Visceral, Abdominal Subcutaneous and Gluteofemoral Fat Depots." *Nature Communications*, vol. 13, article no. 3771, 30 June 2022, <https://doi.org/10.1038/s41467-022-30931>

Your results

Gene:

MAP2K5

SNP:

rs2241423

Alleles:

A/G

Mitogen-activated protein kinase kinase 5.

Enzyme involved in the response of the cell to external stimuli.

Possible small BMI-associated genetic tendency for this variant. [\[40\]\[41\]](#)

The MAP2K5 gene encodes a kinase involved in intracellular signaling pathways that contribute to the regulation of cellular responses to external signals. Large genetic association studies have explored whether rs2241423 is associated with BMI and identified the G allele as being associated with slightly higher BMI, with an additive or per-allele pattern across genotypes.[\[40\]\[41\]](#) Having one copy (heterozygosity) of the G allele, corresponding to the A/G genotype, is consistent with a possible modest BMI-related genetic tendency for this variant.[\[40\]\[41\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

MAP2K5

SNP:

rs997295

Alleles:

G/T

Mitogen-activated protein kinase kinase 5.

Enzyme involved in the response of the cell to external stimuli.

Possible modest genetic tendency toward higher BMI. [\[30\]](#)[\[42\]](#)

The MAP2K5 gene encodes a kinase involved in intracellular signaling pathways that contribute to cellular responses and processes related to energy balance. Large genetic association studies have explored whether rs997295 is associated with BMI and identified the T allele as being associated with slightly higher BMI, with an additive or per-allele pattern across genotypes.[\[30\]](#) Having one copy (heterozygosity) of the T allele, corresponding to the G/T genotype, is consistent with a possible modest BMI-related genetic tendency for this variant.[\[30\]](#) Additional research suggests that the association of rs997295 may become more pronounced at higher levels of BMI.[\[42\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

FTO

SNP:

rs8050136

Alleles:

C/C

FTO alpha-ketoglutarate dependent dioxygenase.

Enzyme involved in RNA modification and cellular processes related to energy balance and metabolism.

Possible relatively lower BMI and body-fat-related genetic tendency. [\[43\]](#)[\[44\]](#)

The FTO gene encodes an enzyme involved in RNA modification and cellular processes related to energy balance and metabolism. Genetic association studies have explored whether rs8050136 is associated with BMI and body-fat-related measures and identified the A allele as the higher-BMI-associated allele.[\[43\]](#)[\[44\]](#) Having two copies (homozygosity) of the C allele, corresponding to the C/C genotype, does not carry the BMI-associated A allele and is consistent with a possible relatively lower BMI- and body-fat-related genetic tendency for this variant.[\[43\]](#)[\[44\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

FTO

SNP:

rs3751812

Alleles:

T/T

FTO alpha-ketoglutarate dependent dioxygenase.

Enzyme involved in RNA modification and cellular processes related to energy balance and metabolism.

Possible greater genetic tendency toward higher BMI. [\[45\]](#)[\[46\]](#)

The FTO gene encodes an enzyme involved in RNA modification and cellular processes related to energy balance and metabolism. Large genetic association studies have explored whether rs3751812 is associated with BMI and identified the T allele as being associated with slightly higher BMI, with an additive or per-allele pattern across genotypes.[\[45\]](#)[\[46\]](#) Having two copies (homozygosity) of the T allele, corresponding to the T/T genotype, is consistent with a possible greater BMI-related genetic tendency for this variant.[\[45\]](#)[\[46\]](#) Physical activity has been reported to attenuate the BMI-related association of this variant.[\[46\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

ADIPOQ

SNP:

rs182052

Alleles:

A/A

Adiponectin.

Hormone produced mainly by adipose tissue that contributes to the regulation of glucose and lipid metabolism, insulin sensitivity, and energy balance.

Possible waist-circumference-associated difference for this variant. [\[47\]](#)

The ADIPOQ gene encodes adiponectin, a hormone produced by adipose tissue that contributes to insulin sensitivity and glucose and lipid metabolism. An observational study explored whether rs182052 is associated with body-size measures and reported progressively higher waist circumference, body weight, and BMI with increasing numbers of the A allele.[\[47\]](#) Having two copies (homozygosity) of the A allele, corresponding to the A/A genotype, is associated with the higher waist-circumference, body-weight, and BMI profile reported with increasing numbers of the A allele.[\[47\]](#) This genetic result does not determine individual body weight or fat distribution. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

Intergenic (near TFAP2B)

SNP:

rs734597

Alleles:

G/G

Intergenic region near TFAP2B.

Intergenic region near TFAP2B; rs734597 has been associated with BMI, but a direct functional effect has not been established.

Possible relatively lower BMI-related genetic tendency. [\[40\]\[48\]](#)

The rs734597 variant is located in an intergenic region near TFAP2B, a genomic region that has been associated with BMI in large genetic studies.[\[40\]\[48\]](#) Large genetic association studies have explored whether this region is associated with BMI and identified the rs734597 A allele as the BMI-increasing allele.[\[40\]\[48\]](#) Having two copies (homozygosity) of the G allele, corresponding to the G/G genotype, does not carry the BMI-increasing A allele and is consistent with a possible relatively lower BMI-related genetic tendency for this variant.[\[48\]](#) A direct functional effect of rs734597 has not been established. This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

FTO

SNP:

rs62033400

Alleles:

G/G

FTO alpha-ketoglutarate dependent dioxygenase.

Enzyme involved in RNA modification and cellular processes related to energy balance and metabolism.

Possible greater genetic tendency toward higher BMI. [\[49\]\[50\]](#)

The FTO gene encodes an enzyme involved in RNA modification and cellular processes related to energy balance and metabolism. Large genetic association studies have explored whether rs62033400 is associated with BMI and identified the G allele as being associated with slightly higher BMI, with an additive or per-allele pattern across genotypes.[\[49\]\[50\]](#) Having two copies (homozygosity) of the G allele, corresponding to the G/G genotype, is consistent with a possible greater BMI-related genetic tendency for this variant.[\[49\]\[50\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

FTO

SNP:

rs17817449

Alleles:

T/G

FTO alpha-ketoglutarate dependent dioxygenase.

Enzyme involved in RNA modification and cellular processes related to energy balance and metabolism.

Possible modest genetic tendency toward higher BMI. [\[51\]\[52\]](#)

The FTO gene encodes an enzyme involved in RNA modification and cellular processes related to energy balance and metabolism. Large genetic association studies have explored whether rs17817449 is associated with BMI and identified the G allele as being associated with slightly higher BMI, with an additive or per-allele pattern across genotypes.[\[51\]\[52\]](#) Having one copy (heterozygosity) of the G allele, corresponding to the T/G genotype, is consistent with a possible modest BMI-related genetic tendency for this variant.[\[51\]\[52\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

FTO

SNP:

rs9936385

Alleles:

T/C

FTO alpha-ketoglutarate dependent dioxygenase.

Enzyme involved in RNA modification and cellular processes related to energy balance and metabolism.

Possible modestly lower lean-mass-related measures. [\[53\]\[54\]](#)

The FTO gene encodes an enzyme involved in RNA modification and cellular processes related to energy balance and metabolism. Large genetic and smaller population studies have explored whether rs9936385 is associated with lean-mass and skeletal-muscle-related measures.[\[53\]\[54\]](#) Having one copy (heterozygosity) of the T allele, corresponding to the T/C genotype, is consistent with a possible modestly lower lean-mass-related profile based on a per-allele association, although the smaller study identified the clearer lower-limb skeletal-muscle association in women with T/T.[\[53\]\[54\]](#) This genetic result does not determine individual muscle mass, body composition or body weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

FTO

SNP:

rs7202116

Alleles:

A/A

FTO alpha-ketoglutarate dependent dioxygenase.

Enzyme involved in RNA modification and cellular processes related to energy balance and metabolism.

Possible relatively lower BMI-related genetic tendency. [\[40\]\[55\]](#)

The FTO gene encodes an enzyme involved in RNA modification and cellular processes related to energy balance and metabolism. Large genetic association studies at the FTO locus have explored whether rs7202116 is associated with BMI and identified the G allele as being associated with slightly higher BMI, with an additive or per-allele pattern across genotypes. [\[40\]\[55\]](#) Having two copies (homozygosity) of the A allele, corresponding to the A/A genotype, does not carry the BMI-associated G allele and is consistent with a possible relatively lower BMI-related genetic tendency for this variant. [\[40\]\[55\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

FTO

SNP:

rs9930506

Alleles:

A/A

FTO alpha-ketoglutarate dependent dioxygenase.

Enzyme involved in RNA modification and cellular processes related to energy balance and metabolism.

Possible relatively lower BMI-, body-weight-, and hip-circumference-related genetic tendency. [\[45\]\[56\]](#)

The FTO gene encodes an enzyme involved in RNA modification and cellular processes related to energy balance and metabolism. Genetic association studies have explored whether rs9930506 is associated with body-size measures and identified the G allele as being associated with higher BMI, body weight, and hip circumference in an additive pattern. [\[45\]\[56\]](#) Having two copies (homozygosity) of the A allele, corresponding to the A/A genotype, does not carry the G allele associated with higher BMI, body weight, and hip circumference and is consistent with a possible relatively lower body-size-related genetic tendency for this variant. [\[45\]\[56\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

FTO

SNP:

rs12149832

Alleles:

A/A

FTO alpha-ketoglutarate dependent dioxygenase.

Enzyme involved in RNA modification and cellular processes related to energy balance and metabolism.

Possible greater genetic tendency toward higher BMI. [\[57\]](#)[\[58\]](#)

The FTO gene encodes an enzyme involved in RNA modification and cellular processes related to energy balance and metabolism. Large genetic association studies have explored whether rs12149832 is associated with BMI and identified the A allele as being associated with slightly higher BMI, with an additive or per-allele pattern across genotypes.[\[57\]](#)[\[58\]](#) Having two copies (homozygosity) of the A allele, corresponding to the A/A genotype, is consistent with a possible greater BMI-related genetic tendency for this variant.[\[57\]](#)[\[58\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Gene:

FTO

SNP:

rs1121980

Alleles:

G/G

FTO alpha-ketoglutarate dependent dioxygenase.

Enzyme involved in RNA modification and cellular processes related to energy balance and metabolism.

Possible relatively lower BMI- and waist-circumference-related genetic tendency. [\[45\]\[59\]\[60\]](#)

The FTO gene encodes an enzyme involved in RNA modification and cellular processes related to energy balance and metabolism. Genetic association studies have explored whether rs1121980 is associated with BMI and waist circumference and identified the A allele used in this report as the higher-BMI-associated allele.[\[45\]\[59\]\[60\]](#) Having two copies (homozygosity) of the G allele, corresponding to the G/G genotype, does not carry the A allele associated with higher BMI and waist circumference and is consistent with a possible relatively lower BMI- and waist-related genetic tendency for this variant.[\[45\]\[59\]\[60\]](#) Physical activity has been reported to attenuate the BMI- and waist-related association of this variant.[\[60\]](#) This genetic result does not determine individual body weight or the ability to lose weight. A qualified healthcare professional can help assess whether your overall diet and lifestyle are appropriate for your individual needs.

Your notes

Scientific Glossary

When discussing genetics, it's often necessary to use many technical terms, and there's no way to avoid it if we want to maintain accuracy in explanations. That's why we have compiled a scientific glossary - to enable everyone to understand without getting overwhelmed.

Anyway, it is important to emphasize that our scientific glossary does not aim to be exhaustive and is not intended to replace the advice provided by your healthcare provider. Professional medical support is essential for a proper interpretation of genetic data and for developing a personalized health and wellness plan.

Allele

An allele is one of the different forms of a specific gene. The differences among alleles arise from small changes in the DNA sequence and can lead to changes in the characteristic controlled by the gene itself.

Chromosome

An allele is one of the different forms of a specific gene. The differences among alleles arise from small changes in the DNA sequence and can lead to changes in the characteristic controlled by the gene itself.

Dietary supplement

A dietary supplement is a product that contains one or more dietary ingredients, such as vitamins, minerals, herbs, amino acids, enzymes, or other substances, intended to supplement the diet. These supplements are available in various forms, including pills, capsules, tablets, powders, or liquids.

DNA

DNA stands for Deoxyribonucleic Acid. It is the macromolecule containing the information to build the organism. It is made up of 4 different nucleotides (Adenine, Cytosine, Guanine and Thymine). The human DNA have 3 billion nucleotide basepairs.

Gene

A gene is a segment of a chromosome that occupies a given locus on it and codes for a protein, each one with a specific function: some build the structure of our cells, some respond to signaling molecules, some catalyze reactions (these are called enzymes), and so on.

Genetic Variant

A genetic variant is a change or alteration in the DNA sequence of a gene. The main genetic variant types include base substitutions, deletions, or insertions.

Genomics

Genomics is a field of biology that focuses on the study of an organism's entire genome, which is the complete set of its genetic material. It involves the comprehensive analysis of genes, their functions, interactions, and variations within and between populations.

Genotype

The genotype is the genetic makeup of an organism, then the combination of alleles presents in an individual's DNA at a particular locus on a chromosome. The genotype represents the specific genetic information that an organism inherits from its parents.

Heterozygosity

Heterozygosity refers to having two different alleles at a specific genetic locus. If an individual has one copy of the "A" allele and one copy of the "B" allele for a certain gene (AB genotype), they are said to be heterozygous for that gene.

Homozygosity

Homozygosity refers to having two identical alleles at a specific genetic locus. If an individual has two copies of the "A" allele for a certain gene (AA genotype), they are said to be homozygous for that gene.

Macronutrient

Macronutrients are essential nutrients that are required by the body in large quantities to maintain proper functioning, growth, and overall health. These nutrients provide the necessary energy and building blocks needed for various physiological processes. The three primary macronutrients are: carbohydrates, lipids (fat), and proteins.

Micronutrient

Micronutrients are essential nutrients required by the body in smaller quantities but are equally important for maintaining overall health and supporting various physiological functions. Micronutrients include two main groups: vitamins and minerals.

Nutritional deficiency

Nutritional deficiency, also known as malnutrition, refers to a condition in which the body does not receive enough macronutrients or micronutrients, which are needed to support proper growth, development, and overall wellness.

Phenotype

The phenotype is any observable trait arising from a complex interplay between a given genotype and environmental factors. Examples of phenotypes are height, eye color and blood type.

rsID number

rsID numbers are identifiers used by researchers to name different SNPs.

SNPs (Single Nucleotide Polymorphism)

A SNP, or single nucleotide polymorphism, is a genetic variant in one of the nucleotide bases composing DNA and found in more than 1% of the population.

Scientific References

- [1] Micheletti, C., et al. "Nutrigenomics: SNPs Correlated to Vitamins' Deficiencies." *La Clinica Terapeutica*, vol. 174, suppl. 2, no. 6, 2023, pp. 173–182. <https://doi.org/10.7417/CT.2023.2485>.
- [2] Frosst, P., et al. "A Candidate Genetic Risk Factor for Vascular Disease: A Common Mutation in Methylenetetrahydrofolate Reductase." *Nature Genetics*, vol. 10, no. 1, 1995, pp. 111–113. <https://doi.org/10.1038/ng0595-111>.
- [3] Zelicha, H., et al. "Effect of Green Mediterranean Diet on Serum Folate and Its Interaction with Genetic Variation in Folate Metabolism: The DIRECT PLUS 18-Month Dietary Randomized Controlled Trial." *Clinical Nutrition*, vol. 63, 2026, article 106701. <https://doi.org/10.1016/j.clnu.2026.106701>.
- [4] van der Put, N. M. J., et al. "A Second Common Mutation in the Methylenetetrahydrofolate Reductase Gene: An Additional Risk Factor for Neural-Tube Defects?" *American Journal of Human Genetics*, vol. 62, no. 5, 1998, pp. 1044–1051. <https://doi.org/10.1086/301825>.
- [5] Weisberg, I., et al. "A Second Genetic Polymorphism in Methylenetetrahydrofolate Reductase (MTHFR) Associated with Decreased Enzyme Activity." *Molecular Genetics and Metabolism*, vol. 64, no. 3, 1998, pp. 169–172. <https://doi.org/10.1006/mgme.1998.2714>.
- [6] Weisberg, I. S., et al. "The 1298A→C Polymorphism in Methylenetetrahydrofolate Reductase (MTHFR): In Vitro Expression and Association with Homocysteine." *Atherosclerosis*, vol. 156, no. 2, 2001, pp. 409–415. [https://doi.org/10.1016/S0021-9150\(00\)00671-7](https://doi.org/10.1016/S0021-9150(00)00671-7).
- [7] Hazra, A., et al. "Common Variants of FUT2 Are Associated with Plasma Vitamin B12 Levels." *Nature Genetics*, vol. 40, no. 10, 2008, pp. 1160–1162. <https://doi.org/10.1038/ng.210>.
- [8] Tanaka, T., et al. "Genome-Wide Association Study of Vitamin B6, Vitamin B12, Folate, and Homocysteine Blood Concentrations." *American Journal of Human Genetics*, vol. 84, no. 4, 2009, pp. 477–482. <https://doi.org/10.1016/j.ajhg.2009.02.011>.
- [9] Ahn, J., et al. "Genome-Wide Association Study of Circulating Vitamin D Levels." *Human Molecular Genetics*, vol. 19, no. 13, 2010, pp. 2739–2745. <https://doi.org/10.1093/hmg/ddq155>.
- [10] Wang, T. J., et al. "Common Genetic Determinants of Vitamin D Insufficiency: A Genome-Wide Association Study." *Lancet*, vol. 376, no. 9736, 2010, pp. 180–188. [https://doi.org/10.1016/S0140-6736\(10\)60588-0](https://doi.org/10.1016/S0140-6736(10)60588-0).
- [11] Chambers, J. C., et al. "Genome-Wide Association Study Identifies Variants in TMPRSS6 Associated with Hemoglobin Levels." *Nature Genetics*, vol. 41, no. 11, 2009, pp. 1170–1172. <https://doi.org/10.1038/ng.462>.
- [12] Nai, A., et al. "TMPRSS6 rs855791 Modulates Hpcidin Transcription In Vitro and Serum Hpcidin Levels in Normal Individuals." *Blood*, vol. 118, no. 16, 2011, pp. 4459–4462. <https://doi.org/10.1182/blood-2011-06-364034>.
- [13] Buerkli, S., et al. "The TMPRSS6 Variant (SNP rs855791) Affects Iron Metabolism and Oral Iron Absorption – A Stable Iron Isotope Study in Taiwanese Women." *Haematologica*, vol. 106, no. 11, 2021, pp. 2897–2905. <https://doi.org/10.3324/haematol.2020.264556>.

- [14] Fabozzi, Gemma, et al. "Personalized Nutrition in the Management of Female Infertility: New Insights on Chronic Low-Grade Inflammation." *Nutrients*, vol. 14, no. 9, 2022, article 1918. <https://doi.org/10.3390/nu14091918>.
- [15] Madeo, G., et al. "Nutrigenomics: SNPs Correlated to Lipid and Carbohydrate Metabolism." *La Clinica Terapeutica*, vol. 174, suppl. 2, no. 6, 2023, pp. 200–208. <https://doi.org/10.7417/CT.2023.2488>.
- [16] de Luis, Daniel Antonio, et al. "Role of the Variant in Adiponectin Gene rs266729 on Weight Loss and Cardiovascular Risk Factors after a Hypocaloric Diet with the Mediterranean Pattern." *Nutrition*, vol. 60, 2019, pp. 1–5. <https://doi.org/10.1016/j.nut.2018.08.018>.
- [17] Song, Y., et al. "The rs7799039 Variant in the Leptin Gene Promoter Drives Insulin Resistance through Reduced Serum Leptin Levels." *Frontiers in Endocrinology*, vol. 16, 2025, article 1589575. <https://doi.org/10.3389/fendo.2025.1589575>.
- [18] de Luis, Daniel A., et al. "Effect of -55CT Polymorphism of UCP3 on Insulin Resistance and Cardiovascular Risk Factors after a High Protein/Low Carbohydrate versus a Standard Hypocaloric Diet." *Annals of Nutrition & Metabolism*, vol. 68, no. 3, 2016, pp. 157–163. <https://doi.org/10.1159/000444150>.
- [19] de Luis, D., et al. "Effect of the PPARG rs1801282 Polymorphism on Weight Reduction and Metabolic Syndrome Outcomes in Obese Individuals Undergoing a Partial Meal Replacement Hypocaloric Diet." *Journal of Diabetes and Its Complications*, vol. 40, no. 1, 2026, article 109209. <https://doi.org/10.1016/j.jdiacomp.2025.109209>.
- [20] Deeb, S. S., et al. "A Pro12Ala Substitution in PPAR γ 2 Associated with Decreased Receptor Activity, Lower Body Mass Index and Improved Insulin Sensitivity." *Nature Genetics*, vol. 20, no. 3, 1998, pp. 284–287. <https://doi.org/10.1038/30999>.
- [21] Ek, J., et al. "Studies of the Pro12Ala Polymorphism of the Peroxisome Proliferator-Activated Receptor- γ 2 (PPAR- γ 2) Gene in Relation to Insulin Sensitivity among Glucose Tolerant Caucasians." *Diabetologia*, vol. 44, no. 9, 2001, pp. 1170–1176. <https://doi.org/10.1007/s001250100629>.
- [22] Zhang, Xiaomin, et al. "FTO Genotype and 2-Year Change in Body Composition and Fat Distribution in Response to Weight-Loss Diets: The POUNDS LOST Trial." *Diabetes*, vol. 61, no. 11, 2012, pp. 3005–3011. <https://doi.org/10.2337/db11-1799>.
- [23] Basiri, Marjan Ghane, et al. "APOA2 -256T>C Polymorphism Interacts with Saturated Fatty Acids Intake to Affect Anthropometric and Hormonal Variables in Type 2 Diabetic Patients." *Genes & Nutrition*, vol. 10, 2015, article 15. <https://doi.org/10.1007/s12263-015-0464-4>.
- [24] Corella, D., et al. "APOA2, Dietary Fat, and Body Mass Index: Replication of a Gene-Diet Interaction in 3 Independent Populations." *Archives of Internal Medicine*, vol. 169, no. 20, 2009, pp. 1897–1906. <https://doi.org/10.1001/archinternmed.2009.343>.
- [25] Corella, D., et al. "Association between the APOA2 Promoter Polymorphism and Body Weight in Mediterranean and Asian Populations: Replication of a Gene-Saturated Fat Interaction." *International Journal of Obesity*, vol. 35, no. 5, 2011, pp. 666–675. <https://doi.org/10.1038/ijo.2010.187>.
- [26] Jiang, Chao Qiang, et al. "A Single Nucleotide Polymorphism in APOA5 Determines Triglyceride Levels in Hong Kong and Guangzhou Chinese." *European Journal of Human Genetics*, vol. 18, no. 11, 2010, pp. 1255–1260. <https://doi.org/10.1038/ejhg.2010.93>.
- [27] Triglyceride Coronary Disease Genetics Consortium and Emerging Risk Factors Collaboration, et al. "Triglyceride-Mediated Pathways and Coronary Disease: Collaborative Analysis of 101 Studies." *Lancet*, vol. 375, no. 9726, 2010, pp. 1634–1639. [https://doi.org/10.1016/S0140-6736\(10\)60545-4](https://doi.org/10.1016/S0140-6736(10)60545-4).

- [28] Xu, C., et al. "Effects of APOA5 -1131T>C (rs662799) on Fasting Plasma Lipids and Risk of Metabolic Syndrome: Evidence from a Case-Control Study in China and a Meta-Analysis." PLoS One, vol. 8, no. 2, 2013, article e56216. <https://doi.org/10.1371/journal.pone.0056216>.
- [29] Stocks, Tanja, et al. "TFAP2B – Dietary Protein and Glycemic Index Interactions and Weight Maintenance after Weight Loss in the DiOGenes Trial." Human Heredity, vol. 75, nos. 2–4, 2013, pp. 213–219. <https://doi.org/10.1159/000353591>.
- [30] Guo, Y., et al. "Gene-Centric Meta-Analyses of 108,912 Individuals Confirm Known Body Mass Index Loci and Reveal Three Novel Signals." Human Molecular Genetics, vol. 22, no. 1, 2013, pp. 184–201. <https://doi.org/10.1093/hmg/dds396>.
- [31] Ramos-Lopez, O., et al. "Association of the Gly482Ser PPARGC1A Gene Variant with Different Cholesterol Outcomes in Response to Two Energy-Restricted Diets in Subjects with Excessive Weight." Nutrition, vol. 47, 2018, pp. 83–89. <https://doi.org/10.1016/j.nut.2017.10.008>.
- [32] Goyenechea, E., et al. "Enhanced Short-Term Improvement of Insulin Response to a Low-Caloric Diet in Obese Carriers the Gly482Ser Variant of the PGC-1alpha Gene." Diabetes Research and Clinical Practice, vol. 82, no. 2, 2008, pp. 190–196. <https://doi.org/10.1016/j.diabres.2008.08.011>.
- [33] Mager, U., et al. "Variations in the Ghrelin Receptor Gene Associate with Obesity and Glucose Metabolism in Individuals with Impaired Glucose Tolerance." PLoS One, vol. 3, no. 8, 2008, article e2941. <https://doi.org/10.1371/journal.pone.0002941>.
- [34] de Luis, D., et al. "Role of rs490683 Variant in the Promoter Region of the Ghrelin Receptor Gene on Body Weight and Metabolic Syndrome after a Partial Meal Replacement Hypocaloric Diet." Journal of Diabetes and Its Complications, vol. 39, no. 8, 2025, article 109062. <https://doi.org/10.1016/j.jdiacomp.2025.109062>.
- [35] Warodomwicht, D., et al. "ADIPOQ Polymorphisms, Monounsaturated Fatty Acids, and Obesity Risk: The GOLDN Study." Obesity, vol. 17, no. 3, 2009, pp. 510–517. <https://doi.org/10.1038/oby.2008.583>.
- [36] de Luis Roman, D., et al. "The Polymorphism rs17300539 in the Adiponectin Promoter Gene Is Related to Metabolic Syndrome, Insulin Resistance, and Adiponectin Levels in Caucasian Patients with Obesity." Nutrients, vol. 15, no. 24, 2023, article 5028. <https://doi.org/10.3390/nu15245028>.
- [37] Corbi, G., et al. "Adiponectin Expression and Genotypes in Italian People with Severe Obesity Undergone a Hypocaloric Diet and Physical Exercise Program." Nutrients, vol. 11, no. 9, 2019, article 2195. <https://doi.org/10.3390/nu11092195>.
- [38] de Luis, D. A., et al. "Serum Lipid and Adiponectin Improvements after a Mediterranean Dietary Pattern in Non-G-Allele Carriers of the Variant rs3774261." Lifestyle Genomics, vol. 13, no. 6, 2020, pp. 164–171. <https://doi.org/10.1159/000508819>.
- [39] de Luis Roman, D. A., et al. "Adiponectin Gene Variant rs3774261, Effects on Lipid Profile and Adiponectin Levels after a High Polyunsaturated Fat Hypocaloric Diet with Mediterranean Pattern." Nutrients, vol. 13, no. 6, 2021, article 1811. <https://doi.org/10.3390/nu13061811>.
- [40] Speliotes, E. K., et al. "Association Analyses of 249,796 Individuals Reveal 18 New Loci Associated with Body Mass Index." Nature Genetics, vol. 42, no. 11, 2010, pp. 937–948. <https://doi.org/10.1038/ng.686>.
- [41] Rask-Andersen, M., et al. "The MAP2K5-Linked SNP rs2241423 Is Associated with BMI and Obesity in Two Cohorts of Swedish and Greek Children." BMC Medical Genetics, vol. 13, 2012, article 36. <https://doi.org/10.1186/1471-2350-13-36>.

- [42] Abadi, Arkan, et al. "Penetrance of Polygenic Obesity Susceptibility Loci across the Body Mass Index Distribution." *American Journal of Human Genetics*, vol. 101, no. 6, 2017, pp. 925–938. <https://doi.org/10.1016/j.ajhg.2017.10.007>.
- [43] Haupt, A., et al. "Impact of Variation in the FTO Gene on Whole Body Fat Distribution, Ectopic Fat, and Weight Loss." *Obesity*, vol. 16, no. 8, 2008, pp. 1969–1972. <https://doi.org/10.1038/oby.2008.283>.
- [44] Peng, S., et al. "FTO Gene Polymorphisms and Obesity Risk: A Meta-Analysis." *BMC Medicine*, vol. 9, 2011, article 71. <https://doi.org/10.1186/1741-7015-9-71>.
- [45] Saldaña-Alvarez, Yolanda, et al. "Gender-Dependent Association of FTO Polymorphisms with Body Mass Index in Mexicans." *PLoS One*, vol. 11, no. 1, 2016, article e0145984. <https://doi.org/10.1371/journal.pone.0145984>.
- [46] Liaw, Y. C., Y. P. Liaw, and T. H. Lan. "Physical Activity Might Reduce the Adverse Impacts of the FTO Gene Variant rs3751812 on the Body Mass Index of Adults in Taiwan." *Genes*, vol. 10, no. 5, 2019, article 354. <https://doi.org/10.3390/genes10050354>.
- [47] da Fonseca, Ana Carolina Proença, et al. "Adiponectin, Retinoic Acid Receptor Responder 2, and Peroxisome Proliferator-Activated Receptor- γ Coactivator-1 Genes and the Risk for Obesity." *Disease Markers*, 2017, article 5289120. <https://doi.org/10.1155/2017/5289120>.
- [48] Paternoster, L., et al. "Genome-Wide Population-Based Association Study of Extremely Overweight Young Adults – The GOYA Study." *PLoS One*, vol. 6, no. 9, 2011, article e24303. <https://doi.org/10.1371/journal.pone.0024303>.
- [49] Pei, Y. F., et al. "Meta-Analysis of Genome-Wide Association Data Identifies Novel Susceptibility Loci for Obesity." *Human Molecular Genetics*, vol. 23, no. 3, 2014, pp. 820–830. <https://doi.org/10.1093/hmg/ddt464>.
- [50] Peters, U., et al. "A Systematic Mapping Approach of 16q12.2/FTO and BMI in More than 20,000 African Americans Narrows in on the Underlying Functional Variation: Results from the Population Architecture Using Genomics and Epidemiology (PAGE) Study." *PLoS Genetics*, vol. 9, no. 1, 2013, article e1003171. <https://doi.org/10.1371/journal.pgen.1003171>.
- [51] Hubáček, J. A., et al. "The FTO Gene and Obesity in a Large Eastern European Population Sample: The HAPIEE Study." *Obesity*, vol. 16, no. 12, 2008, pp. 2764–2766. <https://doi.org/10.1038/oby.2008.421>.
- [52] Cha, S. W., et al. "Replication of Genetic Effects of FTO Polymorphisms on BMI in a Korean Population." *Obesity*, vol. 16, no. 9, 2008, pp. 2187–2189. <https://doi.org/10.1038/oby.2008.314>.
- [53] Zhang, Xianpeng, et al. "The Association between Sarcopenia Susceptibility and Polymorphisms of FTO, ACVR2B, and IRS1 in Tibetans." *Molecular Genetics & Genomic Medicine*, vol. 9, no. 8, 2021, article e1747. <https://doi.org/10.1002/mggg.3.1747>.
- [54] Karasik, David, et al. "Disentangling the Genetics of Lean Mass." *American Journal of Clinical Nutrition*, vol. 109, no. 2, 2019, pp. 276–287. <https://doi.org/10.1093/ajcn/nqy272>.
- [55] Yang, J., et al. "FTO Genotype Is Associated with Phenotypic Variability of Body Mass Index." *Nature*, vol. 490, no. 7419, 2012, pp. 267–272. <https://doi.org/10.1038/nature11401>.
- [56] Scuteri, A., et al. "Genome-Wide Association Scan Shows Genetic Variants in the FTO Gene Are Associated with Obesity-Related Traits." *PLoS Genetics*, vol. 3, no. 7, 2007, article e115. <https://doi.org/10.1371/journal.pgen.0030115>.
- [57] Okada, Y., et al. "Common Variants at CDKAL1 and KLF9 Are Associated with Body Mass Index in East Asian Populations." *Nature Genetics*, vol. 44, no. 3, 2012, pp. 302–306. <https://doi.org/10.1038/ng.1086>.

- [58] Akiyama, K., et al. "Systematic Fine-Mapping of Association with BMI and Type 2 Diabetes at the FTO Locus by Integrating Results from Multiple Ethnic Groups." PLoS One, vol. 9, no. 6, 2014, article e101329. <https://doi.org/10.1371/journal.pone.0101329>.
- [59] Hotta, Kikuko, et al. "Variations in the FTO Gene Are Associated with Severe Obesity in the Japanese." Journal of Human Genetics, vol. 53, no. 6, 2008, pp. 546–553. <https://doi.org/10.1007/s10038-008-0283-1>.
- [60] Vimalaswaran, K. S., et al. "Physical Activity Attenuates the Body Mass Index-Increasing Influence of Genetic Variation in the FTO Gene." American Journal of Clinical Nutrition, vol. 90, no. 2, 2009, pp. 425–428. <https://doi.org/10.3945/ajcn.2009.27652>.
- [61] Heianza, Yoriko, et al. "Starch Digestion-Related Amylase Genetic Variant Affects 2-Year Changes in Adiposity in Response to Weight-Loss Diets: The POUNDS Lost Trial." Diabetes, vol. 66, no. 9, 2017, pp. 2416–2423. <https://doi.org/10.2337/db16-1482>.

Disclaimers

This DNA Wellness Report is intended only for general wellness, educational, and informational purposes. It provides information about selected genetic variants that may be associated with non-diagnostic wellness traits, such as general nutrition, fitness, lifestyle, and other wellness-related characteristics, based on scientific literature and internal interpretation methods available at the time the report is prepared.

This test and report have not been reviewed, cleared, approved, or authorized by the U.S. Food and Drug Administration, unless expressly stated otherwise in writing. This report is not intended to diagnose, treat, cure, mitigate, or prevent any disease or medical condition, and it should not be used as a substitute for professional medical advice, diagnosis, treatment, screening, or care.

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The test analyzes selected genetic variants only. It does not analyze all variants in any gene, does not evaluate the entire genome, and may not detect genetic factors that are relevant to a trait or condition. Wellness traits are influenced by many factors, including genetics, diet, physical activity, sleep, environment, age, sex, health history, and other personal circumstances. A genetic association does not guarantee a particular trait, outcome, benefit, or response.

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