A GENETIC LIFEHACKS GUIDE:

SATURATED FATS & YOUR GENES

Wondering if eating too much saturated fat will impact your health? The answer may be in your genes!

BY DEBORAH MOON

Disclaimer: All information provided in this guide is for educational purposes only and not to be considered medical advice.

www.geneticlifehacks.com

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The science of saturated fats and your genes

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INTRODUCTION

Welcome to the wonderful world of genetics, nutrition, and YOU! This workbook is designed so that you can learn more about how your genes work and find ways to optimize your diet and lifestyle to fit your genes.

Here you will find information about genetics that is based **entirely on research**. The information is **fully referenced** and presented in easy to understand language, selected to fit consumer genetics tests, and consolidated to fit this specific topic of interest.

The workbook format is designed for you to fill in your own genetic information, as well as your family's information if you have it.

Why a workbook for the whole family? Seeing everyone's information side by side is a fascinating glimpse into how traits are passed down from parent to child. It also highlights how even within a family you can all have unique nutritional needs.

Plus it is just handier to have all your genetic information in one spot and a lot less expensive to buy one workbook for the whole crew. (Yep, I inherited the frugal gene!)

I hope you all enjoy this deep dive into the science of genetics and diet!

Deborah Moon

My genotype:
A A

Spouse's genotype:
AG

Son #1's genotype:
A A

Son #2's genotype:
A G

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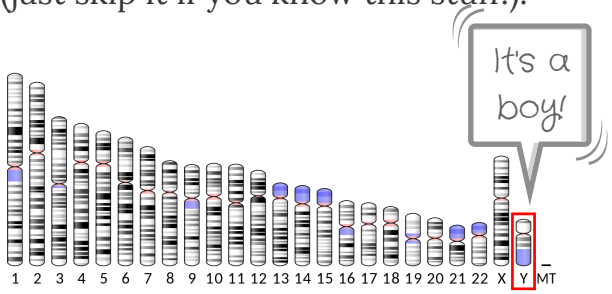
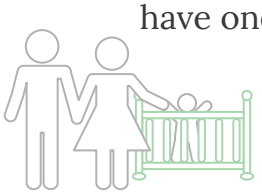
GENETICS PRIMER:

You spit in a tube and mailed it off to get your genes sequenced. Pretty exciting stuff! But what exactly does the genetic data mean?

Let's start with a quick overview of the basics (just skip it if you know this stuff!):

Humans all have 23 *pairs* of chromosomes. The chromosomes are referred to as #1 - 22, plus the X and Y chromosomes. Females have two copies of the X, while males usually have one X and one Y.

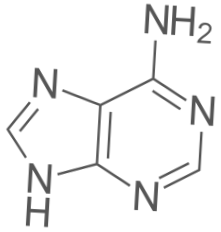




Everyone inherits one set of chromosomes from their mother and the other set from their father.

So what exactly is a chromosome? A chromosome is simply a part of your DNA molecule that is packaged up together. It contains genes -- anywhere from hundreds of genes to thousands of genes, depending on the chromosome.

Going even deeper here, what is DNA? The DNA molecule consists of nucleotides that are bonded to a sugar (called deoxyribose) and phosphate. All together it becomes deoxyribonucleic acid, a.k.a. DNA.



There are four nucleotide bases that make up DNA:
adenine (A), thymine (T), cytosine (C), and guanine (G). This is what you are looking at in your genetic data - the A, C, G, and T's.

Genes	Marker (SNP)	Genomic Position	Variants	Your Genotype
ACE	rs4343	61566031	A or G	A / A

23andMe data example

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Each nucleotide base can only pair together with its complementary base on the other side of the DNA strand, making the familiar double helix.

Complementary DNA pairs:
A pairs with T
G pairs with C

23andMe and AncestryDNA both give the genotype information for just one side of the DNA strand. The side that they use is known as the plus or forward strand. This becomes important to know when reading through research studies because sometimes a study will refer to a position on the minus or reverse orientation, and you will need to translate that using A=T and G=C.

Each of your 23 chromosomes contains millions of nucleotide base pairs for a total of 3 billion base pairs in your whole genome. Both AncestryDNA and 23andMe cover around 600,000 base pairs in their sequencing of your DNA. This is a lot of information, but also only a small portion of your whole genome.

In your genetic raw data file, you will see an rs id number that starts with "rs..." and then a bunch of numbers. The rs id number is a way of referring to a specific position on a chromosome that has been recorded in a database by researchers.

# rsid	chromosome	position	genotype
rs12564807	1	734462	AA
rs3131972	1	752721	GG
rs148828841	1	760998	CC
rs12124819	1	776546	AA
rs115093905	1	787173	GG
rs11240777	1	798959	AG
rs7538305	1	824398	AA
rs4970383	1	838555	AC
rs4475691	1	846808	CT

Example of 23andMe raw data file

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The acronym SNP stands for Single Nucleotide Polymorphism, which means that one nucleotide base has changed at a specific location in a gene. A SNP (said 'snip') is simply a variation that is found in more than 1% of the population, while a mutation is defined as a variation occurring in less than one percent of the population.

There are thought to be about 20,000 genes in the human genome that code for proteins. I say "about" because new research comes out all the time with new information and reclassification of genes. This is another reminder of how new all of this research is on genetics!

USING YOUR GENETIC DATA:

The genetic tests that are 'direct to consumer' such as 23andMe and AncestryDNA are not guaranteed to be 100% accurate. What does this really mean? If you are making decisions on whether to add more beets to your diet, it is probably safe to make that decision based on the genetic information you have. For more serious considerations, such as anything to do with rare genetic diseases or cancer-causing genes, I highly suggest getting a second test done to confirm anything that is clinically significant.

> *What? Why even do the test is it isn't going to tell me everything I ever wanted to know?*

Look at it this way: Whether the information is 96% accurate or 99% accurate... it is still a really good starting point: a direction to go rather than just taking a stab in the dark like you do with most diet and lifestyle suggestions.

Also, keep in mind that genetics is a fairly new field, there could be errors in the research, and there are all kinds of confounding and conflicting factors that can affect your outcomes. Other factors that come into play include your gut microbiome, toxins from the environment, epigenetic modifications from what your mom was exposed to before you were born, and other rare genetic variants that all work together to create a unique individual -- YOU.

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Not medical advice:

Let me clearly state that everything presented in this guide is for informational purposes only and not intended as medical advice.

I know – you read that disclaimer everywhere…
Let me explain a little bit more: Everything in this guide is backed up by research, but it is research on people that aren't YOU. If the suggestions given don't match up with YOU, then ignore them.

A lot of the research studies that are highlighted here are on common dietary situations that won't harm most people. Healthy middle-aged man? Adding in some leafy green veggies to get more folate in your diet is probably a pretty safe bet. Adding in those healthy leafy green veggies when you are taking Warfarin for atrial fibrillation could cause you problems. So if you are under a doctor's care – and especially if you are on a medication – talk with your doctor before making a big change. Take them the information and make an informed decision together.

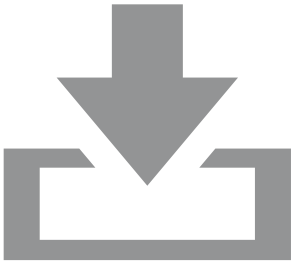
Again, I want to reiterate that most direct to consumer genetic testing is not intended for medical advice nor is it guaranteed by the provider to be completely accurate. It's a good starting point for investigating diet and lifestyle options.

HOW-TO'S FOR 23ANDME.COM DATA:

23andMe (at the time of publication) has a feature on their website that allows you to search through your 'raw data'. Once you have logged in on 23andMe.com, click on your name in the upper right corner and choose Browse Raw Data from the drop-down.

Notice on that page for browsing your raw data that you can also download the file. **Download it!**

The data is yours; keep it in a safe place!



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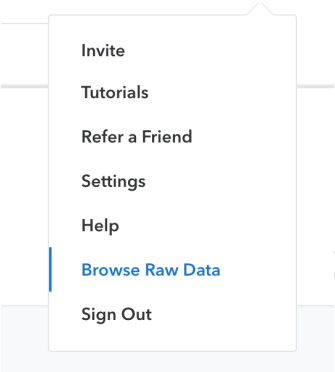
When you come to an rs id number in this workbook to look up:



1. Log into your 23andMe.com account.

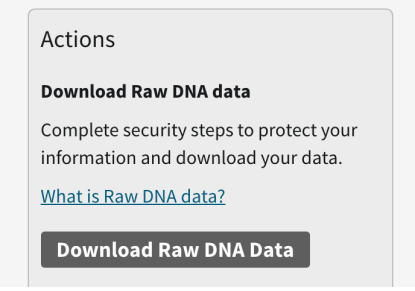
2. Go to your name in the upper right corner and choose "Browse Raw Data".

3. Type in the rs id that you are looking for in the search box.

4. Write down your genotype information in the workbook box.

HOW-TO'S FOR ANCESTRYDNA.COM DATA:

AncestryDNA.com is a little bit more difficult to work with here. Their website doesn't have a feature for looking up your genetic information, so you will need to download the raw data file.



To download your data file, click on Settings on the upper right side of the page. From there, you can click on the button to Download Raw DNA Data. Follow the directions there to complete the download.

The raw data file will be a large text file. Instead of just searching the text file, I suggest importing it into Excel.

In Excel, you can import the file as a .txt file that is tab delimited. Then you will easily be able to search and find your genotype information in the excel file.

rsid	chromosome	position	genotype	allele1	allele2
rs190214723	1	693625	TT	T	T
rs3131972	1	752721	GG	G	G
rs12562034	1	768448	AG	A	G
rs115093905	1	787173	GG	G	G

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SATURATED FAT AND YOUR GENES

There has been a decades-long debate about which type of fat is healthiest and how much fat you should eat. Saturated? Unsaturated? Monounsaturated? Avoid fat at all costs? Eat only fat?

People following a Paleo or ketogenic diet are often quick to tout the benefits of saturated fat. On the other hand, the American Heart Association promotes polyunsaturated fats as being the healthier option.

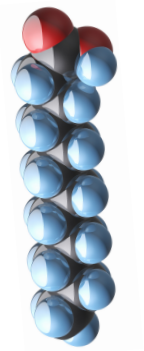
Most people remain just a bit confused about the arguments on either side...

The answer to these questions may be different for you than for your friend who is thriving on a keto diet. Genes matter!

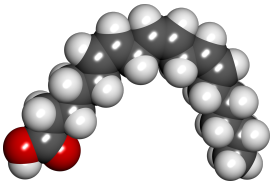
A QUICK OVERVIEW OF FATS...

It all comes back to chemistry and chemical bonds.

Fats are made up of chains of fatty acids and glycerol, and they can be classified based on how the bonds between the fatty acids combine. They can form either double bonds between the carbons or single bonds. Saturated fats are completely 'saturated' with hydrogen and have no double bonds. Unsaturated fats, on the other hand, have one or more double bonds, and can be further classified as polyunsaturated or monounsaturated fats.



Saturated Fat
(Lauric acid)



Unsaturated Fat
(Gamma linolenic acid)

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Saturated fats generally have higher melting points, thus making them more likely to be solid at room temperature. Unsaturated fats are usually liquids at room temperature. Examples of foods that contain saturated fats include cheese, butter, fatty meats, coconut, egg yolks, lard, and tallow. Examples of unsaturated fats would include corn oil, canola oil, soybean oil, and fish oils.

When looking at saturated fat, two big things commonly come to mind:

1. CARDIOVASCULAR DISEASE
2. WEIGHT GAIN & OBESITY

Genetics can play a role in how saturated fat interacts with both of these conditions. For example, some people may be at a higher risk of cardiovascular disease due to saturated fat intake, while for others it may have little or no effect. Likewise, saturated fat intake can play a role in obesity – depending on the genetic variants that you carry.

Wondering how much saturated fat you normally eat? You can track your dietary consumption using an online tool such as Cron-o-meter or My Fitness Pal. You can also look up the nutrition data for most foods at nutritiondata.self.com.

Several of the saturated fat studies define a high saturated fat intake as being over 22g of saturated fat per day.

Here is the saturated fat content of some common foods to give you an idea of how saturated fat content adds up over a day (from nutritiondata.self.com).

T-bone steak (9 oz, broiled):	20.3g
McDonald's single hamburger:	3.1g
Bacon (1 slice, pan-fried):	1.0g
McDonald's BEC Biscuit:	11.7g
Chick-fil-A Chicken Sandwich:	3.5g
Egg (cooked, scrambled):	8.1g

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PART 1. CARDIOVASCULAR DISEASE

Saturated fat and heart disease...
the big questions that come to mind are:

Will eating bacon dipped in coconut oil on a ketogenic diet give me a heart attack?
Is it OK to drown my veggies in a lot of butter?

Realistically, no one can fully answer these questions based just on looking at a couple of genetic variants. But - and this is a kind of big but - if you carry the genetic variant listed below, you may want to pay more attention to the amount of saturated fat you are eating and keep an eye on your heart health through tracking blood pressure, testing blood lipids, and getting a regular check-up.

What do recent studies show about saturated fat and heart disease for the overall population? A 2016 review looked into the topic extensively. It concluded that replacing saturated fat with low-quality carbs, especially sugar, may worsen cardiovascular disease risk, but replacing saturated fat with polyunsaturated fat (specifically from eating fish, vegetables, and nuts) may reduce the risk of cardiovascular disease. Overall, saturated fat from dairy products was considered to be either neutral or beneficial for cardiovascular disease. (2)

The big take away from several of the more recent studies is that saturated fat, especially full-fat dairy, may not be a risk factor for cardiovascular disease for the *general population*. It also doesn't seem to affect insulin sensitivity in a healthy population. (3) But we can do better than just general population studies by looking into specific genetic interactions with dietary fats.

Let's dive into the genes!

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ANGIOTENSIN-CONVERTING ENZYME (ACE) GENE

The angiotensin-converting enzyme (ACE) helps the body maintain blood pressure at a normal level. It works within the body's blood pressure regulation system converting angiotensin I into angiotensin II.

Like most of our body's systems, balance is key. The right amount of angiotensin II is important -- too much will cause blood vessels to constrict resulting in a subsequent increase in blood pressure. A frequently prescribed type of blood pressure medication is an ACE inhibitor, which decreases the amount of angiotensin II.

A common variant of the ACE gene, rs4343, is known as the ACE deletion. It changes the amount of the ACE enzyme that your body normally makes.
A little terminology here:

- Some people have two copies of the ACE insertion variant (ACE I/I).
- Some have one copy of the insertion and one copy of the deletion (ACE I/D)
- Approx. 20% are homozygous for the ACE deletion variant (ACE D/D).

rs4343	Studies show:
AA	**ACE I/I** **normal response to saturated fat**
AG	**ACE D/I** **normal response to saturated fat**
GG	**ACE D/D** **high saturated fat diet may increase blood pressure and risk of heart disease** **a long-term high-fat diet may also impair glucose tolerance.**

My genotype:

_____'s genotype:

_____'s genotype:

_____'s genotype:

STUDIES ON THE ACE GENE:

A 2017 study looked at the interaction between copies of the ACE deletion variant and saturated fat. It found that for those with two copies the ACE deletion, a diet higher in saturated fat was significantly associated with increased blood pressure and heart disease. In those who did not carry two copies of the deletion variant, saturated fat consumption had no effect on the risk of heart disease.

Another 2017 study looked at a group of 96 adults who first were on a low-fat diet for 6 weeks and then on a high saturated fat diet for 6 weeks. The high-fat diet contained more than 45% fat with an emphasis on red meat, sausage, bacon, and full-fat dairy. During the initial six-week low-fat diet there was no significant difference in blood pressure by ACE genotype. After 6 weeks on the high-fat diet, though, those with the ACE D/D genotype had about a 10 point increase in systolic BP compared to the ACE I/D or ACE I/I genotypes. Things to note... The study participants were healthy, normal weight people with an average age of 31 and average BMI of 22.8. The high saturated fat diet caused an increase in cholesterol (both HDL and LDL) in everyone, regardless of ACE genotype. (4)

Another published study found that normal-weight people for six weeks. They found that when the subjects consumed a diet higher in fat than their baseline diet they had impaired glucose tolerance and double the risk of diabetes. To confirm their results, the researchers also looked at data from other study groups and found that when grouped by genotype and fat intake, those with the ACE D/D genotype whose diet was more than 37% fat had an over a four-fold risk of diabetes compared with those on a lower fat diet. (5)

LIFEHACK SUGGESTIONS (ACE GENE)

Common sense ideas:
If you have high blood pressure and the ACE D/D genotype, cutting down on saturated fat might be a way to control your blood pressure with diet. If you are under a doctor's care, you should talk to your doctor before making dietary changes, especially if you are on blood pressure medication.

If you are healthy and have normal blood pressure, the ACE deletion genotype isn't an automatic reason to avoid trying a ketogenic diet or a Paleo diet that is higher in saturated fat. It is a real reason, though, to keep track of your blood pressure while on any diet high in saturated fat.

Do keep in mind the study showing impaired blood glucose tolerance in those with the ACE D/D genotype eating a diet high in saturated fat. If you are struggling with pre-diabetes or diabetes, lowering saturated fat might help your blood glucose levels. Again, I suggest that you keep track of your blood glucose level to see how diet affects it. Test your blood glucose levels with an inexpensive meter to see if this holds true for you. And do talk with your doctor if you are under a doctor's care for diabetes.

These herbs have been shown to be natural ACE inhibitors:

- Hibiscus tea is a folk remedy used for lowering blood pressure. A study found that the way that hibiscus tea lowers blood pressure is through inhibiting the ACE enzyme activity. (6)
- Barberry (berberine is the active component) has also been found to naturally inhibit ACE activity. (7)
- Mucuna pruriens, also known as velvet bean, is another plant that has been shown to be a natural ACE inhibitor. (8)

2. WEIGHT GAIN AND SATURATED FAT

Will eating saturated fat make me fat?

The answer to that question may depend both on your genes and on what you are eating alongside your saturated fat. I don't think it matters too much if your donut is fried in lard or corn oil -- both are going to be fattening!

There are many mouse studies showing that diets high in saturated fats can make mice fat. (9) There are even studies in rats showing that saturated fat may be worse for rats than soybean oil. (10)

But what do human studies show?
One recent study investigated this question with overweight, healthy young adults. The study participants switched a big part of their normal fat intake to saturated fat. The results showed an increase in cholesterol but a slight decrease (statistically significant) in weight after two weeks. (11) On the other hand, a recent meta-analysis research article recommends switching out saturated fats for polyunsaturated fats. (12)

Once again, the answer to the question is that we are all unique and we have individual responses to what we eat. For some people, saturated fat consumption may lead to a greater risk of obesity, while for other people it may not be obesigenic. (As part of a healthy diet -- I'm still not talking about donuts here...)

There are four genes that have been shown to affect the risk of obesity due to saturated fat in the diet: APOA2, PPARG, FTO, and ADIPOQ.

Let's dive into the genes!

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APOA2 GENE

The protein produced by APOA2 is a type of high-density lipoprotein (HDL cholesterol). Different variation in this gene can result in either APOA2 deficiency or hypercholesteremia (too much HDL cholesterol).

One well-studied variant, rs5082, decreases the level of APOA2. It has been linked in research to increased BMI, visceral fat, and HDL levels along with a lower risk of heart disease. Yes - even though it is linked to an increase in body fat and higher BMI, the variant is shown to also be protective against heart disease.

The rs5082 genetic variant has been studied in association with obesity for a couple of decades, but not all of the early studies showed that the variant caused an increased risk of obesity. More recent studies that segregated participants by both genetics and the amount of saturated fat in the diet consistently show that the variant is a risk factor for obesity when combined with a high saturated fat diet.

rs5082	Studies show:
AA	**normal risk of obesity w/ saturated fat (most common genotype)**
AG	**normal risk of obesity w/ saturated fat**
GG	**increased risk of obesity, especially with a diet high in saturated fat**

My genotype:

_____'s genotype:

_____'s genotype:

_____'s genotype:

STUDIES ON THE APOA2 GENE:

Using a compilation of data from three different ethnic groups, researchers determined that high saturated fat intake combined with the rs5082 GG genotype to increase the risk of obesity by 82%. The study found an average 6% increase in BMI for those with a saturated fat intake >22 grams per day. The researchers theorize that APOA2 is acting as a satiety signal, so having less APOA2 would mean that a person wouldn't get the message that they had already eaten enough when eating a meal high in saturated fat. (13)

A study of 303 teenagers in Egypt found that those with the rs5082 GG genotype were at a higher risk for obesity and had higher HDL cholesterol than those with AA or AG genotypes. The study also noted that, on average, those with the GG genotype ate more calories and had a greater intake of saturated fat than those with the AA or AG genotypes. (14) These results echo the theory that people with the GG genotype are not receiving the same satiety signal from saturated fat.

Researchers in Iran looked at the APOA2 gene and diet of 737 type-2 diabetes patients. The results showed that average BMI was higher in people with the rs5082 GG genotype who ate a higher saturated fat diet (>22g/day). The study also found that average ghrelin levels were higher in GG carriers. (15) Ghrelin is known as the 'hunger hormone', and it is theorized that the impaired satiety signaling from the saturated fat may play a role in higher food intake.

Another study looked at the impact of eating full-fat dairy products vs. low-fat dairy products. The researchers again found a connection between eating more saturated fat, carrying the rs5082 GG genotype, and having an increased BMI. (16)

LIFEHACK SUGGESTIONS (APOA2 GENE):

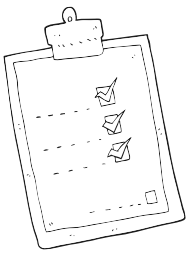
Common sense ideas:

If you carry the rs5082 GG genotype and are worried about gaining weight, cutting down on saturated fat may help prevent weight gain. (OK, this isn't earth-shattering genius-ry here, but I just had to include it!)

Knowing that you are less likely to get the signal that you are satiated from foods high in saturated fat, portion control can become important.
Here's a scenario for you: Instead of sitting down with a spoon and a pint of Ben and Jerry's, take a look at the label and notice that there are four servings in that container. Knowing that you aren't going to get the same satiety signal that other people do, get out a small bowl and measure out a serving size. Then combine your Ben and Jerry's treat with something that you find more filling and has fewer calories.

One study found that people with the rs5082 GG genotype were more likely to skip meals and less likely to plan meals in advance. These habits, along with an increase in ghrelin, the hunger hormone, can lead to poor eating habits and unhealthy meal choices. Planning out meals in advance may be a good way to avoid grabbing the chips and dip when hunger strikes.

Knowledge is power here! If you don't feel full after eating foods high in saturated fat, combine smaller amounts of those foods with something that is more satiating for YOU.



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PPARG GENE

The PPARG (peroxisome proliferator-activated receptor gamma) gene has been associated in multiple studies with obesity, metabolic syndrome, and risk for type-2 diabetes. PPARG is a cellular receptor that plays several different roles in the body including being involved in the regulation of fatty acid storage and in glucose metabolism. It is activated by omega-6 polyunsaturated fatty acids and regulates the creation of new fat storage cells. In addition to regulating the genes involved in storing fat, PPARG also aids in regulating the circadian rhythm of insulin sensitivity over a 24-hour cycle. (18)

PPARG could be thought of as a gateway that activates a fat cell to store more fatty acids, thus taking the fatty acids out of circulation. This then causes cells in the body to take up glucose (mainly from carbohydrates) and use it for energy.

The most commonly studied variant, rs1801282 or Pro12Ala, is thought to naturally decrease PPARG activity, protecting against weight gain for some people -- depending on the amount of omega-6 fat in the diet.

rs1801282	Studies show:	My genotype:
CC	**normal risk of obesity with saturated fat (most common genotype)**	_____'s genotype:
CG	**increased risk of obesity with more saturated fat in the diet**	_____'s genotype:
GG	**increased risk of obesity with more saturated fat in the diet**	_____'s genotype:

STUDIES ON THE PPARG GENE:

A study found that those carrying the rs1801282 G allele were more likely to be obese when eating a diet higher in saturated fat; people with the G allele were not at a higher risk for obesity if their fat intake was based more on polyunsaturated fats. (20) This makes sense when you think about it. In general, people eating a diet higher in saturated fats are usually eating less unsaturated fat. In those with the PPARG variant, the body isn't getting the normal signal from polyunsaturated fatty acids.

A nine-year study on a French population (4,676 people) that looked at PPARG rs1801282 found that those who had two copies of the variant had an average BMI that was 2.2 points higher, but only among those with higher fat consumption. (22) One thing to note here is that French cuisine is known for being higher in saturated fat.

A mouse study supports some of the human studies and explains the differences seen in people who eat different diets. The mice, bred to carry the rs1801282 variant, were a little bit leaner, had better glucose tolerance, and lived a little longer -- when they were fed normal chow. They also were a little more active in the evening, thus possibly expending a little more energy while eating the same amount of food. But when those same mice with the PPARG variant were fed high-fat chow, they became fatter than the mice without the variant. (21)

LIFEHACK SUGGESTIONS (PPARG GENE):

Diet:

For those with the PPARG rs1801282 G allele variant, sticking to a diet lower in saturated fat may be protective against weight gain. This may be more important in those eating a normal diet with a mixture of fats, carbs, and protein, rather than for someone on a ketogenic diet that is high in all types of fats.

A study in Brazil looked at the effects of adding olive oil to the traditional Brazillian diet. It found that adding about 2 tsp of olive oil to each meal helped significantly reduce weight -- but only for those with the PPARG GG genotype. (43)

While poly-unsaturated fats in general bind to PPARG and stimulate the uptake of fatty acids into fat cells, one type of medium chain triglycerides may act in a different way. A study showed that decanoic acid (DA), a 10 carbon medium chain triglyceride found in coconut oil, binds to PPARG differently and specifically does not cause an increase in fat cells. This study (in animals) found that DA decreased blood glucose levels without causing weight gain. (24)

Herbals:

A recent study found that citrus bergamot extract could suppress adipogenesis (creation of fat cells) through decreasing PPARG.(25)
Rhodiola rosea, a traditional herbal adaptogen, has been shown to down-regulate PPARG. (27)

Circadian Rhythm

Focusing on the circadian rhythm function of the gene (18), it is important to block blue light in the evening from screens and CFL/LED bulbs. Blocking blue light in the evening allows melatonin levels to rise naturally and regulates your circadian rhythm. There are inexpensive blue light blocking glasses available if you don't want to give up your cell phone or TV in the evenings.

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The science of saturated fats and your gene

FTO GENE

The FTO gene was identified about ten years ago through a genome-wide association study that looked at over 35,000 people to determine genes involved in obesity. But identifying the FTO gene didn't explain why it was so widely linked to higher BMI as well as ADHD, depression, and dementia.

Early studies concluded that those with increased BMI and FTO variants also tended to have an increased energy intake with an association with elevated fat consumption. (29) But what was causing them to want to eat more?

In 2013, a study found that those with variants in the FTO gene express *more* FTO. This is thought to cause higher ghrelin (the 'hunger hormone') levels. (30) This makes sense in conjunction with mouse studies which found that FTO deficient mice suffer from growth retardation and have both reduced lean mass and fat mass. (31)

rs9939609	Studies show:
TT	**normal risk of obesity with saturated fat (most common genotype)**
AT	**increased risk of being overweight or obese, especially with a high saturated fat diet**
AA	**increased risk of being overweight or obese, especially with a high saturated fat diet**

My genotype:

_____'s genotype:

_____'s genotype:

_____'s genotype:

STUDIES ON THE FTO GENE:

Nicknamed the 'fatso' gene due to its early association with obesity and its large effect on obesity risk, further studies over the last decade have found that diet may play a role in how much the FTO genetic variants contribute to a person's weight gain. (32) This actually gives us a bit of hope since most of the early studies on this gene just showed that, across the board, the variant increases the risk of having a higher BMI.

So let's get specific with studies on saturated fat interacting the with FTO variants:

When looking at two different study group populations, scientists found that the average BMI was 1.6 to 2.4 points higher in those with the rs9939609 AA genotype *when they had a high saturated fat intake*. The study specified also that there was no interaction between carbohydrate intake and obesity. (33)

A second study that looked at participants over a 7.5 year period found that carriers of the FTO rs9939609 A allele were overall 65% more likely to be overweight or obese no matter what their energy intake or physical activity levels were. The study also notes that high dietary saturated fat intake (defined here as >15.5%) "further accentuated the risk" of being overweight or obese. (34)

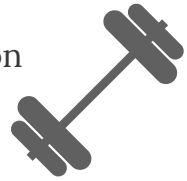
A 2016 study found that teenagers carrying the rs9939609 A allele who ate diets that were lower in fat (less than 30% of the diet) had no increased risk for weight gain. But those who ate diets containing more than 35% fat had up to a 5% increase in total fat mass when compared with teens who did not carry the A allele.

LIFEHACK SUGGESTIONS (FTO GENE):

Diet:
Some studies point to high protein, low-fat diets working better for weight loss for people carrying the FTO risk allele (AA or AT genotype). (35) This may be due either to the reduction of fat or the increase in protein due to FTO playing a role in sensing the level of essential amino acids in the body. (32)

A good dietary plan for mitigating the effect of the FTO variant may be to increase lean protein while reducing your saturated fat. Lean protein options include skinless chicken breast, low-fat cottage cheese, non-fatty fish such as cod or haddock, beans, legumes, lean cuts of beef, and grilled or boiled shrimp.

Physical activity:
Several studies have found that increased physical activity reduces the risk of obesity with the FTO genetic variant. (36) To be fair, though, not all studies show that physical activity decreases the risk of higher BMI with FTO variants. (37) Take, for example, two people – one carrying the FTO risk allele and the other not – the same amount of exercise may not have the same effect on weight. The person carrying the FTO risk allele may need to exercise more than the person without the risk allele to gain the same weight reduction benefits.



Artificial sweeteners:
For men with the obesity causing FTO variant, artificial sweetener consumption was linked to increased weight gain. The study didn't define which artificial sweeteners, just that the more they were consumed, the greater the weight gain. (38)

ADIPOQ GENE

I'm going to shift gears here a little bit with a gene that has specifically to do with the amount of monounsaturated fat in comparison to saturated fat consumption. If you are decreasing saturated fat due to another genetic variant, this may help you with the decision on whether to switch to monounsaturated fats or polyunsaturated fats.

The ADIPOQ gene codes for the creation of adiponectin, a protein produced in fat tissue that causes muscle and skeletal cells to take up glucose. People with metabolic syndrome or obesity tend to have lower adiponectin levels. Low adiponectin levels have been shown to be causal in type-2 diabetes, insulin resistance, and obesity. (39) One commonly used type of diabetes drug, thiazolidinediones, increases the body's adiponectin levels which then causes blood glucose levels to decrease.

Adiponectin levels are estimated to be about 50% heritable, so while diet and lifestyle play a role, genes contribute to about half of your either higher or lower adiponectin levels. (40)

Quick takeaway points:
Higher adiponectin levels are generally considered good, are protective against heart disease and type-2 diabetes.

Lower adiponectin levels are associated with increased BMI, increased risk of type-2 diabetes.

OPTIMIZE YOUR HEALTH

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ADIPOQ GENE

rs182052	Studies show:
GG	**increased adiponectin when replacing saturated fat with monounsaturated**
AG	**decreased adiponectin when replacing saturated fat with monounsaturated**
AA	**decreased adiponectin when replacing saturated fat with monounsaturated**

My genotype:

_____'s genotype:

_____'s genotype:

_____'s genotype:

Summary of studies on the rs182052 variant:

A dietary intervention study with 448 participants showed that the effect of replacing saturated fat with monounsaturated fat differed depending on ADIPOQ genotype. Those who carried the GG genotype (most common) had an increase in adiponectin levels of 3.8% when replacing saturated fat with monounsaturated fat. This would be the expected outcome since several studies had shown a minor increase in adiponectin level with monounsaturated fat. This expectation, though, only held true for carriers of the GG genotype. Those carrying the minor A allele (AG and AA genotype) had a decrease in adiponectin levels by 2.6% when replacing saturated fat with monounsaturated fat.39

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ADIPOQ GENE

rs17300539	Studies show:
GG	**normal adiponectin levels**
AG	**weight may decrease on a high monounsaturated fat diet**
AA	**weight may decrease on a high monounsaturated fat diet**

My genotype:

_____'s genotype:

_____'s genotype:

_____'s genotype:

Summary of studies on the rs17300539 variant:

A study of more than a thousand people looked at how ADIPOQ gene variants and diet influence adiponectin levels. The study found that saturated fat didn't seem to make a big difference in adiponectin levels; however, people who consumed more than 13% monounsaturated fat and carried the GA or AA genotype had a significantly decreased BMI compared to those with the GG genotype. (40)

LIFEHACK SUGGESTIONS (ADIPOQ GENE):

Diet:
Depending on your genetic variants above for ADIPOQ, you may want to add more monounsaturated fat to your diet. Foods high in monounsaturated fats include olive oil, grapeseed oil, walnuts, sunflower seeds, and avocados.

With higher adiponectin levels being tied to a decreased risk of type-2 diabetes and also being protective against heart disease, you may be wondering what increases adiponectin levels....

Blueberries:
A mouse study found that blueberry juice increased adiponectin levels in mice. (41)

Coffee:
A study of 665 Japanese men found that coffee consumption (but not green tea consumption) was associated with adiponectin levels. The more coffee consumed in a day, the higher the adiponectin levels. (42)