

## Is there a link between DNA and Obesity? Rohail A, Year 8

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People become obese for different reasons. Some people might eat a lot of high calorie foods but not become obese and some people might not eat very much but still become obese. Is becoming obese some people's fate? It seems that there is more than what meets the eye. Could this be because of DNA? I will be presenting what I have researched to answer this question: Is there a link between DNA and Obesity?

But first we will have to start with the simple questions:

### What is Obesity?

Obesity describes someone who is very overweight and has a lot of body fat. People use BMI, which means Body Mass Index, to see if you are a healthy weight for your height.

### What is it caused by?

Obesity is generally caused by consuming lots of calories, which are fatty and sugary foods, that you do not burn off through physical exercise, generally causes obesity. This basically means that you eat a

lot more than you exercise. There can be conditions that also support to weight gain. For example: hypothyroidism. This means an underactive thyroid gland. This is basically when your thyroid gland isn't working enough to produce thyroxin which supports the metabolism of your body.

### Risks

Obesity can cause lots of risks, even life threatening conditions such as:

- Diabetes
- Heart disease
- Cancer
- Stroke
- Psychological problems

### Coping with Obesity

There are lots of ways you can cope with obesity:

- Eat a balanced, controlled calorie diet recommended by your GP or dietician
- Join a local weight loss group
- Do activities, jogging, swimming etc. regularly
- Eat slowly and try to avoid to overeat

### What is DNA?

DNA is also known as deoxyribonucleic acid. It is the material passed on by humans and almost all other organisms. Nearly every cell in that person's body has the same DNA.

Most DNA is found in the nucleus cell. The rest are found in the mitochondria.

DNA has information stored as a code. This code is made of four chemical bases: adenine(A), guanine(G), cytosine(C), thymine(T). There are about three million bases in our DNA. The order of these bases determines the information for building an organism.

The DNA bases pair up to make base pairs. Adenine goes with Thymine and Guanine goes with Cytosine. Each base is attached to a sugar phosphate molecule. A base, a sugar molecule and a phosphate

molecule form a nucleotide. These nucleotides are arranged in long strands that form a double helix. The base pairs are the double helix's stairs and the sugar phosphate molecules are the side pieces. DNA has a certain property of making exact copies of itself. This is important for bacteria or healing wounds as the DNA makes copies of itself and then it splits.

### So is there a link?

Scientists are continuing to find links between DNA and Obesity, so they can warn people early of the risks of Obesity and how to plan to cope with it.

Researchers had been investigating genetic samples from about three hundred thousand people and they found more than one hundred and forty locations in their DNA that contribute to Obesity. One very important link between DNA and Obesity is a certain gene called the FTO gene. The FTO gene was the first gene to be identified as having a great influence with weight.

FTO gene stands for Fat mass and Obesity associated gene

All people have this certain gene. This gene is involved in appetite. People with the high risk variant (alternate version) of this gene become more vulnerable to Obesity. The FTO gene also changes the way how the brain responds to food

Study (reference 5) of the FTO gene shows that people with the Obesity-risk FTO variant have higher levels of ghrelin. Ghrelin is a hunger hormone.

The study (reference 5) took two groups of participants, the high obesity-risk FTO variant(AA) and the low Obesity-risk FTO variant(TT).

Usually ghrelin levels would rise before a meal and drop after the meal. This is what happened to the TT group. However, this did not happen to the AA group. The AA group felt hungrier after the meal.

They also studied how the two group's brains reacted when they saw high-calorie foods. The AA group found the high-calorie foods more appealing than to the TT group. With the brain responses, there were major differences in particular parts of the brain. One particular part was the hypothalamus. The hypothalamus is responsible for

hormone production in the brain. The hormones produced by the hypothalamus, for example, are hunger, moods, thirst and sleep.

### Is there more to the FTO gene?

Researchers have also found something else about the FTO gene. Every gene is interrupted by a DNA strand known as an intron. Researchers discovered a “genetic switch” with in the FTO gene’s intron. If a human has the high obesity-risk variant gene, it affects the introns. The introns, when affected by this FTO gene variant, doubles the activity of two other genes. These two genes are called the IRX3 and the IRX5 genes. These two genes determine what kind of fat cells are produced. There are two different types of fat cells, energy burning beige fat cells and energy storing white fat cells.

Normally a protein called ARID5B would stop all of this. But people with the high risk variant of the FTO gene, ARID5B can’t stop the introns from making the IRX genes from doubling up their activity. This then lets the IRX genes create even more energy storing white fat cells.

When scientists managed to turn down the activity of the IRX genes. The energy storing white fat cells turned to energy burning beige fat cells.

### Is there another gene?

According to scientists (reference 3), the FTO gene is not the only gene to manipulate your brain into being attracted to sugary and high-calorie foods.

The other gene responsible for this is called the DRD2 gene. The high risk FTO gene variant depends on the variant of the DRD2 gene.

The FTO gene targets a part of the brain called the striatum. When people look at sugary or high calorie foods, the FTO genes increases the activation of the striatum. But all this depends on the variant of the DRD2. The DRD2 gene alters how the dopamine system works. The dopamine system controls the brain process of certain things, for example our emotional responses and the ability to experience pleasure and pain. Also drugs of abuse can increase dopamine

directly or indirectly because the dopamine is increased in the pleasure and motivation pathways. This probably why people would be addicted to these kind of things.

People with the high risk FTO variant are likely to become obese because the dopamine signals, which the DRD2 gene alters, boost cravings when people see high calorie or sugary foods. So the FTO gene is not the only one to blame for Obesity.

### Conclusion

In conclusion, with all the research found, there is a link between Obesity and DNA. This link is mostly influenced by the FTO gene. Everyone has this gene, but it depends on which variant of the gene you have. There is the normal variant and the high risk obesity variant.

People with the high risk variant are likely to be attracted to high calorie and sugary foods. This is because the FTO gene works with the DRD2 gene, which alters how the dopamine system in the brain works, to send dopamine signals to boost cravings when people see high calorie or sugary foods.

The FTO gene also boosts the levels of hunger hormones know as Ghrelin. This means people feel hungrier after their meal, therefore wanting to eat even more.

We also found something else in the FTO. When people have the high risk variant of the FTO gene, there is a genetic “switch” in the introns in the gene. This then doubles the activity of the IRX genes, turning fat cells from energy burning to energy storing. So there was more to the FTO gene than we thought.

But with all this research, is this link strong or weak?

You could live in the country side with some fast food restaurants but they would be quite far away. You would either have to walk a long way or take a car. If you were walking the you would be doing lots of exercise, burning off the fat cells. So this in turn counters the activity of the IRX genes. If you are taking a car, you would soon get tired of going all the way to a fast food restaurant so you would not bother any way.

In places like cities, you are more likely to become obese. This is because there are lots of fast food places in cities and lots of sugary and high calorie foods. So you would be contributing to your own obesity if you have the high risk variant of the FTO because the IRX genes would already be making twice as much energy storing white fat cells.

So in my opinion, I think there is a strong link depending upon genetic and environmental factors.

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