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Obesity Is a Brain Disease

Analysis by Dr. Joseph Mercola ✔ Fact Checked | January 28, 2023

STORY AT-A-GLANCE

- There's an ongoing effort to categorize obesity as a disease — one that needs to be treated with drugs
- The latest "miracle" drug for obesity — Wegovy — can cause gastrointestinal side effects and has raised concerns of pancreatitis, pancreatic cancer and retinopathy complications, including hemorrhage and blindness
- Obesity is largely related to excess consumption of linoleic acid, an omega-6 polyunsaturated fat, or PUFA
- One way LA increases weight is by stimulating the endocannabinoid receptors; this causes "the munchies," very similar to cannabis
- There's no magic pill for obesity, but drastically reducing LA intake, combined with time-restricted eating, will lead to weight loss in many

In the U.S., 41.9% of adults are considered obese, with prevalence increasing from significantly in recent decades.[1] What's driving this public health epidemic is the burning question, with a wide range of factors, from ultraprocessed foods and linoleic acid to environmental chemicals, likely playing prominent roles.

In a "60 Minutes" episode hosted by Lesley Stahl,[2] however, we see Pharma's ongoing effort to categorize obesity as a disease — one that needs to be treated with drugs. Dr. Fatima Cody Stanford, an obesity doctor at Mass General Hospital and associate

professor at Harvard Medical School, told Stahl obesity is a brain disease. "The brain tells us how much to eat and how much to store."[3]

Is Obesity Due to Your Brain?

Stanford suggests your brain has a certain set weight point that it maintains in your body by controlling food intake and energy storage. It could be a survival mechanism that evolved to help humans hold on to fat during periods of limited food.

"My last patient that I saw … was a young woman who's 39 who struggles with severe obesity. She's been working out five to six times a week, consistently. She's eating very little. Her brain is defending a certain set point,"

Stanford told Stahl.[4] Chronic stressors, she says, may cause your weight point to get reset to a higher number. "Maintain that weight for, let's say, at least three to six months, then you recalibrate that set point to a different set point."[5] Stanford also stated that the No. 1 cause of obesity is genetics.

"That means if you were born to parents that have obesity, you have a 50% to 85% likelihood of having the disease yourself even with optimal diet, exercise, sleep management, stress management."[6] Stanford's credibility is highly questionable, though, as she is an adviser to companies developing drugs for obesity, including Novo Nordisk, maker of Wegovy.[7]

Drugs Being Pushed for Weight Loss

One problem with labeling obesity a brain disease or a genetic disorder is that it opens the floodgates for drug treatment. Even the American Academy of Pediatrics — instead of tackling the drivers of childhood obesity — gave a wholehearted endorsement for weight loss drugs and surgery in children as young as 12 and 13, respectively, in its updated childhood obesity guidance.

"There is no evidence to support either watchful waiting or unnecessary delay of appropriate treatment of children with obesity," the guidance explains, instead setting the tone that early and aggressive drug and surgical treatment is warranted.[8]

The "60 Minutes" interview also sets the tone that drug treatment is the solution, the only problems being a national shortage of certain medications, which Novo Nordisk says has since been remedied.[9] There's also a lack of insurance coverage for some weight loss drugs, including Wegovy, which is given by injection once a week and costs more than $1,300 a month. Stanford said:[10]

> *"If those that have the means are able to get them, yet the people that really need them are unable to, then that creates a greater disparity, right? The haves and the have nots.*
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> *The vast majority of people with obesity simply can't afford Wegovy and most insurance companies refuse to cover it partly because, as AHIP — the health insurance trade association — explained in a statement, these drugs 'have not yet been proven to work well for long-term weight management and can have complications and adverse impacts on patients.'"*

Wegovy, for instance, can cause gastrointestinal side effects, including nausea and vomiting, and has raised concerns of pancreatitis, pancreatic cancer and retinopathy complications, including hemorrhage and blindness.[11] This does not include the law of unintended consequences in using a drug that in no way, shape or form addresses the cause of the disease, and will inevitably cause other complications that were not identified in the trials used to approve the drug.

There Is No Magic Pill for Obesity, but Rimonabant Came Close

Promoting weight loss drugs as the solution for obesity is criminally misleading and dangerous, as there is no magic pill for obesity. The closest I have seen to an effective drug for obesity, however, is Rimonabant,[12] which blocks endocannabinoid receptors in the brain, and 15 years ago was being promoted as the new "miracle" drug for obesity.

It works, but causes an increase in suicidal depression, so it was removed from the market — illustrating the law of unintended consequences in every drug I have ever seen for obesity. Still, there's a lot to be learned about why Rimonabant worked for weight loss. In my interview with Tucker Goodrich on the topic of **linoleic acid** (LA), he explained:

> *"What Alheim and Ramston observed is that, back in 2006, there was a drug introduced called Rimonabant, which was an anti-obesity drug. It was a bit of a miracle drug. I want to quote this exactly because it's so important to understand the effects that this drug had on humans.*
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> *'Large randomized trials with Rimonabant have demonstrated efficacy in treatment of overweight and obese individuals with weight loss significantly greater than a reduced calorie diet alone.*
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> *In addition, multiple other cardiometabolic parameters were improved in the treatment groups, including increased levels of HDL, reduced triglycerides, reduced weight circumference, improved insulin sensitivity, decreased insulin levels. And in diabetic patients, improvements in HBA1C.'*
>
> *This paper was released in 2007. Unfortunately, Rimonabant had a side effect that it caused people to want to kill themselves. So, it was withdrawn from the market and it largely killed research for several years into that area."*

Why Rimonabant Worked — and a Safer Solution

Why does Rimonabant work to address the cause of obesity? Because it's largely related to excess consumption of LA, an omega-6 polyunsaturated fat, or PUFA, which acts as a metabolic poison when consumed in excess quantities.

One of the ways LA increases weight is by stimulating the endocannabinoid receptors. This causes "the munchies," very similar to cannabis. The drug effectively blocks this effect, causing people to lose weight. Goodrich explained:

"What Alheim did in 2012 was demonstrate that the mechanism behind Rimonabant is to block the metabolism of seed oils into the chemicals in your body and the endocannabinoid system that cause overeating. My experience when I stopped eating seed oils was that I forgot to eat carbohydrates.

The effect of Rimonabant in these mouse models is to make them crave carbohydrates and to stimulate them to eat sweet foods and carbohydrates. Everybody's familiar with this effect. It's called the munchies. And it's what you get after you smoke pot, because the endocannabinoid system is the system that marijuana affects and the chemical that Rimonabant blocks is your body's homologue to the THC in marijuana.

So essentially what we've done to ourselves is given ourselves a chronic case of the munchies, which is blocked by this unfortunately very harmful drug. This is as open and closed a case for causation as you're going to find in the medical literature.

We have a human drug that treats this, and as I just read, it treats all these different aspects of this disease. And it works through this one pathway that we have a clear demonstration of in animal models. In this case, the drug is completely pointless because the dietary fix is well known and is simple."

Soybean Oil, High in LA, Drives Obesity

Soybean oil, which is rich in LA, is also linked to obesity. In 2015, a research team found soybean oil induced obesity, insulin resistance, diabetes and fatty liver in mice. "Our results indicate that in mice a diet high in soybean oil is more detrimental to metabolic health than a diet high in fructose or coconut oil," they wrote in PLOS One, concluding:[13]

"While this study was in progress, two groups published papers with results similar to ours — namely, that a high fat diet supplemented with oils high in LA leads to obesity and fatty liver. Other studies have also shown that dietary LA

can cause adiposity in humans and lead to hyperglycemia as well as obesity in mice."

Two years later, they confirmed this by showing soybean oil modified to be low in LA caused less obesity and insulin resistance than the unmodified soybean oil.[14] Interestingly, in 2020 the team published additional research showing soybean oil.

Both the modified and unmodified versions produced genetic changes in the brains of mice, including pronounced effects on the hypothalamus, which regulates metabolism and stress responses.[15,16] In the case of the brain changes, LA was surprisingly ruled out as a cause, but it still identifies yet another reason to avoid consuming soy products, including soybean oil.

Excess LA Is the Key Reason Why Most People Are Overweight

Collectively, consuming too much LA is the primary factor driving the overweight and obesity epidemics. The obvious solution? Radically limit PUFA and LA to not stimulate the endocannabinoid receptors in the first place. PUFA also impairs mitochondrial function to decrease energy production, along with impairing thyroid function, so there are additional reasons to cut way down on your intake, even if you're not overweight.

Examples of seed oils high in omega-6 PUFAs include soybean, cottonseed, sunflower, rapeseed (canola), corn and safflower.[17] These processed seed oils and vegetable oils get integrated in your cell and mitochondrial membranes, and once these membranes are damaged,[18] it sets the stage for all sorts of health problems. With a half-life of 600 to 680 days,[19] it can take years to clear them out of your body.

They also get incorporated into tissues such as your heart and brain. One result of this could be memory impairment and increased risk of Alzheimer's disease. Canola oil, in particular, has been linked to Alzheimer's.[20]

How Much Linoleic Acid Are You Consuming?

Ideally, consider cutting LA down to below 7 grams per day, which is close to what our ancestors used to get before all of these chronic health conditions, including obesity, diabetes, heart disease and cancer, became widespread.

To do so, you'll need to avoid nearly all ultraprocessed foods, fast foods and restaurant foods, as virtually all of them contain seed oils. The easiest way to do this is to prepare the majority of your food at home so you know what you are eating.

Also, be aware that, because animals are fed grains that are high in linoleic acid,[21] it's also hidden in "healthy" foods like chicken and pork, which makes these meats a major source that should be avoided. Olive oil is another health food that can be a hidden source of linoleic acid, as it's often cut with cheaper seed oils. Instead, use tallow, ghee or butter to cook with. Ghee is better than butter as it has a higher smoke point.

If you're not sure how much LA you're eating, enter your food intake into **Cronometer** — a free online nutrition tracker — and it will provide you with your total LA intake. The key to accurate entry is to carefully weigh your food with a digital kitchen scale so you can enter the weight of your food to the nearest gram.

Cronometer will tell you how much omega-6 you're getting from your food down to a 10th of a gram, and you can assume 90% of that is LA. Anything over 10 grams is likely to cause problems.

The Timing of Your Meals Also Matters

Many people will experience weight loss as a "side effect" of reducing intake of LA. However, time-restricted eating (TRE) is another simple yet powerful intervention for weight loss. Our ancient ancestors did not have access to food 24/7, so our genetics are optimized to having food at variable intervals, not every few hours.

When you eat every few hours for months, years or decades, never missing a meal, your body forgets how to burn fat as a fuel. TRE mimics the eating habits of our ancestors and restores your body to a more natural state that allows a host of metabolic benefits to occur.[22]

If you're overweight or obese, I recommend limiting your eating window to six to eight hours per day instead of the more than 12-hour window most people use.

In one study, people following TRE had significantly reduced body weight and fat mass, while preserving fat-free mass, along with improved blood pressure, fasting glucose and cholesterol profiles compared to those following a regular diet.[23]

Ideally, stop eating for several hours before bedtime, then start your eating window in mid- to late morning after you wake up. TRE, combined with limiting LA intake, will help many people to lose weight and maintain a healthy weight, naturally.

Once you regain your metabolic flexibility then TRE becomes unnecessary, although it is still wise to not eat more than 12 hours per day, and to avoid food for three hours prior to bedtime, whatever your weight is.

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