CHAPTER THREE

…The action of alcohol on these chronic alcoholics is a manifestation of an allergy; that the phenomenon of craving is limited to this class and never occurs in the average temperate drinker. These allergic types can never safely use alcohol in any form at all…. – *Big Book of Alcoholics Anonymous, page xxviii*

The Physical Allergy

The Big Book of Alcoholics Anonymous, in the chapter "The Doctor's Opinion" describes addiction as having a physical/body component (physical allergy) and a mind/brain component (mental obsession). Dr. Silkworth, whose letters are quoted in this chapter, believed that alcoholics were "bodily and mentally different from their fellows." He believed that alcoholics (all addicts for that matter) were physically and mentally different than their non-addict peers. The current science (almost 100 years later) backs up Dr. Silkworth's original ideas.

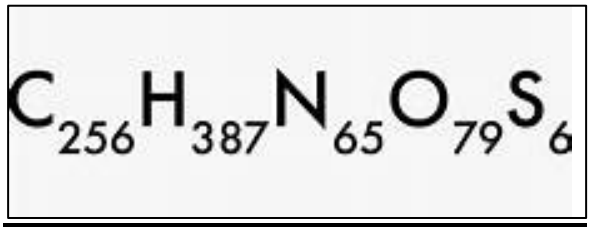
Compulsive eaters often have an abnormal reaction to food. Some of us overindulge and can't quit, and then we crave more. – *OA 12 & 12, page 4*

The phenomenon of craving is the hallmark of our "physical allergy." Dr. Silkworth used the term "allergy" because back then he didn't really understand it completely and he did not know what else to call it. Now the science clearly explains this phenomenon – how our bodies crave more despite our desperate desire to stop.

At its most basic, the physical allergy is hormonal dysregulation. Hormones are very powerful chemical messengers in our bodies and brains. They circulate in our bloodstream and tell our organs and cells what to do. They are in charge of our metabolism and metabolic functioning. Hormones have A LOT to do with eating behavior and fat storage. There are hundreds and hundreds of hormones in our bodies and new ones are being discovered all the time.

Several different hormones are messed up in our disease. This chapter focuses on the two most important hormones at the core of our physical allergy: insulin and leptin. I'm going to talk about them separately, but they work very intricately together. They also work along with two other brain systems that affect our disease: the lipostat and the satiety system. Again, I will talk about these things one at a time, but they all work together.

Insulin and Insulin Resistance



Whenever you hear the word insulin, you typically think of diabetes. It is true, diabetes is a disease that involves problems with insulin. However, insulin is involved in much more than just diabetes.

Dysregulated insulin is the foundation upon which our physical allergy is based. Insulin is a hormone made in the pancreas. Its job is to move glucose into our cells for energy and then store any extra glucose as fat. *So, most importantly for us**, insulin is the fat storage hormone.**

How is insulin supposed to work normally? Food we eat gets digested and the glucose (or sugar) from our food is released into the blood stream. In response to the level of sugar in our blood, insulin is released to move that sugar into our cells to use immediately as fuel, or store in our muscles for future use (glycogen). If there is any left-over sugar, then that sugar is stored as fat.

Almost all cells have receptors for insulin. Insulin functions like a key that unlocks the cell membrane so that glucose can move into the cell. After glucose moves into the cell, insulin levels are supposed to quickly go down to normal. However, when we have more glucose in our blood than our cells need, or can store, insulin will drive the excess glucose into our fat cells where it is metabolized and made into fat. When our bodies function well, this is a very balanced system. Our cells get the fuel they need, and our levels of body fat are in the optimal range for health.

Obviously, this is not how it works for most obese people. We have higher than normal levels of insulin floating around in our bodies ALL THE TIME. Our insulin levels never go back down like normal peoples' do. High insulin levels drive the whole obesity train and these high levels keep the physical allergy going.

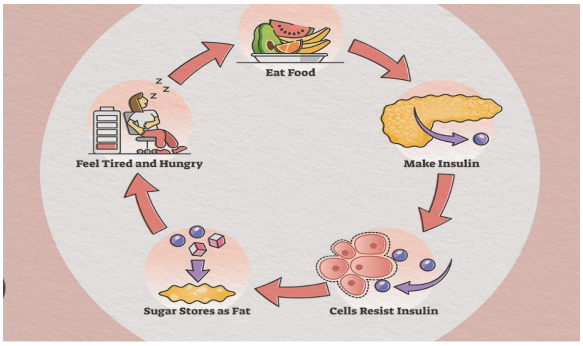
Obese people have much higher baseline, or fasting, insulin levels than normal weight people do. Our insulin levels are high whether we have food on board or not. We also secrete much higher levels of insulin in response to food than normal people do – even to the same types and amounts of food. This means we have an exaggerated insulin response to food. Our level of insulin remains elevated or higher after a meal. Our insulin level doesn't drop naturally after a meal like it does in normal weight people.

Remember, insulin is the fat storage hormone. Because of the overall higher level of insulin floating around our bloodstream, we are super primed to make fat. We are fat-making machines. How does this happen? How do we get this way? All the changes listed in the above paragraphs happen because **we are insulin resistant.**

We become insulin resistant because we consistently flood our bodies with insulin from overeating and bingeing on high-sugar, processed food. Our poor bodies are drowning in insulin. Our

bodies try to compensate and bring things back into balance. Our bodies want to be in what is called homeostasis (a state of balance required for our body to work properly). Our bodies want our biochemistry to be within certain parameters or ranges.

The way our bodies try to get things back in balance is by making our bodies resistant to insulin. The body does this by decreasing the number of receptors for insulin on our cells so that our cells are protected from getting too much glucose. **BUT** the combination of the decreased insulin receptors and our continued overeating/bingeing requires our body to increase the amount of insulin we secrete because it has to deal with the amount of sugar in our blood.



This is when we are considered insulin resistant. Our cells don't accept the key of insulin to allow glucose in, but we keep throwing sugar into our bodies which requires more and more insulin to get it out of our blood stream and stored safely away as fat. As a result, more of the sugar we eat gets stored as fat and less sugar gets into our cells for energy.

This is a self-perpetuating cycle – too much insulin leads to insulin resistance. Insulin resistance leads to more insulin being secreted which leads to more insulin resistance and on and on and on.

Eventually, even your fat cells become insulin resistant. They become unable to store the excess glucose from our bingeing, and this is when blood sugar levels remain high, and you are considered diabetic. This is why patients often report unexplained weight loss just before diagnosis – because ALL the cells are starving for glucose.

*** The first part of our physical allergy is insulin resistance. ***

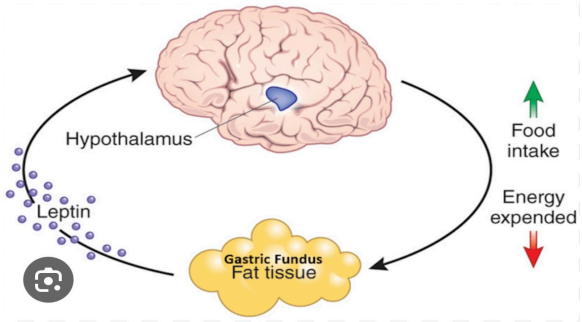
<u>Leptin and Leptin Resistance</u>

Now we are going to talk about the other half of the hormonal dysregulation of our physical allergy – leptin. Leptin is a hormone that is mostly secreted by our actual fat cells. It is a major satiety hormone. That is – **Leptin signals our brain that we are full and don't need to eat anymore**. It tells our brain to lose interest in food and to get physically moving. When leptin is working normally, it travels to the brain to turn down hunger and prevent more fat storage.

Leptin is the way our brain knows how much stored energy (fat) we have on our bodies. Humans need a baseline percentage of body fat for optimal health. When the brain doesn't register or "see" leptin, it panics. It believes we are STARVING. And I mean literally starving. Our brain isn't seeing how much fat we have on our bodies. It thinks we have zero fat stores – which is a serious threat to our survival. So our brain tells us to eat everything in sight and to conserve energy by being a couch potato.

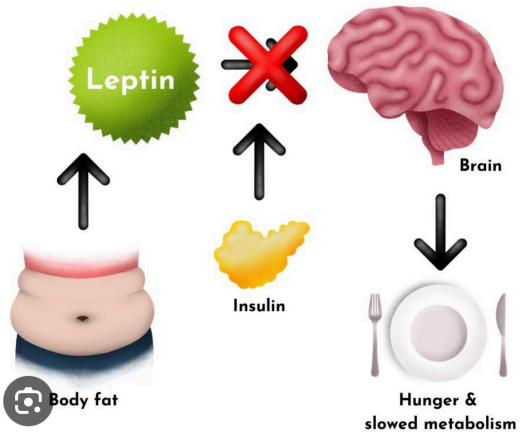
Now, you'd think as food addicts with plenty of fat on our bodies, we would be drowning in leptin. And we are! So why doesn't it work to stop us from eating like it does for normal people? It doesn't work because **we are leptin resistant.** Our leptin never reaches our brains to shut off our feelings of hunger.

We become leptin resistant because of our insulin resistance. Insulin blocks leptin because these two hormones use the same brain pathway to reach the brain. Since we are insulin resistant, our constant high level of insulin "gunks up or clogs up" the pathway making it impossible for leptin to also use this pathway. **Our insulin resistance causes our leptin resistance.**



First and foremost, leptin is secreted by our fat cells to keep our brain informed about how much fat we have stored in our bodies. BUT it is also secreted by our stomach after we eat, to tell our brain that we've eaten enough energy for a particular meal. However, obese people's secretion of leptin from the stomach actually FALLS after eating (it should go up). This leads to less leptin going to the brain. So instead of feeling full, we feel hungrier. This is why, at Thanksgiving, your family is in a food coma on the couch and you're in the kitchen eating up leftovers. This is one of the reasons why we NEVER feel full.

This is why I ate past being full, past feeling sick, past feeling pain and only stopped when I ran out of food.



***** The second part of our physical allergy is leptin resistance. *****

Insulin resistance and leptin resistance become a nonstop cycle. Overeating/bingeing leads to insulin resistance which leads to leptin resistance which leads to more overeating/bingeing which leads to more insulin resistance and on and on and on.

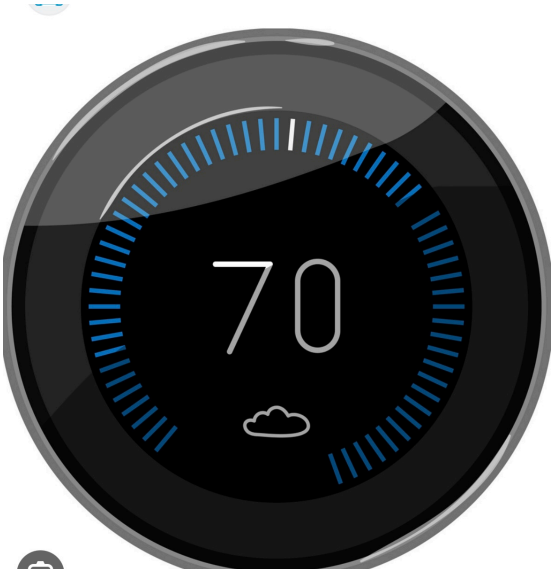
This is one way insulin and leptin resistance work in our disease. They keep us eating and making/storing more fat. But insulin and leptin resistance work in another way to keep us in our disease and prevent us from losing weight.

<u>The Lipostat</u>

Insulin and leptin work with something called the lipostat to keep us eating and to keep us fat. The lipostat is located in the hypothalamus of the brain. It determines how much fat your body

SHOULD store. How much it thinks you should store to be in optimal health. (Again, because we do need some fat stores on our body for it to work properly.)

Think about the thermostat in your home. You set your thermostat at 70 degrees Fahrenheit. The thermostat will keep track of the temperature of the house. If the temperature falls below 70 degrees, the thermostat will turn on the furnace. Your lipostat works the same way. It keeps track of the amount of fat on your body and drives you to eat more if you fall below that level of fat.



Of course, in our disease, the lipostat system is also dysregulated. Our high levels of insulin push the setting of our lipostat higher. So instead of being set at 70 degrees – it's mistakenly set at 90 degrees. The furnace just keeps pumping more heat, in other words we just keep eating more and more. When our lipostat is set too high due to our insulin and leptin resistance, our brain sets our optimal level for body fat too high – and it will do anything and everything it can to maintain that higher level of fat on our bodies. It will fight tooth and nail to keep us fat and prevent us from losing weight.

The lipostat keeps us fat by regulating both our hunger hormones and our satiety hormones. It increases the hormones that make us feel hunger and it decreases the hormones that make us feel satiated or full. It also makes us burn less calories by slowing our overall metabolic rate.

So, in a nutshell, the whole cycle works like this:

- We eat tons of highly processed food, fast food and tons of sugar.
- This causes high levels of insulin secretion which eventually leads to insulin resistance.
- Insulin resistance leads to leptin resistance which leads to more bingeing.
- All this drives up our lipostat fat setting which leads our bodies to defend against and mightily resist fat/weight loss.

<u>The Satiety System</u>

The satiety system is a system in the brain that registers when we've had enough food, and it stops us from eating more. This system works in conjunction with the lipostat. The fact that your non-addict friends can eat two bites of dessert and stop has nothing to do with willpower. It just means their satiety system is working correctly. The satiety system makes the decision if we've eaten enough by taking in information from 3 areas: The digestive tract, the brain reward system and the lipostat.

First, it gets information from the digestive tract – mostly through volume (the amount you are eating) and the protein/fiber content of your food. The satiety system asks: Is there enough volume, fiber and protein in this meal to be full? If not, you're not full, keep eating.

Second, our satiety system also receives information from the reward system in our brain (where we get our EFFECT). The satiety system shuts down the feeling of satiety and fullness when we eat highly rewarding foods like pizza, ice cream and donuts. That means we can eat a lot more of that kind of food than we can broccoli. This means we can eat a whole apple pie but would have trouble eating three whole apples in one sitting. So the question for the satiety system is: Is highly rewarding food available right now? If so, then you're not full, keep eating.

Third, the satiety system also gets important information from the lipostat. The lipostat decreases our feelings of fullness to help maintain the fat stores on our body according to the fat set point it has set. And ours is set too high. So, the lipostat tells the satiety system if it thinks you have enough stored fat on your body. So the question for the satiety system is: Does the lipostat think you have enough fat on your body? If not, you're not full, keep eating.

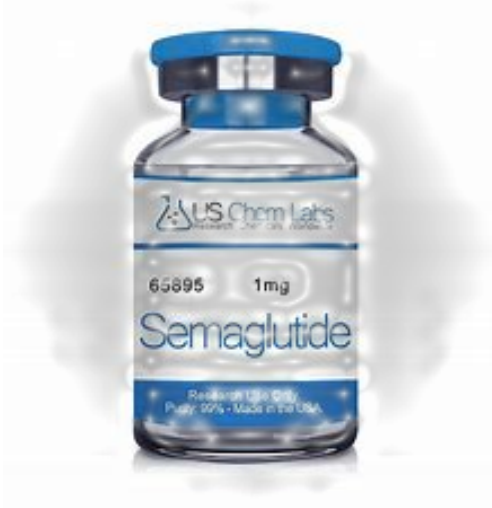
Conclusions

The GOOD news is that the hormonal dysregulation and dysfunction of our disease can be corrected. We can reverse our insulin and leptin resistance. We do this through our abstinence and food plans.

Most of the obesity experts I read believe the NUMBER ONE thing we need to do to lose weight and re-regulate our hormonal system is to lower our insulin levels. This is where I started in developing my food plan. I wanted to drive down my levels of insulin so my leptin could be read by my brain and the fat setting on my lipostat could be turned down.

*****We will go over strategies for dealing with insulin resistance/leptin resistance during the workshop session. *****

A Word About Semaglutide



Ozempic/Wegovy medications are all the rage right now to fight the obesity epidemic. These drugs are GLP-1 agonists. GLP-1 (glucagon-like peptide) is a hormone that is released in the gastrointestinal tract in response to eating. These drugs are "agonists" meaning they make your GLP-1 work

better. These drugs work to help you lose weight in a few different ways:

1. They delay stomach emptying (slows down how quickly food travels through your digestive tract) and they send signals back to the brain's hypothalamus that promote feelings of fullness and suppress hunger. This is how they increase satiety (feeling of fullness) and decrease appetite.

2. They also promote insulin sensitivity. They make insulin work better so you don't need to secrete as much. This helps lower your overall/baseline insulin level. This lowering of your insulin also lowers the lipostat's set point for your weight/adiposity (fatness).

3. These drugs also decrease the amount of reward your brain gets from food. It dampens the motivational dopamine system which is part of your brain's reward circuitry. (We will be talking about this reward circuitry later in this study guide.)