

I will not
be **FAT** again

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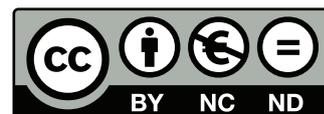
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I also want to thank Gary Taubes for his terrific work exposing the energy balance paradigm. His ideas changed my life for the better.

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INTRODUCTION

My name is Vicente and I am an engineer. I lack formal background in medicine or nutrition, and this document is just a summary of my opinions, not a medical advice or treatment. I was overweight and obese as an adult. In mid-2013 a physician advised me to lose weight, and he recommended me to count the calories I ingested. I followed his advice, I started to control my calorie intake, and I lost a few kilos, but the weight loss stalled. And this happened while I was still eating a low amount of food, the same quantity that made me lose weight. This led me to start reading on the internet about the rebound effect, looking for a way to avoid it. According to what I learned, I changed my way of eating and, perhaps as a result of that, I started to lose weight again. At the end of 2013 I reached my weight-loss goal. That was more than two years ago and I have not regained body fat. Perhaps I have gained muscle mass.

My way of eating, my diet, when I had a weight problem was not ideal. I used to drink more Coke than water and, although my way of eating was generally healthy, I often ate pastry or products with added sugars.

Looking at it from a distance, I was not that smart. It never occurred to me to read about nutrition and to seek a solution for my weight and health problems.

Since I started to read about nutrition, I have not stopped doing so. Perhaps I know nothing about nutrition but I have my ideas and a rather critical opinion about many of the things we are told about what are the causes and solutions for obesity, and about what we are told has been published in the scientific literature on obesity. From my point of view, it is necessary to call into question everything we think we already know about nutrition, obesity and weight loss. No source is reliable, not even official bodies.

I have no hidden economic agenda driving my opinions. I do not have it and I will NEVER have it. If I started a blog, it was only because of my desire to communicate my views. Although I have spent a lot of time reading and writing about nutrition, I have spent that time because I believe that if we had better information about nutrition, perhaps our weight and health problems could be prevented or reverted. We should have an active attitude towards the problem, and we should be much more critical of the messages that we receive from most of the people who are supposedly caring of our health.

THE FRAUDULENT ENERGY PARADIGM

We live in the paradigm of the amount of energy. If you have excess weight, you are told you have eaten too much, and you are told to count calories and to reduce your caloric intake. If you look at the packaging of any edible product, you will find a label that tells you how many calories that product has. You can install apps on your mobile to help you track how many calories you eat and how many calories you burn walking throughout the day. The message we receive is that the body weight management is a matter of energy: we are told to regulate how much energy we ingest and how much energy we spend with our physical exercise.

But this is a fraudulent paradigm. Our body weight does not react to the energy in food nor does physical exercise allow us to control our energy expenditure.

When we make decisions thinking in terms of energy, unfortunately we make wrong decisions.

I will try to summarize my ideas in this document. Thank you very much for reading this book.

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English is not my mother tongue; please excuse any errors on my part.

Declaration of conflict of interest: no conflict of interest.

CAP. 1 THE ENERGY BALANCE EQUATION

Imagine that your business is having a tough time due to the robberies it suffers on a regular basis. You decide to consult an economic adviser. You explain the situation to her and she diagnoses that the problem is that your earnings are lower than your expenses. According to her, that is what a basic economic law says: profit is equal to income less expenses. She suggests that the solution is to take measures that will increase the revenue and/or reduce the expenses. For example, she proposes that you double the price of the products you sell, which according to her will increase the income, and that you sack half of the workforce, which she believes will reduce the expenses.

Surreal, right? If we were to describe this adviser's behaviour, we would say that she made a wrong diagnosis, although formally it is true that the problem is that your income is less than your expenses, and that she proposed as a solution the adoption of stupid measures, which are stupid because they neglect the actual consequences of implementing them.

The subtraction of two numeric values, income and expenses, does not help understand your business' problem and neither will it help to find appropriate solutions.

You may think that no one can be as stupid as the economic adviser in the example. Well...you are wrong. What I have described is exactly what it is happening in the nutrition field with obesity and weight-loss diets.

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The nutritional adviser ignores the real cause of the problem and diagnoses that the cause is always eating more energy than is spent. The solutions she proposes are based on a general law of physics, and, just as in the case of the business, the adviser despises the actual consequences of such measures.

The height of this nonsense is that the adviser brags about being the science defender and accuses her critics of denying inviolable laws.

We are not talking about rare black sheep, but rather what most nutrition experts have been doing for decades. Are we surprised that we can't control our body weight?¹

THE CONSERVATION OF ENERGY PRINCIPLE

The first law of thermodynamics states that energy is neither created nor destroyed. Once we select a geometric boundary for its application, energy that enters that boundary can not disappear. When this law is applied to the human body, it says that the energy that enters the body is either stored, or it exits the body. It can not disappear:



The first law of thermodynamics is always fulfilled. What are open for discussion are theories supposedly deduced from that law, which, as I will explain, are the result of misguided thinking.

THE CONSERVATION OF ENERGY PRINCIPLE DOES NOT SPEAK OF BODY FAT

The apologists for the use of the energy balance equation in obesity mix up the energy accumulated in the body with body fat.

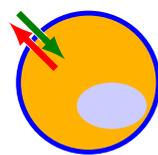


But that is not necessarily true. Energy is also accumulated in other forms, *e.g.* muscle or glycogen. The first law of thermodynamics does not talk about body fat.

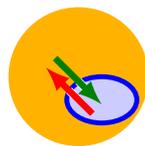
This may seem an unimportant detail, but it is very revealing. If anyone believes that the laws of thermodynamics say that an excessive energy intake and a sedentary lifestyle cause accumulation of body fat, they have a difficult task explaining what these laws say about what makes us store energy in the form of

muscle mass. It is no coincidence that muscle mass is quietly removed from the equation, because it exposes the fraud of the energy balance theory.

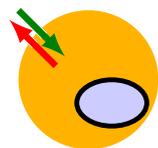
When applying the first law of thermodynamics, we must choose a specific physical boundary, a physical limit for its application. If the human body as a whole is chosen, that law tells us nothing about energy accumulation in a particular tissue and it is, therefore, irrelevant to the study of the accumulation of triglycerides in the adipose tissue, in the same way that it tells us nothing about why our muscles increase in size. If, on the other hand, we choose to apply the law in a specific organ or tissue, the law is useless, as we know that biological forces (hormones, enzymes, etc.) are responsible for making fatty acids enter or leave the fat cells. The energy balance theory combines terms that come from applying the first law of thermodynamics using both boundaries, what is a conceptual aberration.



First law of thermodynamics applied in the **body**



First law of thermodynamics applied in the **tissue**



Energy Balance Theory

A PSEUDOSCIENCE THAT LACKS PHYSIOLOGIC PLAUSIBILITY

What is the physiologic mechanism that links the changes in all the energy stored in our body—in all its forms—and our caloric intake (which is the only term in the energy balance equation that is under our control)?

Just like any other pseudoscience, the energy balance theory is fundamentally fictional, outside of real complications and unable to propose a plausible physiologic working mechanism.

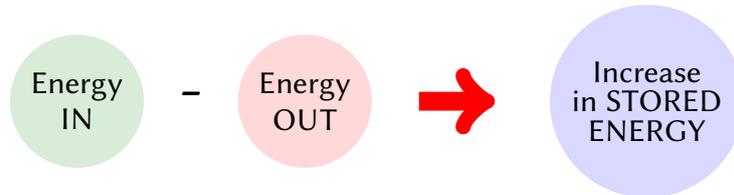
THE ILLUSION OF CONTROL

The illusion of control is the belief that we control aspects of a situation that are not actually under our control. We can control how much food we eat, no doubt about that, but we have no control over the effects of that food. We cannot control how much of the ingested energy will be used for mechanical work, how much will be used for maintaining our body's temperature or how much will be accumulated in the form of body fat or muscle mass. The energy partition is not static², nor is determined by the calories in the food, which is the only term

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under our control in the energy balance equation. The human body does not work with a fixed efficiency; it can dynamically change how much energy goes to each body function. This partition depends on many factors: our usual diet³, what we have eaten today⁴, our metabolic status⁵, our usual physical activity⁵, if we are restricting the amount of food⁶, etc.

The energy balance theory, on the other hand, is based on the illusion of control: this theory tells us that the energy expenditure term is constant or at least controllable by means of our physical exercise. If that were true, we could control our body's energy reserves by changing the amount of food we eat and the amount of exercise we do:



But this is not true. We have no control on the energy expenditure of our body⁶⁻⁸ and therefore we cannot control how much energy it accumulates nor in what format (body fat, muscle, glycogen, etc.).

The most common expression of this fallacy is to assume that the energy expenditure has a specific value: “if today I eat 2500 kcal and I spend 2400 kcal I am going to gain weight”. That rhetorical trap sets up an unwarranted causality in the laws of physics. Since the energy expenditure is not under our control, in the previous argument it should be made clear that the energy intake is the only term under our control: “if today I eat 2500 kcal, that energy will be partitioned between changes in the accumulated energy and the energy expenditure”. To say that the energy balance theory is a hoax is not the same as saying that the laws of physics are not fulfilled.

IF ALL YOU HAVE IS A HAMMER, EVERYTHING LOOKS LIKE A NAIL

For the advocates of the energy balance theory, all causes and solutions for obesity are energy-related. But that misconception is caused by looking at the problem from a one-dimensional point of view. The first law of thermodynamics only considers energy. The causes and solutions for obesity that are wrongly deduced from this law only consider variations in energy as options: increase or reduce calorie intake, increase or reduce energy expenditure.

Under this paradigm, if something is fattening, it is so because it makes us eat too much or makes us have a reduced energy expenditure. If something makes us slim down, it must be because it makes us eat less or increases our energy expenditure.

Does it seem logical? It is not. We are making the mistake of assuming that our body responds to energy. We have based the analysis on a law that only speaks of energy and, logically, the conclusions that can be reached only contemplate one dimension: energy.

Under this paradigm, two diets with the same energy, *i.e.* isocaloric, must produce the same results in terms of body fat accumulation. We are told that the

composition of the diet is not that important (although it may be so for health, they concede).

Although the scientific evidence clearly contradicts the idea of calories determining our body weight⁹, understanding the conceptual errors at the basis of the energy balance pseudoscience might not be that easy. I believe that the best way to explain those errors is to talk about the causality problem.

MAKING UP A CAUSALITY WHERE THERE IS NONE

Let us assume that the following hypothesis is correct:

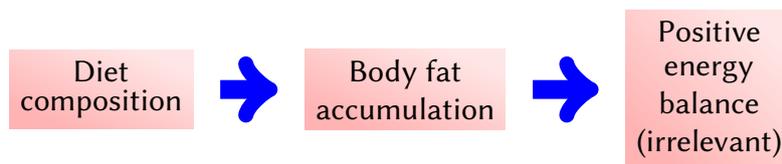
The accumulation of fat in our adipocytes is hormonally regulated.
The main hormone involved in the regulation of body fat is insulin.

I do not pretend to convince anyone now of the previous idea. I just want to use it as a hypothesis in order to analyze the problem of causality.

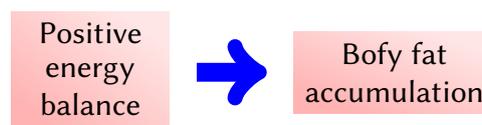
Imagine that you ingest a certain amount of food. Because of the composition of that food your body secretes insulin, in a quantity that depends also on your physiologic status.

Under the working hypothesis, the insulin segregation that the food causes in a person will determine how much fat is stored in the adipose tissue of that person. The rest of the energy in food can be stored as muscle mass or can be dissipated as heat or used to perform mechanical work.

What has happened in this example is that the composition of the food has determined how much fat is stored, something that has not been determined by the amount of energy consumed, but rather by other characteristics of the diet and the person.



I reiterate that I do not claim at this moment that our body works in this way. What I am saying is that it is a possibility. The laws of thermodynamics do not say that the accumulation of body fat will be determined by the difference between the energy intake and the energy expenditure. That assumption would mean interpreting that there is a specific causality, *i.e.* that changes in two terms of the energy balance equation determine how the third term of the equation changes.



This interpretation is unwarranted: our body does not necessarily behave in this way. Not necessarily it is a “caloric excess”, the existence of a difference between energy intake and energy expenditure, what **causes** body fat accumulation¹⁰. Let us consider that the effects caused by a stimulus (*e.g.* diet or exercise)

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in an animal are studied by observing its physiology and behaviour. We cannot predict the result from general laws of physics that are of universal application. Especially when they consist of a mere subtraction.

The food and diet industries want us to deduce that causality, but it is a fraud:

The underlying cause is a positive energy balance, having weight gain as a result, *i.e.* when the calories consumed exceed the calories that are spent.

Of the two formulations shown below, which one is the right formulation for the First Law of Thermodynamics, the one on the left or the one on the right?

The caloric intake and the energy expenditure determine the changes in the body weight

The caloric intake and the changes in the body weight determine the energy expenditure

None of them is the First Law of Thermodynamics, although both of them are compatible with this law of physics.

The formulation on the left is the CICO theory (Calories In Calories Out). This formulation implicitly assumes that the adipose tissue is a passive tissue that stores that part of what we eat that is left after the body uses what it needs. My point here is that this formulation defines a physiologic behavior for the body and that physiologic behavior is compatible with the First Law of Thermodynamics, but it is not necessarily correct.

The formulation on the right is also compatible with the First Law of Thermodynamics. In the physiologic behavior proposed by this second formulation, the adipose tissue has an active role in the fattening process, while it is assumed that the rest of tissues and organs of the body can manage what the adipose tissue does not store.

According to the formulation on the left, any weight problem can be solved by adjusting the energy intake and the energy expenditure. This detail is important.

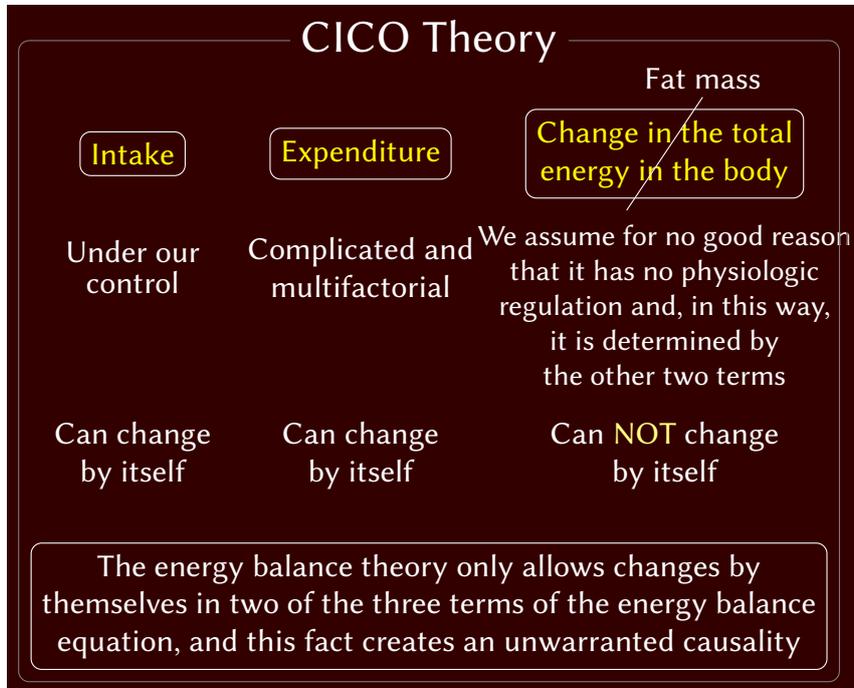
According to the formulation on the right, the energy intake and the energy expenditure have no influence on the growth of the adipose tissue. According to this formulation, talking about the terms of the energy balance does not allow us to control our body weight. Again, I want to emphasize that this is an important detail.

In conclusion, talking about calories only makes sense if the formulation on the left is correct, if, by chance, the human body behaves as this formulation assumes. If the formulation on the right is the correct one, trying to manage our body weight by changing the energy intake and the energy expenditure is completely meaningless.

"Caloric excess", "eating above our needs", "caloric deficit", etc. are expressions that only make sense within the formulation on the left, and, therefore, their use is always fallacious, since their mere use is equivalent to assuming the validity of the CICO theory.

The greatest stupidity in the history of humanity is to have assumed as obvious that the formulation on the left, the CICO theory, is the First Law of Thermodynamics itself, and that, therefore, it is not a hypothesis but an indisputable

fact of physics, because "energy has to be conserved". We are told that CICO "is physics" and that "it is correct" because "in terms of energy $2+2=4$ ". The direct consequence of this insane mistake is to assume that it is possible to control our body weight by controlling the calories we eat or the physical exercise we do. This conclusion will always be reached in the CICO theory, since it is implicit in its unwarranted formulation.



Notice how the advocates of the energy balance pseudoscience blame the complexity of the intake and energy expenditure terms for the bad results of their quackery. But, for them, only two of the three terms of the energy balance equation are relevant. The third one has no dependencies, no complexity, nor, of course, the possibility to change by itself.

The CICO dogma is that the adipose tissue is passive, receiving what the rest of the body does not need. You can consider a high level of complexity for those two terms of the equation, but that does not make this theory correct, nor, of course, what physics says.

THE TAUTOLOGY

Saying that a person gains weight because he consumes more calories than he spends is like saying students who arrive late to class do so because they enter the class after the bell rings¹¹. That description does not help at all, because it does not help understand the causes of the problem, nor it helps propose useful solutions. Perhaps the student has arrived too late because her car has suffered a mechanical breakdown, while by saying that the problem is caused by arriving later than the ringing of the bell, the only options we are offered are arriving earlier or delaying the time the bell rings. The tautology does not correctly identify the cause of the problem, nor does it help to deduce useful solutions^{1,12,13}.

Another analogy: our son has unintentionally broken the commander of the thermostat in our house. When we arrive from work, we notice that the house

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is colder than expected. By using the laws of physics, you can diagnose that the problem is that insufficient heat is generated, relative to the amount of heat that is lost, and you can think that installing more heat radiators or improving the thermic insulation of windows and doors are sensible solutions to this problem. The laws of physics are of no help to diagnose or to solve the problem.

CONTRAST WITH OTHER GROWTHS IN AN ANIMAL

If there is accumulation of energy in an animal, that is, if the animal grows in any way, in the organ that grows more energy comes in than goes out. That is true. No one denies that the first law of thermodynamics is fulfilled. The error is inferring causality, as I have explained above. The error is saying that energy accumulates **because** we ingest more energy than we spent. Making up the causality is unwarranted.

If a person increases her muscle mass, she ingests more energy than she spends. Does she increase her muscle mass because she ingests more energy than she spends? Is that what the laws of thermodynamics say, that to build our muscles we have to eat a lot and exercise as little as possible? Do these laws say that in the case of body fat but not in the case of muscle mass? The contradiction is an insurmountable obstacle for the defenders of this pseudoscience.

If a child is growing, he ingests more energy than he spends. Is he growing because he consumes more energy than he spends?

Why does a tumor grow? What causes gigantism?¹⁴ What causes dwarfism?

Why is obesity the only tissue growth that is considered to be caused by ingesting more energy than is spent? Is obesity the only physiologic condition where the energy balance is the cause of the growth, and in all other tissue growths the cause is hormonal/biological?

Among all the tissue growths in a living being, **only in one case** it is proposed that the growth can be successfully managed by controlling the caloric intake and the energy expenditure.

And we are told that this proposal derives from laws of physics that are both **inviolable** and **universal**.

99.9% of scientists see nothing wrong with this exposition.

MAKING UP A PHYSIOLOGIC BEHAVIOUR FROM A PHYSICS CONSTRAINT

When you eat more calories than you burn, the excess calories are primarily shunted into your adipose tissue. Your adiposity, or body fatness, increases. It really is as simple as that

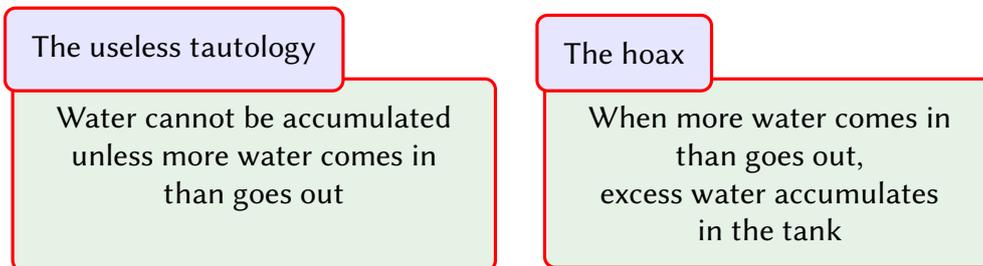
Stephan Guyenet, PhD

No, it is not as simple as that. As a matter of fact, that idea is a perfect example of the energy balance pseudoscience. The laws of physics do not tell you how things work, but rather the constraints under which they work. Whatever happens in a system, its behaviour cannot violate nature laws: matter cannot be created from nothing, an object will not accelerate unless a net force is applied, or energy cannot be created nor destroyed. Nevertheless, those limitations are often irrelevant in practice. For example, according to the Conservation of Matter Principle, you cannot accumulate matter in your body unless more matter enters the body than exits. But that fact is irrelevant for understanding growths in a living being¹⁵.

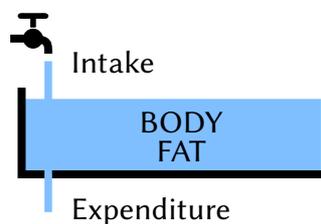
What I want to explain in this section is that the fraud in the energy balance theory does not lie in the maths —since this theory is indeed compatible with The First Law of Thermodynamics—, but in the physiologic behaviour that this theory makes up by using language tricks.

I am going to use a water tank as an analogy. Water is poured regularly into the tank and part of its contents is lost through a drain. We know that water cannot accumulate in the tank unless more water comes in than is lost through the drain. That is as true as useless, because it is just saying “accumulation” with different words. But, do you think that “when more water comes in than goes out, the excess water is accumulated in the tank”?

I am going to present two simple models based on a water tank. Both of them are compatible with the physics laws, since water is not created nor destroyed in any of them, but they behave differently. I insist: my point is that the energy balance theory is a fraud not because of its maths but because it makes up an unwarranted physiologic behaviour.



Model #1



Let us assume, for example, the following behaviour of the drain: the rate at which water flows from the tank is constant. In this case, if the rate of water flowing into the tank is bigger than the drain rate, water will accumulate in the tank. We could say “excess water” accumulates in the tank.

But let us assume now that the drain rate is adaptative and equal to the rate of water poured into the tank. Would you say that, in this case, when more water comes in than goes out, excess water accumulates in the tank? No, it does

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not, and, in this case, there is no such thing as “excess water”. Not always it is correct to say that “when more water comes in than goes out, the excess water is accumulated in the tank”.

It is our physiology knowledge what would lead, where appropriate, to talk about “caloric excess”. From the First Law of Thermodynamics we cannot deduce a physiologic behavior, which is what the fraudulent theory of the energy balance does.

For example, the storage of carbohydrates as glycogen cannot be caused by an intake that exceeds their oxidation, because that would mean that by increasing our carbohydrate intake we could gain as much weight as we wanted. But physiology says it is not like that.

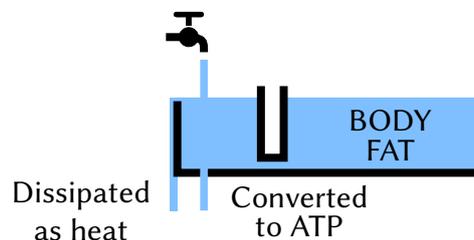
a chronic imbalance between carbohydrate intake and oxidation cannot be the basis of weight gain because storage capacity is limited and controlled, conversion to fat is an option which only occurs under extreme conditions in humans, and oxidation is increased to match intake

Broskey et al.

Physiology determines if it is correct to speak of a specific “excess” as a cause of a specific accumulation.

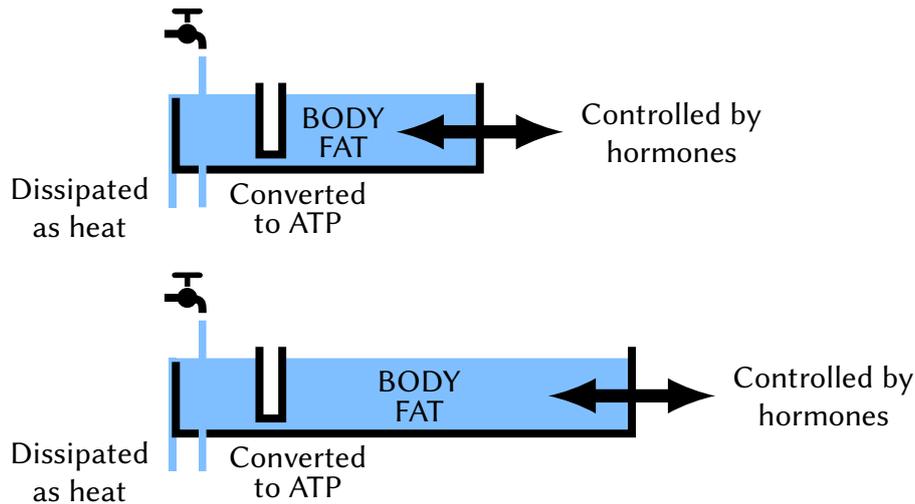
Model #2

In this model part of the contents of the tank is lost through the drain, but part is lost because it overflows the walls.



Please note that our body has physiologic mechanisms that can dissipate unnecessary nutrients as heat. One of these mechanisms are the uncoupling proteins, which can be found in several organs and tissues in our body.

Moreover, in this model the walls of the tank are not fixed, but they can dynamically expand or contract, changing the total volume of the tank. In this model the position of the walls is regulated by the concentration of specific substances in the water.



Let us assume that a specific substance is present in the water and it makes the tank expand. As a consequence of that expansion, the total volume of water stored in the tank increases. Would you say that, in this case, when more water comes in than goes out, “excess water” accumulates in the tank? No, this not correct. The use of the term “excess water” is unwarranted.

Water is not accumulated because more water comes in than goes out, although more water will come in than go out when water accumulates.

This model does not violate universal laws of physics —water is not magically created nor destroyed— and the existence of an alternative model that is also compliant with those laws, but has a behaviour different from the energy balance theory, shows that the energy balance theory goes beyond the physics constraint established by the First Law of Thermodynamics: it introduces an unwarranted physiologic behaviour. As I said before, the fraud of the energy balance pseudoscience does not lie in the maths but in the physiologic behaviour that it makes up.

Fundamentally, obesity is a problem of energy imbalance, which only develops when energy (food) intake exceeds total energy expenditure

Schrimpton *et al.*

Obesity is not a problem of energy imbalance: it is a problem of triglyceride accumulation in the adipose tissue. There is no physiologic basis for talking about “energy” or “energy excess”.

CALORIC SURPLUS AND CALORIC DEFICIT ARE ALWAYS FALLACIOUS EXPRESSIONS

“Caloric surplus” and “caloric deficit” are terms that implicitly assume that the energy balance theory is correct^{16,17}. Therefore, their use in the premises of a pretended demonstration of the validity of this theory is always fallacious.

If you are in caloric surplus you
will gain weight, regardless of your
insulin levels

Anonymous

In the above sentence the result (“weight gain”) is redefined using an expression (“caloric surplus”) that would be only warranted if the theory that is supposedly being demonstrated were correct. This expression implicitly points out calories as the cause of the result. When this expression is included in the premises of an argument that supposedly proves that the energy balance theory is correct, the *petitio principii* fallacy is used.

In addition, “caloric surplus” is included in the premises of the argument with one meaning (“eating a lot”, something under our control) but it is later interpreted with a different meaning (“the intake is greater than the expenditure”: the output “weight gain”, something that is not under our control). This trick deceitfully makes that premise, that talks about calories and that is reinterpreted as “weight gain”, seem as inevitably required for “weight gain”. The use of the *petitio principii* and ambiguity fallacies lead to the deceptive conclusion that the physiologic processes are irrelevant and that calories are all that matter.

The result is redefined using an expression (“caloric deficit”) that would be only warranted if the theory that is supposedly proven correct were correct. This expression points out calories as the cause of the result.

*Petitio
principii
fallacy*

Thanks to the simultaneous use of two different meanings of “caloric deficit” the result is included as a premise of the argument. Thanks to this trick, that premise seems an unavoidable requirement for weight loss.

*Ambiguity
fallacy

Petitio
principii
fallacy*

GLUTTONY AND LAZINESS

As I mentioned when I was talking about the illusion of control, the defenders of the energy balance theory claim that an excessive energy intake and/or lack of physical exercise are the obvious causes of obesity¹⁸. If the energy intake is tracked down and they do not see anything unusual, they conclude that the problem then has to lie in the lack of physical inactivity¹⁹. If they measure the physical activity and they do not see anything unusual, they conclude that the problem has to be overeating²⁰.

They made us all believe this. But, as I have already explained, a universal law of physics cannot be used to deduce the response of an animal to a stimulus. There is no reason to think that a person who does not exercise is going to get fat, nor to expect that a healthy person that follows a healthy diet will gain weight,

no matter the calories they consume. An experiment in which someone is forced to eat an exorbitant amount of food does not contradict this thought.

An excessive calorie intake or lack of physical exercise are not causes of obesity deduced from the laws of thermodynamics. The origins of these ideas are the bias and prejudices against the obese, *i.e.* the irrational idea that their weight problem is a consequence of lacking willpower.

Moreover, we should not confuse an association with a cause-effect relationship. Although, hypothetically, we could prove that there is a lack of physical activity and an uncontrolled appetite in obese people, these could just be symptoms of the problem and not necessarily their cause^{21,22}.

PREJUDICE MAY HAVE A BASIS IN REALITY

It is a possibility that the real cause of obesity is that, out of a sudden, all the Western World is consuming too much food and being sedentary. I do not think so and it seems to me an absurd hypothesis that that has happened simultaneously in places in the world whose local customs and ways of life are quite different. Even if such prejudice against the obese had some sort of basis, what is in no way acceptable is saying that this ideology is deduced from the laws of physics. It is not and this is not debatable.

CAP. 2 CALORIES DO NOT MATTER

The previous chapter was dedicated exclusively to explaining why the energy balance theory is a fraud. Why do I go on talking about calories in the second chapter? Because I suspect that, although we understood that the use of the energy balance theory in obesity is nothing but a con, it is possible that you still find it sensible to say that if you ingest 1000 extra kcal per day you will gain more weight and, therefore, calories do matter, whatever I may say about it. It is also possible that you still think that the solution to obesity should be eating less, certainly not eating more.

In my opinion, focusing on calories always implies ignoring the composition of the diet, as calories make the information about what exactly we are eating disappear. Since this information is important to control our body weight, talking about calories is always a mistake.

This does not mean that the amount of food may not be important in some cases. The amount of food and calories are not the same thing.

Essentially what I am saying is that the total amount of calories in the diet is not the parameter that determines our body weight. Dietary decisions based on that parameter are wrong decisions.

BUT FAT PEOPLE EAT A LOT OF FOOD

Maybe this is what you think you see. Actually what you are seeing is that they consume a lot of **unhealthy** food. What you also see and you have ignored is that you surely do not know a person who, while eating healthily (water, eggs, meat, green vegetables, fruit, etc.), consumes a large amount of food or has weight problems. Do you think that that person would be fat if she ate a little more of food every day? I do not think so, because I do not think you have to worry about the amount of food you eat when you eat the right foods.

You think you see a problem caused by quantity, but you are seeing a problem caused by quality, which is aggravated because the bad quality, the real cause of the problem, has as a side effect an increase in the amount of food ingested. In the absence of a scientific experiment that confirms if it is possible to gain weight eating lots of healthy food, what you see is the combined effect of poor quality and too much quantity. You cannot blame quantity alone, just because it is what you believe you see. Perhaps quantity is not a problem when quality is OK.

OVEREATING THOUGHT EXPERIMENT

An argument that is often used to defend the energy balance theory is that if you added 500 or 1000 kcal to your usual caloric intake, you would gain weight. The proponents of this idea conclude that this thought experiment demonstrates that calories matter. This argument is flawed in multiple ways.

If you want to know the outcome, you have to do the experiment

An obvious problem of this idea is that it assumes an outcome for the experiment, but nobody is actually doing the experiment²³⁻²⁵. Assuming an outcome only shows what our beliefs are. It is not that clear to me that while eating real food, steadily eating a little more food will make you fat.

The composition of the diet can be changed

A less obvious trick in this argument is that it only considers changing the calories in the food, assuming that the total calories of the diet is the only thing that can be changed on a diet. It is not acceptable, since, as a matter of fact, what we are talking about is precisely whether the composition of the diet matters or not. The argument should be changed to include analyzing the effect of the composition of the diet. If you do not check the relevance of the composition, you cannot conclude that it matters.

Eating more is not the same as consuming more calories

The previous fallacy can be summed up as follows: the total amount of calories is not the only parameter that we can change in a diet. Even if we ignore this trick, another one is to assume that changing the energy intake without altering

the composition of the diet only changes the energy intake. The wording of the thought experiment is consistent with the energy paradigm and conveys the idea that if you change the amount of food, you are only changing the energy content of the food and therefore the outcome of the experiment, whatever it may be, would have to be attributed to the change in the calories of the food. But the truth is that increasing the amount of food does not only increase the amount of calories: it also affects many other characteristics of the food and these changes can also be the cause of, hypothetically, obtaining a different result. If you eat more food, for example, you may be changing the amount of sugars or flours that you consume. Does the outcome change because you eat more calories or does it change because you have changed your consumption of sugar and starch? What I mean is that even when weight gain could be the actual outcome in this thought experiment, this outcome may not be determined by the total amount of calories but by another factor that was also changed, for example a greater secretion of a specific hormone in response to an increase of a specific component of the diet and not in response to the total caloric intake. Attributing the result to the calories would be conceptually wrong. The argument is, therefore, clearly deficient as a demonstration of the importance of calories or of the energy balance.

I insist a little on the idea above: if every day I consume an extra pound of food, am I demonstrating that the changes in body weight and body fat are determined by the amount of kilos of food I ingest? I do not think so.

The origin of the argument that I am talking about right now is the energy balance theory, the belief that we can control our body weight counting calories and increasing our physical activity. In this theory, the main mistake, the main nonsense, is to infer causality of a law, the first law of thermodynamics, which is descriptive, never explanatory. And, since that law only talks about energy, it is inferred that our body reacts only to the energy of the food, and that, therefore, the body's response is independent of the composition of the diet. It is a heap of bullshit:

Based on a universal law that only speaks of energy, it has been wrongly deduced that energy is all that matters.

But the scientific evidence leaves no doubt about the falsity of the energy paradigm: our body reacts to many features of the food, not only to the potential energy that can be extracted from it. In addition, the effect depends on the metabolic status of the person, something that a general law of physics can never take into account. Calories never matter because when we talk about calories, we only consider one aspect of reality, the energy. It is a one-dimensional analysis. To speak of calories always means to ignore the composition of the diet. But I am not saying that how much is consumed from each food is not relevant, especially when the composition of the diet is not healthy. Calories and quantity of food are not the same thing.

Guilt must be proved, not made up

An additional problem within the argument is that the hypothetical situation assumes an extreme/forced amount of calories. The argument itself is an extreme situation that implies that there will be a negative effect (something that is not that clear to me, as I said above) and of course the culprit is assumed to be "excess energy" (something that is not clear either). This extreme situation introduces

a forced culprit in the debate, one that is preset and clearly recognizable. But this extreme situation is not the case of interest. What is to be demonstrated is that in a normal situation, in which a person develops obesity inadvertently, not intentionally, throughout decades, the cause of obesity is eating an excess of calories relative to our energy expenditure. In the case of interest, without forcing an extreme situation, the culprit is not so clear.

When we have a normal intake, without forcing anything, does the composition of the diet affect the accumulation of body fat? Fortunately, the scientific evidence is there, for whoever wants to see it⁹.

IF YOU EAT UNHEALTHY FOOD YOU WILL NOT LOSE WEIGHT

Another argument frequently used to defend the Calories In Calories Out paradigm is that calorie restriction is mandatory for body weight loss: certainly eating **more** will not make you lose weight, they say.

This seems like a common sense issue, but it is a fallacious argument. If the only thing you can change in the diet were the amount of food it would be logical to think that the solution to obesity should be eating less food, not more. But the amount of calories is not the only thing that can be altered in our food. You can change the composition, the quality of what you eat: of course eating **low quality** food will not make you lose weight.

EVEN IF YOU EAT REAL FOOD, IF YOUR INTAKE IS HIGHER THAN YOUR EXPENDITURE, YOU WILL GAIN WEIGHT

This is another argument that is usually used to try to justify that body weight management is all about calories. It is indeed a fallacy that plays with words and it is also a sort of *contradictio in terminis*.

It is easy to see that the argument is nothing but empty talk, absolutely void of useful knowledge. According to the advocates of the energy balance theory, if your energy intake exceeds your energy expenditure you will gain body fat, but according to their ideas gaining weight and having an energy intake greater than the energy expenditure are two ways of saying the same thing. The surreal contribution to human knowledge is that “*even if you eat well, if you get fatter, you get fatter*”. To say nothing useful is used to justify that everything is a question of quantity and calories. It is the definition of sophistry.

“*If you gain weight, you gain weight...*” You can say whatever you want at the beginning of that sentence, and you are never going to get any valid knowledge from it: “*even after two months of starvation, drinking nothing but plain water, if your energy intake is higher than your energy expenditure, you will gain weight*”. True or false? Is it false that if you get fatter, you are going to get fatter? Simply fallacious.

Another example of a sentence that contains a similar fallacy: “*When you jump from the top of a building, if the force upward is greater than the gravity force, you are not going to fall*”. “If you do not fall, you do not fall...” do you think that there

is any option that you are not going to fall if you jump from the top of a building?
Does the fallacy give us any valid knowledge?

ISOENERGETIC DIETS, DIFFERENT ACCUMULATION OF BODY FAT

Ultimately, the energy balance theory states that calories from food determine the changes in our weight/body fat. Does the scientific evidence confirm that the same amount of calories will produce the same result, regardless of the composition of the diet? No, it does not. In that regard the scientific evidence is compelling: isocaloric diets have been shown repeatedly to produce different effects in the body weight⁹, even when those diets have the same ratio of macronutrients²⁶⁻²⁸. If isocaloric diets produce different results in terms of body fat, it is indisputable that the calories of the food do not determine the changes in our body fat. I insist: indisputable.

We are told that experiments where participants are totally controlled demonstrate that the composition of the diet does not matter and that the only thing that matters is the total amount of calories. This is false. Those controlled experiments, when they have been carried out, have always been of short duration and the differences between dietary groups have been small, logically. But the outcome of these studies shows precisely that the composition of the diet is important²⁹. Moreover, experiments with animals are absolutely controlled and there are plenty of them where isocaloric diets produce very different results in terms of accumulation of body fat⁹.

If we talk about calories, we are despising the relevance of the composition of the diet.

On the other hand, saying that with our physical activity we burn calories is simplistic thinking. Our body is much more complex than that^{7,30,31}. But I am not saying that physical exercise does not play a role in managing our body weight, for example in combination with a diet low in carbohydrates (see section “What does the scientific evidence say?” on page 38) or by improving our metabolic flexibility⁵.

CAP. 3 THE PHYSIOLOGY OF GETTING FAT

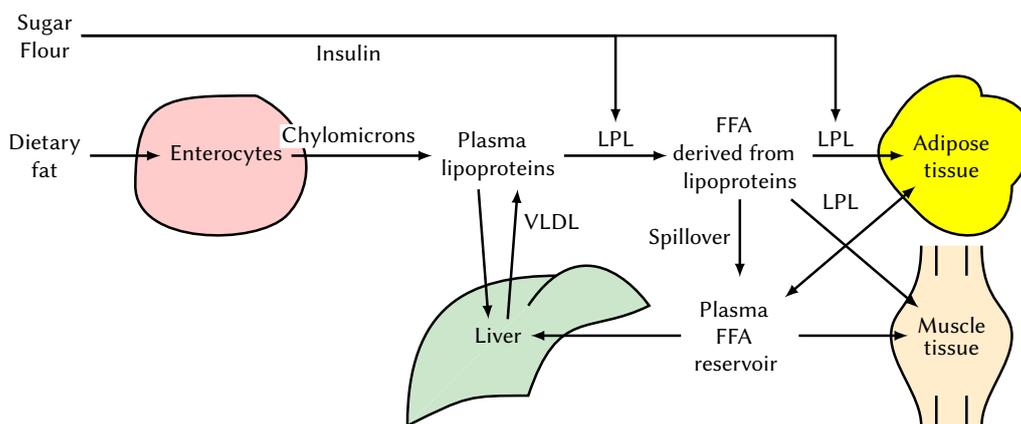
What our body stores in our adipocytes when we get fat are triglycerides. Under normal conditions that fat comes from dietary fat, but our body is not as simple as that you get fat if you eat too much fat nor it is, of course, as simple as that we get fat when we ingest too many calories. The sequence of events is as follows: in the first hours after a meal a fraction of the dietary fat is stored in the adipose tissue, a process that is regulated by hormones. For example, the higher the insulin levels are, the greater the fraction of dietary fat that is initially stored in the adipose tissue. The rest of the dietary fat remains in plasma and there are physiologic mechanisms that allow other organs/tissues to regulate those non-esterified plasma fatty acids so they do not accumulate in blood. It is as simple as dissipating as heat any excess energy without producing ATP molecules that are not needed at that time. What I mean is that the sequence of events may be that the adipose tissue captures in the first place a fraction of the dietary fat that is a function of the hormonal environment and the presence of substrates (triglycerides encapsulated in lipoproteins and non-esterified fatty acids linked to albumin) and that, subsequently, other organs of our body have the capacity to dispose of the dietary fat that has not been captured by the adipose tissue. This could be especially accurate for healthy and physically active individuals.

HOW DIETARY FAT ENDS UP INSIDE OF THE ADIPOCYTES

The behavior of our organs and tissues changes as time passes after a meal. We can make a distinction between the postprandial period, *i.e.* the 4–5 hours following a meal, and the postabsorptive period, which would be the period that runs from the end of the postprandial period until the next meal.

These digestion products are absorbed in the intestine by the enterocytes. Enterocytes synthesize and segregate chylomicrons, a kind of large lipoproteins that transport dietary fat. Fat is not segregated in chylomicrons as soon as it is absorbed. Instead of that, the enterocytes store part of the fat inside of the enterocyte itself (perhaps the newly ingested, while they ship in chylomicrons the triglycerides they already had in storage). The mechanisms that regulate the generation of chylomicrons are not well understood, but they seem to be related to the ingestion: dietary fat from a certain food is shipped in chylomicrons also after the ingestion of the following meals, as part of a first quick phase of increase in the concentration of chylomicrons.

Dietary fat circulates in chylomicrons in the blood, where we also find fat shipped in VLDL (lipoproteins from the liver) and free fatty acids (linked to albumin) that come from the white adipose tissue (intracellular lipolysis) or from lipoproteins (in what is called “spillover”): they are fatty acids that are extracted from lipoproteins by the LPL (lipoprotein lipase) but that do not enter the tissues and therefore remain in plasma. A large part of the chylomicrons’ load, perhaps half of it, ends up in the blood plasma. Schematically:

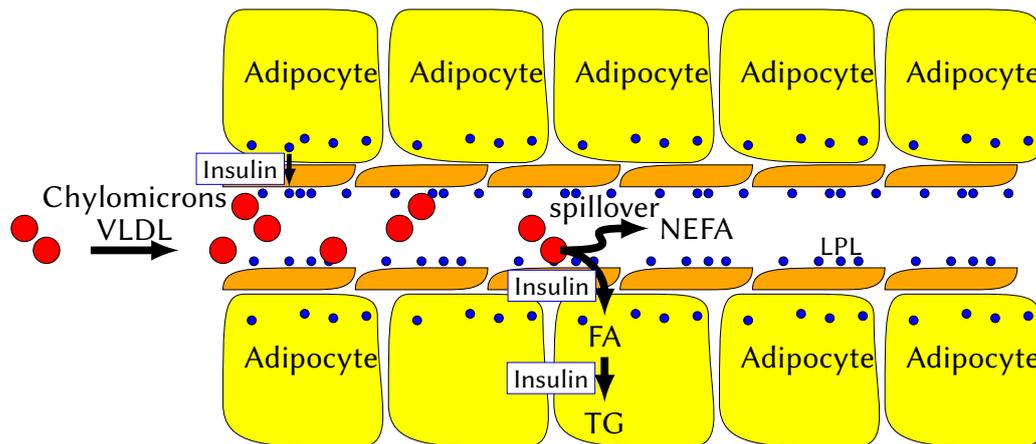


I detail below this line the sequence of events. As a bibliographic reference on this topic, I would recommend an article³² and a blog entry³³.

PHASE 1: POSTPRANDIAL PERIOD

A few minutes after a meal chylomicrons appear in the circulation coming from the enterocytes. The rise of the insulin levels causes the relocation of lipoprotein lipase (LPL), which is synthesized by adipocytes themselves, from the inside of the cells to the capillary endothelium, the surface where lipoproteins will be trapped

in order to remove their content (as if they were freighters that dock at a port to download their content). LPL performs that action and insulin also facilitates the transport of fatty acids extracted from the lipoproteins into the adipocytes (there are transport proteins in the cell membrane that are activated in the presence of insulin). Insulin facilitates also the conversion (esterification), once inside of the adipocytes, of these free fatty acids (FA) into triglycerides (TG) that are stored in the fat cells.



Insulin not only promotes the absorption of dietary fat by the adipose tissue, it also blocks the release (cellular lipolysis) of fatty acids for several hours after a meal.

Although the adipocytes can get fatty acids from chylomicrons, VLDL and plasmatic free fatty acids, the main source of the absorbed fatty acids are chylomicrons.

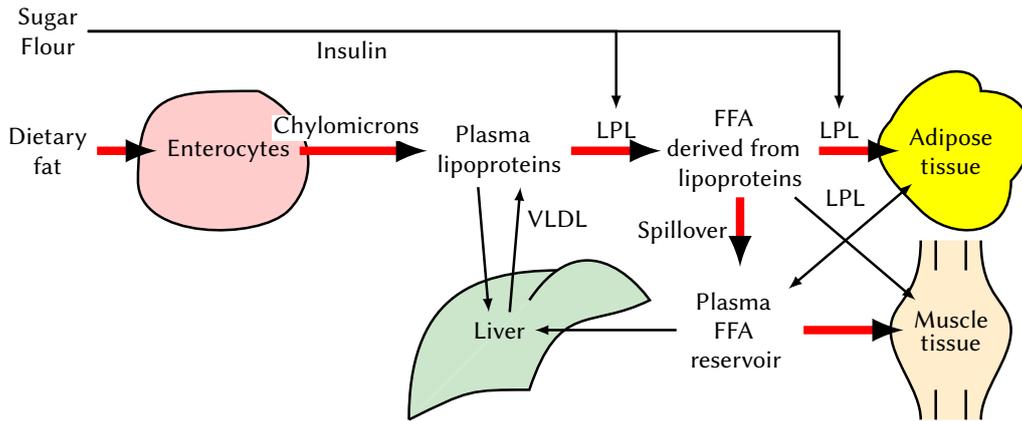
As I have already said, a large part of those fatty acids that are extracted from lipoproteins do not enter into the adipose tissue but are released into the blood and add up to the plasmatic free fatty acids reservoir. Insulin increases the proportion of LPL-derived fatty acids taken up by the tissue and, therefore, reduces the proportion that goes to plasma (spillover).

Over time, insulin concentration is reduced and the adipose tissue is less inclined to capture fatty acids and, therefore, a bigger proportion of the fatty acids that are extracted from the lipoproteins ends up in plasma.

At the end of this phase the muscle tissue increases its capture of fatty acids, allegedly as a result of the rise of the concentration of fatty acids in plasma. The rate of removal of plasma NEFA is, under most conditions, fairly closely proportional to their plasma concentration³². If there is an excess of fatty acids, their potential energy can be dissipated as heat without at the same time generating ATP thanks to the uncoupling proteins present in muscle tissue.

In short, in this phase insulin promotes the storage of food as body fat (at the same time it suppresses the release of fat from adipocytes into plasma).

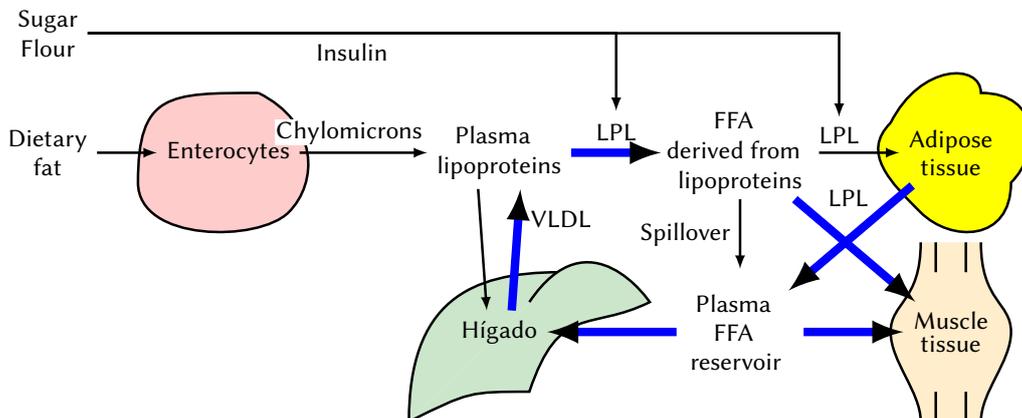
I will not be FAT again



PHASE 2: POSTABSORPTIVE PERIOD

When insulin levels go down, the adipose tissue releases fatty acids and muscle LPL is up-regulated to capture fatty acids contained in lipoproteins (the opposite from adipose tissue). Curiously there is no spillover in the muscle LPL action. However, the activation of the muscle LPL does not change that much throughout a day. Fatty acids can enter the muscle tissue both by passive (extracellular-intracellular concentration gradient) and active (transport proteins that move to the cell membrane and are activated) mechanisms. Muscle is a net consumer of fatty acids: it is believed that there is never a net release of fatty acids to plasma.

When insulin levels go down, the liver increases its secretion of VLDL.



A brief summary of both phases, as a table (simplified, because in reality there are several phases in the postprandial period):

	Insulin	Incoming lipoproteins	Tissues that use lipoproteins	Plasma FFA source	Tissues that use FFA
Postprandial	High	Chylomicrons (enterocytes)	Adipose	Lipoproteins (spillover)	Adipose Muscle
Postabsorptive	Low	VLDL (Liver)	Muscle	Adipose tissue (lipolysis)	Muscle

CONCLUSIONS

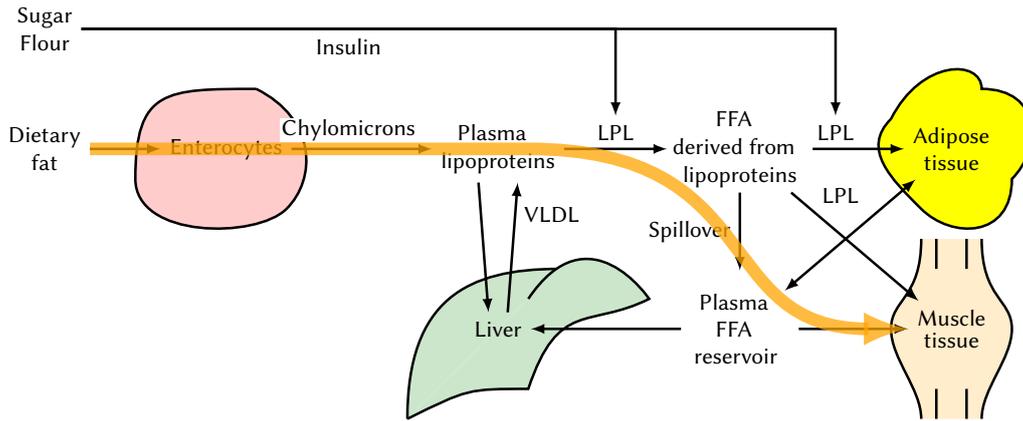
As we have seen in this chapter, after a meal the first event that takes place is the capture of dietary fat by the adipose tissue, a process determined by the hormonal changes induced by the food and the presence of substrates (free fatty acids and triglycerides in lipoproteins) in blood. The part of the dietary fat that is not stored will be dealt with by the rest of organs/tissues, so it can be speculated that this part will not be as fattening as if it had directly entered the adipose tissue in the postprandial period. Note the timeline: the energy balance theory uses fallacies to make us believe that the adipose tissue is the last one to act, storing the leftovers after the rest of organs/tissues have fulfilled their needs. However, it is the adipose tissue who acts in the first place and its action has nothing to do with the energy balance equation, but with the physiologic signals it receives, which are both hormonal and the presence of substrates.

It is important to highlight that the rate of oxidation of fatty acids in the muscle is not constant; it is adaptative and it is influenced by the actions of the adipose tissue: if necessary the muscle can get rid of the fatty acids that the adipose tissue has not captured in the postprandial period, preventing in this way their accumulation in plasma. There are organs/tissues that have the capacity to eliminate (e.g. dissipating their potential energy as heat) those fatty acids that accumulate in excess in the blood, not because those fatty acids are required to meet their energy needs, but rather the opposite: they are accumulating and there is no need for more ATP. In this sense the muscle has a variable and adaptative efficiency. Is not a “blatancy” that we get fat because we eat “too much”: the actual cause may be eating “not-food” (which would directly promote fat storage in the adipose tissue) or lacking the physiologic flexibility required to properly manage the plasmatic fatty acids levels (may be the consequence of a wrong diet but perhaps due to insufficient or inadequate physical activity).

Based on the knowledge of the physiologic processes involved in the accumulation of body fat, it is reasonable to consider that the quality of what we eat and our health status, stress, rest, starvation, etc. are what determine whether we are going to gain or lose body fat. Our desire to maintain our body weight would lead us to the search of ways to create the right conditions so that our adipose tissue is not inclined to store fat and our organs/tissues can properly manage the dietary fat that the adipose tissue does not capture in the postprandial period. To pay attention to the quality of food that we eat would be, as I said, a logic measure in order to avoid weight gain. Being physically active would be one of the obvious ways to keep the physiologic flexibility that would allow our body to successfully manage the variability in the amount of food.

In graphical format, what seems convenient would be, on the one hand, to eat food, not edible food-like products, so they are not easily captured by the adipose tissue and, on the other hand, to maintain a good physical condition thanks to regular physical exercise. The latter would allow a healthy management of the fat in our blood

I will not be FAT again



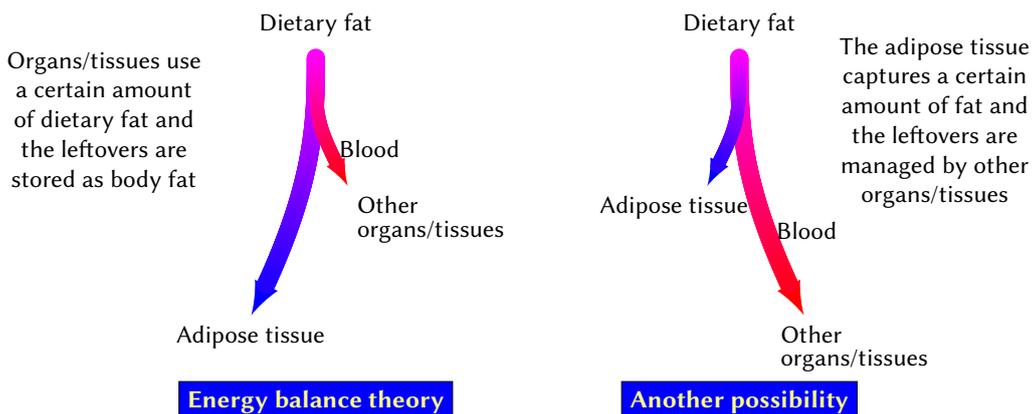
CONCLUSIONS ON CAUSATION

The timeline of the physiologic events that take place since we eat until a few hours after the last meal, leads to the logical conclusion that the reason why we get fat is not necessarily the one fraudulently established by the energy balance theory. In essence, this theory assumes that, for a given level of physical activity, muscle and brown adipose tissue have a constant energy expenditure, so “over-consumption” causes that the food that is not used by these tissues, what is left when all the organs/tissues have consumed what they need, has to be stored in the adipose tissue. This idea is usually expressed as “if your body spends 1800 and you eat 2000, of course you’re going to get fat”.

Tissues spend what they have to spend and the rest of the fat is stored in the adipose tissue

This idea is not incompatible with the laws of physics, but it does not legitimately derive from these laws. Or in other words, the fraud of the energy balance theory does not lie in the maths, it lies in a behavior of the body that

1. is made up from word games,
2. is promoted as obvious, and
3. is said to derive from universal laws.



The “energy” paradigm —fraudulently— concludes that controlling our body weight is ultimately a matter of calories. According to this paradigm, the measures we can take to control our body weight are limited to increase/reduce our energy intake or increase/reduce our energy expenditure by changing our physical activity. Nevertheless, the real behavior of our body may have nothing to do with this hypothesis.

In short, the energy balance pseudoscience uses puns that include tautologies, fallacies and terms with double or ambiguous definitions in order to lead to the fraudulent conclusion that the accumulation of body fat is determined by the difference between what is consumed and what is spent. But this idea does not legitimately derive from the laws of physics, as I have tried to explain in the first chapters of this book. Other behaviors of our body are possible. In that sense, a possibility that escapes from the trap of the energy balance theory is that the energy that is spent may be determined by the difference between the caloric intake and what is stored as body fat. That idea does not violate any universal law and might have a physiologic basis. What is really important here is not to decide if this alternative explanation is wrong or right, but to realize that it is as possible as the energy balance theory, making clear that this latter theory is nothing other than a fraud. The actual behavior of the human body can never be inferred from a general law of physics.

CAP. 4 WHY ARE WE SO FAT?

If it is not caused by excess food, if it is not caused by a sedentary lifestyle, if it is not caused by calories, what makes us fat?

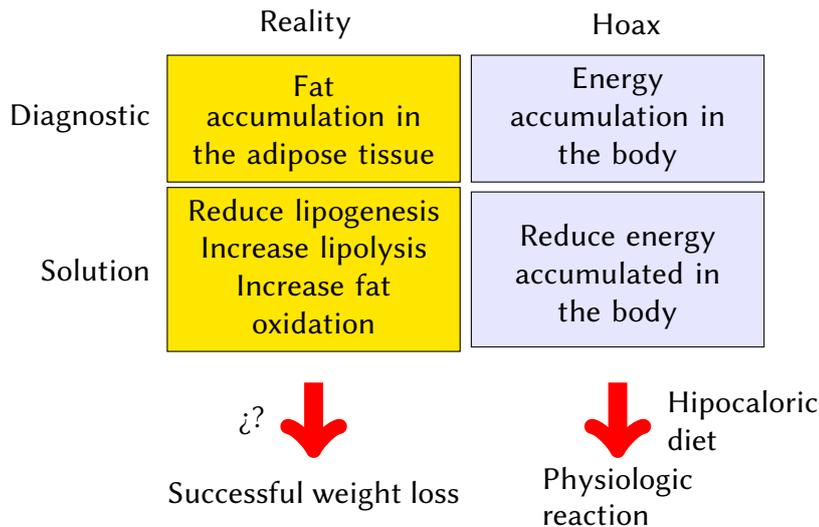
What causes all growths in an animal? What makes a child grow taller? What makes an athlete increase her muscle mass? What makes a tumor grow? The answer comes always from biology, never from the energy balance equation. Saying that in a growth more energy is ingested than spent is descriptive, and in no case, none of them, leads to the real cause of the growth.

OBESITY IS NOT AN ENERGY PROBLEM

Obesity is defined as a condition where there is an excess of fat accumulation in the body. Body fat accumulates in adipocytes, also known as fat cells.

We do not store energy in the body, we store fat in the form of triglycerides in our fat cells. And fat is not the same as energy. This point is important.

If the question is why are we fat, what we are asking is why our adipocytes store excess fat. It seems sensible to think that the changes in the hormonal milieu that bathes the cells are what really cause the excessive accumulation of fat.



PROXIMATE CAUSE AND ULTIMATE CAUSE OF OBESITY

Although I have already explained that the energy balance theory contributes nothing more to the nutritional science than an absolutely useless and harmful tautology (page 9), I am going to insist a little more, because, well... analogies are clarifying. Imagine that the pilot of a light aircraft makes a mistake and the airplane loses altitude and eventually crashes. The proximate cause of the accident would be that the gravitational force has been bigger than the lift force of the aircraft. But this statement tells us nothing about the real cause of the accident. When a plane crashes, it is always going to be true that the downward force has been greater than the upward force, but you cannot say that the plane crashed for that reason.

Similarly, if a student fails a test, the proximate cause would be that he achieved an insufficient number of correct answers, but that tells us nothing about the real cause why the test was failed. When someone fails a test, the number of correct answers is always insufficient, but it cannot be said that the test was failed for that reason.

In the same way, when the adipose tissue grows in size, more energy enters the tissue than comes out, but that tell us nothing about the real cause why it grew. When the adipose tissue is getting bigger, always more energy enters the adipose tissue than comes out, but we cannot say that it has gained fat for that reason¹².

THE HORMONAL HYPOTHESIS

When confronted with a case of an abnormal growth in a child, I presume that the physician would look for hormonal causes, some kind of deregulation, for example in the growth hormone. But biology is despised when talking about obesity and prejudices enter the scene: if someone is overweight, the causes are gluttony and sloth. And the remedy is to be as virtuous as thin people are, who apparently do not eat too much and do enough physical exercise.

According to those prejudices, cause and solution, defect and virtue, are psychological and behavioral issues. As I have said, these prejudices are defended with pseudoscience allegedly derived from the laws of thermodynamics.

The response of adipocytes to the hormonal environment is complex, and there is not a single hormone involved. But among them insulin has a prominent role^{34,35}. Insulin is a hormone secreted by the β -cells of the pancreas, which respond to blood glucose levels³⁶. In particular, foods that contain carbohydrates are the ones that elicit a bigger insulin secretion, especially the carbohydrates in powder or liquid form (saccharose and flours/cereals). Insulin secreted by the pancreas reaches to the liver thanks to the hepatic portal vein, and the liver in response to the concentration of insulin regulates the amount of glucose released to the blood. The liver also removes part of the insulin from the bloodstream, and, therefore, the insulin concentration in the hepatic portal vein is much higher than its concentration in the peripheral circulation.

Insulin, therefore, has as a function to inform the liver of the blood glucose level, in order to keep it in very strict limits. Too much glucose in the blood is toxic for all of us.

But insulin also modulates the amount of fatty acids entering and leaving the adipocytes. Insulin promotes the capture of fatty acids, which are stored in the form of triglycerides in adipocytes, and it also decreases their release rate. In short, insulin promotes net fat storage in fat cells³⁷.

In general, we can say that during the day we store fat in our adipocytes, while at night fat is released³⁸. The explanation could be that during several hours after each meal insulin levels are elevated, and therefore there is a net fat storage in the body. At night, while we sleep, insulin levels are very low and there is a net release of fat.

For example, an inappropriate night's rest could make us fat, simply changing how our body reacts to insulin³⁹. Due to an insufficient rest the liver may not properly detect insulin (it would be insulin-resistant), and this may lead to higher and longer lasting levels of glucose and insulin. And that could make us fat, without having changed our energy intake and without having changed our exercise routines.

In the same way, situations of stress can produce elevated levels of cortisol, a hormone that, among other things, modulates the effects of insulin. This could result in an increase in body fat, a stall in the weight loss or, in some cases, it could produce weight loss⁴⁰.

Neither bad sleep nor stress have calories, but they can, however, affect our fat tissue. Other substances present in the environment or in the foods we eat can have similar effects on body fat, despite not having calories^{28,41}.

The physiologic status of the person matters too. The same foods, with exactly

I will not be FAT again

the same calories, can have a more pronounced hormonal response in one person than in others, and also differently make them store fat⁴².

To think that insulin is the only hormone involved in obesity is simplistic, but to talk about hormones is infinitely more logical and scientific than the “magical” energy balance theory⁴³.

WHAT CAUSES OBESITY?

When a population consumes its traditional diet, whatever the diet, obesity and diabetes have little prevalence. When all or part of these people adopt the western diet, inevitably they suffer the same diabetes and obesity problems suffered by any other population that follows the same diet⁴⁴⁻⁴⁶. I see no reason to blame to gluttony and laziness what has a much more reasonable explanation: we are not eating what as humans we are prepared to eat. There may be a specific component of the diet that is responsible for the negative effects on health, or the effect may be caused by a combination of several or all of the components. In any case the main suspects, in my opinion, are:

- 1 Soft drinks.
- 2 Acellular carbohydrates (sucrose, and cereal/flour, like bread, pizza, pasta, etc.).
- 3 Products processed with all kinds of non-food substances in their list of ingredients.
- 4 Seed oils (soy, sunflower, etc.)

All the above elements have a place in our diet when we count calories. Talking about calories is not just useless, but directly harmful, because it leads us to wrong decisions about what to eat¹¹.

CAP. 5 WHAT CAN WE DO TO LOSE WEIGHT?

Sadly, no one has yet an answer to that question. We can find dozens of tips on the internet but the reality is that we will not find a method that has been scientifically proved to be useful to lose a significant amount of weight and keep it off in the long term. Some people do manage to lose weight and keep it off, but these people do not confirm the effectiveness, but the ineffectiveness of the method. They are the exception that proves the rule that dieting does not work.

The hypocaloric diets recommended by official bodies stand out among those magical diets with a pseudoscientific origin and whose effectiveness has never been proved,⁴⁷. Very unfortunate.

IS IT POSSIBLE TO LOSE WEIGHT?

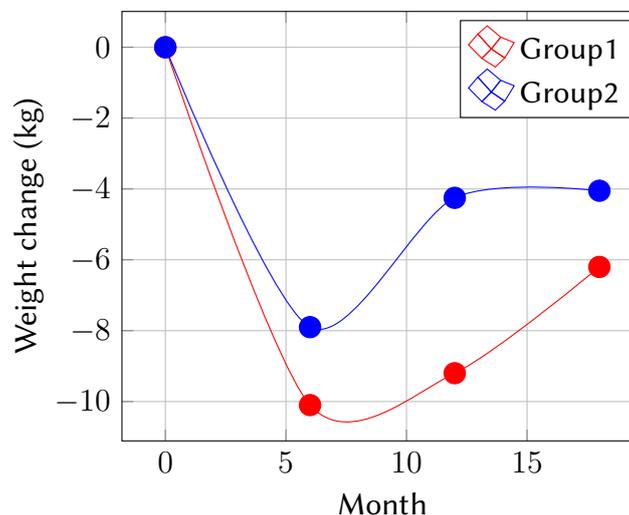
I am not sure of that. Maybe not everyone can lose weight with a diet and exercise plan. I do not think that the current problem of weight gain is a natural process, neither qualitative nor quantitative speaking. It is possible that gaining weight induces changes in our body that are hardly reversible, for example an increase in the total number of fat cells⁴⁸, which make it difficult to reach and maintain a normal weight.

Statistics say that very few people go from obese to lean and maintain the reduced weight in the long term. We are talking about one of every 200 obese men⁷. It is hard to believe that no one has the willpower to follow a diet and lose the excess weight. In my opinion, it is clear that we do not slim down because we do not know how to do it, or because it is not possible to do it. Saying that people do not try hard enough sounds nonsense to me.

LOW-CALORIE DIETS AND THE REBOUND EFFECT

Low-calorie diets are fad diets. Their origin is pseudoscientific, since, logically, a universal law of physics cannot be used to deduce the response of a living being to food restriction. If you believe that mathematics guarantee the success of calorie restriction as a way to lose weight in an animal, you are not thinking straight. What scientific evidence tells us is that with caloric restriction almost nobody manages to keep the reduced weight in the long run^{7,49-51}. Scientific experiments confirm 3 kg lost after 4 years of dieting. 4 kg if you add physical activity to the diet.

Low-calorie diets base their popularity in the fraud of the energy balance theory, but also in a delusion: they produce short-term results. So, yes, they make you lose weight in the short term, but they have another effect: they change your physiology so your body seeks to store fat^{8,52}. With time, weight loss gradually stalls, and you start to regain weight after six months of dieting, more or less. This is known as the rebound effect. The evolution with time of the body weight is quite typical⁵³:



As a corollary, the results of short duration (less than six months) weight loss scientific experiments are useless to evaluate the effectiveness of a weight loss method.

APPETITE CONTROL

Appetite, satiety, portion size, etc. are all terms that belong to the quantity paradigm. Walk the dog when you are hungry, turn off the TV while you eat, eat satiating foods, consume low-calorie products or use smaller dishes are tips that seek to reduce the energy intake. Since low-calorie diets have never been proved to be useful for weight loss, all these tips are, at least, of dubious utility, even if they were shown to be effective to reduce the energy intake.

THE LACK OF ADHERENCE

When talking about weight loss diets, there is a big obstacle: if people do not follow the method, it is impossible to evaluate its actual effectiveness.

CICO defenders argue that low-calorie diets work but people stop too soon to follow the diet. The truth is that in scientific experiments in which people do follow the diet, it has been confirmed that weight loss stalls and weight regain starts after a few months of dieting^{8,54-56}. And the body weight that is regained is composed mainly of body fat^{6,57}.

Probably most of the people stop soon to follow the diet, but there is no guarantee that they would get a better result had they adhered further to the diet. The observation that a dietary program has low adherence does not lead us to the conclusion that low adherence is the cause why low-calorie diets are useless to lose weight.

HUNGER AS A SYMPTOM THAT THE METHOD IS WRONG

If a diet were able to cause the net release of triglycerides from adipocytes and also their subsequent oxidation, *i.e.* its use as an energy source, there would be no reason to expect that we would feel hungry, because there would be no shortage of food⁵⁸. In a nutshell, it can be hypothesized that a weight-loss diet that works should not produce hunger.

On the contrary, low-calorie diets are based on the idea that by forcing hunger our body will respond to that attack releasing and burning body fat. Those diets are not based on what we know about how the human body works, but to combat the sins of gluttony and sloth.

Those who defend the energy balance theory believe that if a diet works, it does so by helping us to eat less. The truth is that if a diet works, you can expect a reduced energy intake due to a lack of appetite. The diet would not work because it reduces the intake, but the intake would be reduced because the diet works.

WHAT DOES THE SCIENTIFIC EVIDENCE TELL US?

Lots of people tell us the secrets for successful weight loss, but the truth is that the evidence of an effective method to lose weight is non-existent. Not a single scientific experiment that used a hypocaloric diet succeeded at avoiding the recovery of the lost weight⁴⁹. After 3 to 4 years weight loss is below 5 kg (and the actual value may be even worse because it is possible that the participants start dieting again before the follow-up period of the studies is completed).

As far as I know, there are two studies, both of them one year long, in which the participants lost around 30 kg of body weight, without signs of a rebound effect^{59,60}. The diets were not hypocaloric, but they focused instead on a reduced carbohydrate intake. It is not a long-term outcome, but the lack of a rebound effect demonstrates that there is something uncommon in these experiments. Maybe the actual cause of the success is a factor other than the reduction of carbohydrates, but I cannot imagine what else could it be. In another study in which we find unusual weight loss outcomes, carbohydrates were also restricted⁶¹. Moreover, in a long-term (three years) study, the increase of the carbohydrate intake was associated with weight regain⁶².

Another interesting result is that, consistently, low-carbohydrate diets produce in scientific experiments more body fat loss than isocaloric diets with more carbohydrates^{29,63-66}. This is so even with identical levels of protein. In general, it seems is that protein helps to preserve muscle mass, while the reduction of carbohydrates helps to lose body fat. The results that I am talking about right now come mostly from short-term experiments.

Diets that restrict carbohydrate almost always give better weight loss results than other diets. Differences between diets are often reduced in the long-term, a fact that could be caused by the lack of adherence to the diets, but it also may be caused by the lack of effectiveness of the diets⁶⁷.

There are two more details that I find significant. They have to do with the idea that a diet that worked to lose weight would, supposedly, reduce hunger, not increase it. A successful diet is supposed to promote the release of fatty acids from the fat cells and use it also as an energy source. In that sense, one of the known characteristics of low-carbohydrate diets is that they reduce (or perhaps I should say “normalize”) hunger, and, consequently, food intake^{65,68,69}. This fact can be interpreted as a sign that body fat is indeed being used as an energy source, or, in other words, that we are losing body fat, which is opposed to low-calorie diets, which cause hunger. The second detail is that when a person is adapted to a diet low in carbohydrates, possibly much more body fat is oxidized during physical exercise than in a person adapted to a diet high in carbohydrates^{69,70}. In my opinion, it is reasonable to infer that carbohydrate restriction unlocks the access to our body fat reserves, allowing us to use it as an energy source. This sounds like losing body fat.

CAP. 6 NUTRITION IS A BIG FARCE

Official health bodies warn us about the dangers of obtaining nutritional advice from non-official sources, but when the messages we receive from those same authorities are critically analyzed, the only possible conclusion is that almost everything they say, almost everything we think we know about nutrition, about cholesterol, saturated fats, whole grains, the food pyramid, balanced diets, energy balance, etc, is baseless.

What happens in the nutrition field is unacceptable for the general population, but especially for people who suffer from diabetes or pre-diabetes, who are currently advised to follow a diet that aggravates their condition and leads to increasingly greater use of therapeutic drugs.

THE BASIS OF THE NUTRITIONAL PYRAMID

We are told that more than half of the food we eat, in terms of calories, has to come from carbohydrates. At the base of the food pyramid we find grains, flour, bread, etc.

But not eating carbohydrates does not generate health problems^{71,72}, nor have we to eat them for our brain's sake⁷³, nor are they required to give us energy, nor anything similar. On the contrary, in controlled experiments their consumption is associated with worsening of cardiovascular risk factors⁷⁴⁻⁸².

SATURATED FAT

There is no reason to avoid the consumption of natural foods high in saturated fat. The recommendations about limiting our intake of saturated fat do not have a solid scientific basis^{80,83-90}.

CHOLESTEROL IN FOOD

In most of us, dietary cholesterol barely changes our blood cholesterol levels⁸⁵. Even assuming as a truth the dogma that having high cholesterol levels is a health risk, the link between dietary cholesterol and blood cholesterol is too weak to use it to promote a limitation in its consumption. Even the American health authorities, which in terms of nutritional science are the lowest of the low, have already removed the limit in the dietary cholesterol intake in the latest version of the Dietary Recommendations for the Americans⁹¹.

RED MEAT

There is no solid scientific evidence to recommend limiting the consumption of red meat^{92,93}.

WHOLE-GRAINS

Whole-grains have a reputation for being healthy. Their promotion is so intense that some people refer to them sarcastically as healthy-whole-grains. I certainly believe whole grains are better for health than refined grains⁹⁴, but that does not mean that it is healthier to consume grains than not to consume them. When diets that include grains are compared with grain-free diets, the result indicates that the consumption of grains, whole or refined, it does not matter, should not be recommended⁹⁵⁻⁹⁹.

DIETS FOR DIABETES

The unfounded fear of saturated fat and dietary cholesterol harms, in my opinion, the health of everybody, but especially of those that suffer from diabetes. The diet that is currently recommended by the official bodies for these people is based on the food group for which they are intolerant, on the pretext of avoiding other foods whose evidence of danger is non-existent.

Scientific evidence is overwhelming in the sense that the first treatment option for anyone with diabetes should be a diet restricted in carbohydrates^{60,62,100-108}, just as common sense tells us. A summary¹⁰¹:

- ① Hyperglycemia is the most salient feature of diabetes. Dietary carbohydrate restriction has the greatest effect on decreasing blood glucose levels
- ② During the epidemics of obesity and type 2 diabetes, caloric increases have been due almost entirely to increased carbohydrates
- ③ Benefits of dietary carbohydrate restriction do not require weight loss
- ④ Although weight loss is not required for benefit, no dietary intervention is better than carbohydrate restriction for weight loss
- ⑤ Adherence to low-carbohydrate diets in people with type 2 diabetes is at least as good as adherence to any other dietary interventions and is frequently significantly better.
- ⑥ Replacement of carbohydrate with protein is generally beneficial
- ⑦ Dietary total and saturated fat do not correlate with risk for cardiovascular disease
- ⑧ Plasma saturated fatty acids are controlled by dietary carbohydrate more than by dietary lipids
- ⑨ The best predictor of microvascular and, to a lesser extent, macrovascular complications in patients with type 2 diabetes, is glycemic control (HbA1c)
- ⑩ Dietary carbohydrate restriction is the most effective method (other than starvation) for reducing serum TGs and increasing high-density lipoprotein
- ⑪ Patients with type 2 diabetes on carbohydrate-restricted diets reduce and frequently eliminate medication. People with type 1 usually require lower insulin
- ⑫ Intensive glucose lowering by dietary carbohydrate restriction has no side effects comparable to the effects of intensive pharmacologic treatment

Note that the risk of hypoglycemia is associated with the use of diabetes drugs¹⁰⁹. By following the current guidelines for the treatment of diabetes, what the patient should expect is the progressive need for higher doses of the drugs¹¹⁰.

CAP. 7 WHAT TO EAT

I do not want to tell anyone what to eat, nor designing a meal plan fits my way of understanding a healthy diet. But I do want to make a few considerations for philosophical or motivational reasons.

Food is only real food when it comes from farms, fields, forests, rivers, or oceans. If it does not come from nature, it is not food, but only *food-like* stuff whose health effects should be under suspicion. If a product only exists because processed natural products were mixed in a factory, changing the properties of those source products, the end product is not food: it is not-food.

What to eat? Food. As simple as that. Everyone is different and you may need or resist a —perhaps very different— different diet, but I find it hard to believe that a diet based on industrial products can be a better choice for you than eating real food.

Please note that “diet” is the total of food consumed by a person. When I use the term diet, I am not referring exclusively to those who pay attention to what they eat: everybody follows a diet, whether we like it or not. On the other hand “being on a diet” means that we are making a temporary change in our habits while pursuing a goal. If the change is permanent I would not say that we are on a diet, but that we have changed our diet.

WE HAVE A HEALTH PROBLEM AND TO SOLVE IT WE MUST CHANGE SOMETHING

My interest in nutrition was triggered by my health and obesity problems. When someone cares about their diet, the reason is usually that what they were doing was not working for them. There is a problem, and if nothing changes, nothing changes.

The “food pyramid”, “everything in moderation”, “to control your calories” or “to follow a balanced diet” define the current paradigm, the one that is making us fat and sick.

Insanity is doing the same thing
over and over again and expecting
different results

Albert Einstein

In my experience, some people will change their diet as they see fit in order to fix a weight or health problem. But others will prefer to be sick or fat rather than stop eating bread and drinking beer. That is just how it is. The latter are, of course, partly responsible for their situation, because they are fooling themselves, and, mainly, because they do not want to listen when someone tells them that eating well is not about calories. But they are not the only culprits: the message we receive is not favorable to change. We are told that we are not allowed to decide for ourselves what we want to eat and what not. We are told that some options are socially unacceptable or that it is not sustainable in the long term to give up the pleasure of food. This kind of thoughts, often created by the food industry, are a self-fulfilling prophecy: they encourage people to stick to their bad habits, instead of encouraging a shift to healthier habits. If you wanted to quit smoking, would it help you the message that since all your friends smoke, quitting will only serve to marginalize you socially and at the end you will smoke again because that situation is unsustainable? Would it help the message that there is no reason to not enjoy cigarettes, that you can smoke in moderation?

Tobacco does not belong to our diet as human beings and it has detrimental effects on our health. Even neglecting the addiction issue, does it make sense to you to say that the solution to smoking is to smoke in moderation or that its negative effects exist only if you smoke “too much”? For sure, to quit smoking has drawbacks —socially or because you stop doing something you like to do—, but, what is the real agenda of those who highlight those disadvantages or tell you about our right to enjoy tobacco? What is the actual agenda of those who say that a diet which denies the “enjoyment of food” is not sustainable in the long term?

HEALTHY EATING IS NOT A FAILURE

We are animals. But, unfortunately, animals smart enough to create food that is tasty, cheap and has long shelf-life, and stupid enough to eat those foods. It is

an aberration. We should not have changed what we eat. At least, we should not have changed what we eat without giving it a serious thought.

Is a cake part of our traditional food as the animals that we are? Is it a doughnut? Is it a loaf of bread? What about breakfast cereals?

Not eating these not-foods is not a sacrifice, because they should have never been included in our diet. Is someone who does not smoke doing a sacrifice? No, they are not giving up anything, because smoking is not something you expect from an animal, no matter how much a person wants to smoke.

I do not keep my way of eating a secret. I always eat as I think I should, and, if necessary, I do not eat at all. When that happens, it is not that hard to bear with, as fasting does not usually cause me an excessive hunger. I was lucky enough to lose the extra weight and keep it off in the long term. When you have been obese, it is easier to deal with the comments that arise because of your food choices. I guess that everybody understands I have good reasons for caring about what I eat.

LET US FORGET ABOUT MACRONUTRIENTS

Why do dietitians talk of macronutrients? Do I need to know the chemical composition of a food, to know if it is healthy?

I insist on what I said at the beginning of the present chapter: food comes from farms, fields, forest, rivers, or oceans. If a product is the result of a processing that changes its properties, in that case that product is not food. I cannot see how macronutrients are relevant to know if a product is healthy.

My diet is low in processed products: I avoid grains, added sugars, seed oils and products with a long list of ingredients. Is my diet also low in carbohydrates? Yes, it is, because I avoid products like potato or fruits with a high sugar content. But that is a personal choice based on my medical history: I lost my extra weight by restricting carbohydrates and this is my bet in order to avoid having weight problems again. But my recommendation for my family is to eat food and to ignore macronutrients.

An edible product is not “human food” just because it is low-carb. Real foods such as cucumber, tomato or pepper, etc. are not unhealthy just because they have carbohydrates. The effect of these foods on our body has nothing to do with the one from grains/flours. Only when you suffer from diabetes or pre-diabetes it seems advisable to pay attention to the sugar content of real food, because those sugars can be unhealthy for a person who is intolerant to carbs. In such a case, it could be sensible to only eat fruit that is low in sugar¹¹¹ and restrict its consumption.

ARE MY IDEAS EXTREME?

I suppose some people can view these ideas as extreme, but eating real food is what is natural: it is to eat what the human being has been eating for millions of years and it is eating the foods our bodies can handle. Is this extreme?

Is it extreme not to eat all those products that have been artificially introduced in our lives, and which, in my opinion, are the main suspects of real and widespread health problems?

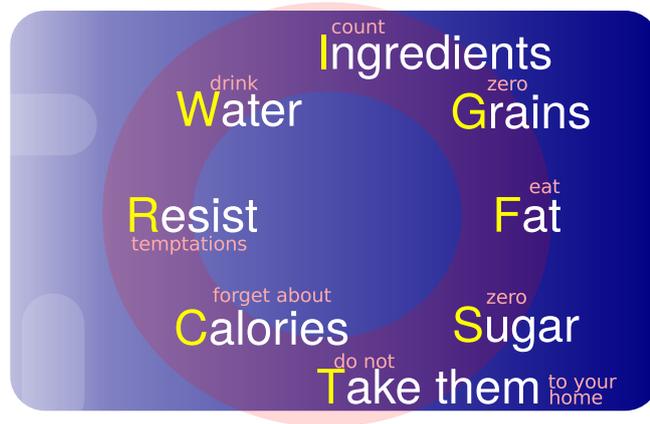
I will not be FAT again

I will not sacrifice my health to fulfill a desire to consume edible products that are not real food. I will not make up false reasons or fraudulent theories to ease my mind and allow myself to consume these products. I do not need them. And if I thought that I needed them, that would worry me.

Vegetables (lettuce, cucumber, tomato, pepper, eggplant, etc.), meat, fish, eggs, fruit and nuts, fermented dairy (yogurt, kefir) without sweeteners, cheese and ham. And water, tea, or coffee (without sweetener) to drink. Can you say this is an extreme or dangerous food list?

For a healthy person and in the context of a healthy diet, I do not think that rice, potatoes or legumes are unhealthy. Dark chocolate (cocoa 85% or higher), even if it has some added sugar, can also be acceptable. It is not part of my diet right now, although it was in the past.

I have a look at the list of ingredients of the product, and if what I see looks like food, then I can eat it. For example, I do not eat milk cream when it has carrageenan as an additive. Are carrageenans something I want to consume when I buy cream? Are they food in their natural state? Another example: I eat chistorra, a typical spanish sausage made from pork, salt, paprika and garlic.



YOU CANNOT COMPENSATE FOR EATING JUNK FOOD

“But on weekends I can drink a beer, right?”.

“Only half sandwich, please, that I am on a diet”.

“I don’t want to lose weight if I am not allowed to drink alcohol when I go out with my friends”.

“I stopped eating bread, but then I started eating it again and I have not lost a single pound. I have to stop eating bread, this time for real”.

“Quitting bread [to avoid having to take medication]? I need time to think”.

All those above are real comments that I got when talking about nutrition with people who want to lose weight. Three of them should lose weight for health reasons. My view is that when someone, before making any real changes in their way of eating, all they want to know is how to cheat the diet, they are not going to

get any results. For me this a symptom that their priority system is not adequate to adhere to healthy habits in the long term. These people did not change their habits and they reached none of their weight loss goals. Maybe they would have neither reached them in case they made a real change, but it is clear to me that when someone makes such comments, the talk will fall on deaf ears.

It is important to remark how harmful the energy paradigm is: *“If you cheat, you can compensate by choosing a salad as your next meal”*. If overeating were the actual cause of obesity, that would make sense. But we already know that it is a misconception. When you eat unhealthy food, choosing real food for the rest of the day does not compensate for this bad decision. Not smoking in the afternoon does not compensate for smoking in the morning. This kind of thinking denotes that we have not actually made any change: it was that kind of thinking what made us fat in the first place. We already knew that every edible product could be part of our diet as long as our energy requirements were not exceeded. We are still inside of the paradigm that made us fat. The energy balance is not only a fraud as a theory, it is harmful for our health¹¹.

WE NEED A PLAN JUST FOR TODAY

To make decisions for the rest of our life can be overwhelming. But that is not real. If I say that I am not going to smoke a cigarette or drink alcohol nor Coke for the rest of my life, perhaps it sounds like a difficult task. However, I am absolutely sure that I can do that. And it will be easy. There is no obstacle.

A technique to prevent being overwhelmed is to make goals only for the short term: *“today I eat healthy”*. And tomorrow is another day. In my experience, the view I had before I changed my way of eating was wrong: after a couple of weeks, all those products that I thought were essential in my life, that I thought gave me happiness, stopped being interesting. And when you are lucky to get good results, to feel OK and to enjoy the new way of eating, then you have no reason to look back. Except maybe to ask yourself why did not you make the decision much earlier.

I AM NOT A GURU

As I said at the beginning of the present chapter, I do not want to tell anyone what to eat. I share my point of view, which, in a nutshell, is that we have been deceived into thinking that the problem of obesity comes from a caloric excess, energy expenditure, portion sizes, appetite control and lack of willpower. But this is nonsense. These ideas lack scientific basis and while the energy paradigm stands most of us will not find a solution to our weight problem. Obesity is not about how much food you eat, but about including in our diet edible products that are not real food.

In some ways my ideas about nutrition are like stealing a candy from an obese child that suffers from tooth decay. He is used to eat candies and cannot understand the reasons why he should quit consuming them. Perhaps if the child never ate candy, he would not think that he is making a sacrifice. But I am not calling anyone childish or capricious, because I was that child who used to do things

I will not be **FAT** again

that he thought were reasonable. But they were not and they were negatively affecting my health.

CAP. 8 BREAD

Bread —or grains/flours— deserve special attention. It is hard to believe, but bread is a deal breaker: some people would rather be fat or ill than quit eating bread. And it is not just a saying. The grain industry tells us that food addiction is a myth, but it is clear to me that something special happens when you eat bread, something that eggs, meat or spinach do not produce. If for health reasons you need to quit eggs, you do. On the contrary, if for health reasons you are advised to quit bread, you need time to think about that.

Is bread unhealthy?

On the one hand, the nutritional value of grain flours is rather poor, and, on the other hand, there is scientific evidence that suggests that we should distrust their effects on our health and body weight. Avoiding grains is not irrational, and, although we can hear otherwise, gluten is not just a common protein, nor it is a health risk exclusively for those who suffer from celiac disease.

THE ARRIVAL OF FLOUR TO OUR DIET

The adoption of agriculture, and, therefore, the mass consumption of grains, is relatively new to our species, from around 10,000 years ago¹¹². Grains have not been part of the traditional diet of human beings during the greater part of our existence, but it is a relatively recent fad. Flour consumption is promoted because of comfort, social and economic reason, but it is debatable if there are nutritional and health reasons to consume it. Interestingly, when 10,000 years ago agriculture was embraced, virtually always our health paid a price¹¹³. We should not take for granted that grain flours are food for human beings nor that avoiding them is a mistake.

Bread, pasta and pizza are created from the same product, wheat flour, a product which is obtained by mechanical procedures that dramatically alter the properties of the raw materials used, wheat seeds. Seeds in their natural state are characterized by not being digestible by birds or by mammals. When they are swallowed, they just enjoy a free ride. To be indigestible is an evolutionary advantage for a plant seed¹¹⁴.

REASONS TO BE WARY

Grain flours are under suspicion for several reasons, among which I highlight:

- ① high glycemic load,
- ② high-glycemic index, and
- ③ gluten

Maybe the effect on health of grain flours has worsened in recent decades. The use of modern windmills that generate ever-finer grain flours is a possible reason why the effect of bread may have evolved from bad to worse with time^{44,115–117}. The finer the flour, presumably the quicker it will be digested and absorbed, and the higher glucose and insulin will rise in blood.

It has also been suggested that the abandonment of the traditional manufacturing processes of bread, those where the fermentation process was long, and the adoption of a shorter process, with an incomplete fermentation and a different sourdough, has contributed to increase intolerance to bread^{118,119}. On the other hand, the modern technique of deamidation of gluten in industrial products, in order to improve, among other things, its solubility, has also been shown to cause adverse reactions in humans aside from wheat allergy¹²⁰.

Another factor to consider is that the wheat plant we have today is not the same as the wheat plant from thousands of years ago. The grain industry denies that wheat has been genetically modified, but it is a fallacious argument, since no one says that genetic engineering was used: changes were made by means other than genetic engineering. The fact is that Norman Borlaug received the Nobel Prize in 1970 for his role in the Green Revolution and the creation of a more productive variety of wheat: semi-dwarf wheat. They say that wheat has not changed but the person responsible for that change was honored with a Nobel

Prize, for changing it. A different question is whether this new wheat is worse than ancient varieties. But some scientific evidence suggests it is^{121,122}.

THE LURE OF THE WHOLE-GRAINS

It is often argued that whole-grains have a healthier glycemic response than refined grains, and that, therefore, they are better for human consumption. But it is not that clear that the glycemic responses are that different¹¹⁵. It is also said that whole-grains have more fiber or have more nutrients.

The grain industry wants to move the goalposts in the debate on the effects of grains on health, trying to focus on whether whole-grains are better or worse than refined flours¹²³. As a result of this deception, you must be careful when reading scientific findings concerning the consumption of flour, because when headlines claim that wheat flour has a protective or beneficial effect what this really means is “when whole-grains are compared to refined flours”. Whole-grains may be better for our health than refined flour, but that does not mean that any of them is healthy. Those results are relative between them. The grain industry seeks to divert our attention from the real question:

Is a grain-free diet better healthier than a diet that includes grains?

Randomized controlled experiments with the paleo diet⁹⁵⁻⁹⁷ or with low-carb diets⁹⁸ seem to support an affirmative answer to that question.

GLUTEN

“Non-celiac gluten sensitivity” (NCGS) makes reference to health problems allegedly caused by gluten in people who are neither celiac nor have an allergy to wheat¹²⁴. Even the grain industry admits that a gluten-free diet may be beneficial for “*gastrointestinal and/or systemic symptoms in individuals with systemic lupus erythematosus, dermatitis herpetiformis, irritable bowel syndrome, rheumatoid arthritis, type 1 diabetes, thyroiditis, psoriasis and autism.*”¹²³

Some people believe NCGS is not real, but scientific evidence seems to support it is^{121,125}. It is not clear what percentage of the population fits into the concept of NCGS, but it could be in the range from 0.6 to 6%¹²⁶. It is not that clear, since, for example, 15% of the population suffers from irritable bowel syndrome¹²⁷, a condition that improves with a gluten-free diet. If we take into account that 1-2% of the population have celiac disease, the incidence of problems caused or aggravated by gluten does not seem negligible.

In a study done in Italy, only 14% of those who believed they had problems with gluten really had them (approximately: one half celiac disease, the other half NCGS)¹²⁸. It seems that in addition to a real basis, there is also certain gluten-related collective psychosis.

Gluten represents a large part of the proteins present in wheat, and is composed of glutenin and gliadin. Unlike other proteins that are found in grains, gluten proteins are partially resistant to be processed in the intestine, causing a continuous exposure of the intestinal immune system to these proteins¹²⁹. In particular, gliadin is a protein that no human being can digest completely, celiac or not. Gliadin increases the gut permeability in all of us (via a protein called

zonulin), an action that could detonate or aggravate autoimmune diseases that *a priori* would seem unrelated to gluten consumption^{94,124,130,131}. Some data suggest a reduction of autoimmune phenomena in people with celiac disease, from the moment in which a gluten-free diet is adopted¹³². An improvement in the functionality of the β -cells of the pancreas has also been reported in people at risk for type 1 diabetes, when gluten was excluded from the diet¹³³, and it has been suggested that perhaps a gluten-free diet could help prevent the development of type 1 diabetes¹²⁹.

In addition, it has been proven *in-vitro* that once gluten is digested, the union of leptin with its receptors is blocked¹³⁴, which could create leptin resistance and, consequently, a deregulation of appetite. The effect was seen at concentrations in the body that are normal when gluten is consumed. In summary, some people want us to believe that gluten is just a protein and that it is only a concern for people with celiac disease. I do not think so.

NOTE: gluten is also present in barley and rye.

GLUTEN-FREE DOES NOT MEAN WITH GLUTEN-FREE PRODUCTS

One of the arguments used to attack people who go gluten-free is to criticize gluten-free products. It is a straw man, because a gluten-free diet does not mean changing wheat for processed gluten-free products. Nobody says you have to exchange wheat bread for bread made with other grains. Just do not eat flour.

QUITTING BREAD IS NOT DANGEROUS

We are not talking here about self-medication, but to quit an edible product that is nutritionally poor^{122,135} and that, arguably, comes loaded with an excess of rapid-absorption sugars, which our body does not need at all^{73,136,137}. Starchy foods (breakfast cereals, biscuits, bread, pasta or pizza) are not required in a healthy diet.

For sure, the effects of grain flours may be different in different people. Anyone who thinks flour is good for them should continue eating flour. And if someone—for example because they have an autoimmune disease—believes that quitting flours may be beneficial for their health, they should give it a try, and observe the outcome in order to decide if they want to eat flour again. But it is not necessary to believe that gluten causes problems to us to justify quitting flour: it is not a wonder-food.

VISIT DR. WILLIAM DAVIS' BLOG

Dr. William Davis (<http://www.wheatbellyblog.com/blog/>) regularly publishes real stories of people who have improved their health and their body weight by quitting grains. Those are only personal anecdotes, but “before” and “after” pictures are really interesting.

CAP. 9 CHOLESTEROL

Cholesterol is an essential substance for our body. It is, for example, the main component of myelin¹³⁸, a substance that coats nerves and allows electrical signals to propagate at high speed along neuronal axons. It is also a substrate used to produce bile salts, hormones and vitamins¹³⁹. Cholesterol is so important that all the cells in the body can create it, although the liver has a main role in its production.

Cholesterol plays an important role in our body. We should not be treated with a drug that alters its function unless there is a well-founded reason for doing so. To date, this reason does not exist, although a large part of the population is treated for hypercholesterolemia (*i.e.* high cholesterol) with drugs whose benefits to health, demonstrated scientifically, are ridiculous. And they do not lack worrisome side effects.

Cholesterol is essential for the functioning of all human organs, but it is nevertheless the cause of coronary heart disease

Akira Endo

I believe that raised cholesterol has nothing whatsoever to do with the heart disease

Dr. Malcolm Kendrick

GOOD CHOLESTEROL AND BAD CHOLESTEROL?

We have all heard about the good cholesterol and the bad cholesterol, right? But there is no such thing. Blood cholesterol circulates in our blood encapsulated, along with triglycerides, in structures called lipoproteins. Lipoproteins can circulate in the blood as their external surface is hydrophilic, unlike triglycerides and cholesterol, which are hydrophobic.

There are different types of lipoproteins. On the surface they have proteins—called apolipoproteins—that somehow allow the type of encapsulation to be identified by receptors that exist in certain cells of the body.

The liver produces and releases a lipoprotein called VLDL (very low density lipoproteins). VLDL particles transport many triglycerides and little cholesterol, and for this reason they are not very dense. VLDL particles circulate in the blood and release their load of triglycerides, which go to the cells of the body (through the action of lipoprotein lipase) and this makes their density change, also changing their name to IDL (intermediate density lipoproteins), which are subsequently converted into LDL (low density lipoprotein). IDL particles have the same apolipoproteins as VLDL, being the only difference that their load of triglycerides has been reduced. Some apolipoproteins are lost in the conversion from IDL to LDL so those two types of particles are viewed differently by the cellular receptors.

Note that lipoprotein lipase is released to the interstitial environment by muscle and fat cells, facilitating that the fatty acids from the VLDL are stored in the adipose tissue or used as a source of energy in the muscles¹⁴⁰. The effect is the same on another type of lipoproteins loaded with triglyceride and cholesterol: chylomicrons created in the intestine that reach the bloodstream through the thoracic duct of the lymphatic system (see Chapter 3). Insulin stimulates the production of lipoprotein lipase in adipocytes, but not in the muscle cells, thus favoring the accumulation of fatty acids as body fat rather than their oxidation¹⁴¹.

In addition to the already mentioned lipoproteins, the liver also secretes HDL (high-density lipoprotein), which has characteristic apolipoproteins, and whose function is to withdraw other lipoproteins from circulation and carry their load back to the liver.

In short, if there were good and bad particles, we would be talking about particles, lipoproteins, and not their load, cholesterol, which is the same, just as good

or bad, when traveling within one or another. It is significant that scientists originally believed that the risk to our health was the amount of particles in our blood, but since at the time (1960's) the technology required for counting particles was not available, they decided to measure instead the amount of cholesterol, as a way of estimating the amount of particles¹⁴². From there, cholesterol has been demonized as if it were toxic to our body.

The marketing to demonize cholesterol has been so long and effective – it is quite possibly the best marketing campaign that has ever been undertaken – that people think that cholesterol lowering is the end goal – a benefit in itself. This is The Great Cholesterol Con

Zoë Harcombe

The fact is that we do not know for sure that these particles, good or bad, play a role at all. If there is a malfunction in our body, it is possible that an excess/shortage of particles is produced, resulting in a pattern of particles slightly different to that from a healthy person.

ARE LIPID RESULTS RELIABLE?

Let us also note also that the lipid profile is not always as reliable as we may think. Some of these data are not measured, but they are calculated by making assumptions that may be correct as an average, but not necessarily accurate for a specific person, such that a specific type of diet is followed, that everyone has the same amount of triglycerides in the lipoproteins or that the LDL particles are all the same size. The fact is that the LDL value is usually calculated by using the Friedewald equation¹⁴³:

$$LDL = TOTAL - HDL - TG/5$$

where TOTAL is the total cholesterol and TG is the amount of triglycerides.

If, despite what you read in this chapter, you still want to keep an eye on your cholesterol levels, in the absence of more reliable data perhaps the best option is to only take into consideration data that has been measured for real, for example the TOTAL/HDL and TG/HDL ratios, which when low can be indicators of good health¹⁴⁴.

EXCESS CHOLESTEROL BUILDS UP IN THE WALLS OF THE ARTERIES AND CAUSES CARDIOVASCULAR DISEASE?

Specifically, are the LDL lipoproteins, the so-called “bad cholesterol”, the cause of atherosclerosis? There is no evidence of that. What we nowadays hear from

official sources is, essentially, that cholesterol sticks to the walls of the arteries and obstructs them and, therefore, causes heart attacks. This is exactly what my little daughter told me, not long ago, that her teacher had explained to her. According to this theory, to have too many LDL lipoproteins is dangerous and the drugs that reduce the number of particles are beneficial to our health.

Coronary heart disease is an extremely complex malady and the expectation that it could be prevented or eliminated by simply reducing cholesterol appears unfounded

Dr. Michel de Lorgeril et al.

Scientists do not know how cardiovascular disease develops^{145,146}. They consider hypothesis, but they are not sure about the causes and mechanisms involved. It is a fact that when atheromatous plaques exist in an artery, in those plaques there is cholesterol, calcium, cells, etc. But that does not mean that the plaque is caused by an excessive amount of cholesterol in our blood. As a matter of fact the negligible effect of the drugs that lower cholesterol levels does not seem to be related to how much the cholesterol is lowered, neither the extent nor the progression of atherosclerosis seem to be related to the cholesterol levels (measured in autopsies or angiography)¹⁴⁷.

We are told that the LDL particles, thanks to their small size, penetrate into the arterial wall and that triggers a series of physiologic processes that end up developing the atheroma. We are also told that oxidized LDL particles or the small, dense LDL particles, are the dangerous ones. Were such hypothesis correct, the real cause of cardiovascular disease would be what makes us have such abnormal particles. Nevertheless, we should keep in mind that we are not talking about a cause-effect relationship here, just that statistics say it is more frequent that there is disease if you have a specific pattern of particles. A change in the pattern of particles, involved or not in the causation chain of the disease, could be just a consequence of the true cause of the disease. And that cause cannot be cholesterol, because we all have cholesterol and not everyone has that kind of particles. To have a high cholesterol does not seem either to cause having an —supposedly— atherogenic pattern of particles¹⁴⁸.

WHERE LIES THE CHOLESTEROL HOAX?

They have made us believe that cholesterol in the blood is a health hazard and that drugs that lower cholesterol are nothing short of a godsend. Those two ideas are false.

How is the deception created? In several ways:^{145,149–151}

- 1 Publication bias: it is the publication of studies that show good results for the drug, but not those that are inconvenient for the drug. All the studies published before 2004 are under suspicion of publication bias. Around the years 2005/2006, new stricter Regulations were introduced in the conduct

and publication of randomized controlled trials (RCTs). Since then, the outcomes of studies on drugs that reduce cholesterol have changed: they still reduce cholesterol levels, but what really matters, the benefits in mortality, are no longer found nor are there benefits for cardiovascular disease, which is what propaganda uses as bait.

- ② Violation of the experiment protocol, stopping at half time, when data seems to favor the drug. That trick always introduces a bias in favour of an illusion of effectiveness, which is justified on ethical grounds, but the fact is that effectiveness data is falsified, causing more harm than good in the long run. In other cases they directly include deceitful stop conditions in the study protocol.
- ③ Promotion as a main outcome of the study of a result of little relevance but that it is expected to be favorable to the drug. Thus, the results of the study can be presented as a success. In this sense, the only reliable outcome is mortality from any cause, because it is a hard outcome, *i.e.* not subject to interpretation, and because it is what the patient really needs to know: whether the drug saves their life or can kill them. For a patient it is irrelevant that the drug prevents a cause of death if other causes have an increased rate.

For example, in one study¹⁵² they defined as relevant end-point if the participants suffered symptomatic ischemia with objective evidence and requiring emergency rehospitalization. To the authors of the study to die was not as relevant.

- ④ Statistical deception, for example using percentage changes in percentage data, creating the appearance that the benefit is greater than it actually is. If, for example, after four years of drug treatment, 1 person out of every 100 dies in the medicated group and 2 people out of each 100 die in the placebo group, the pharmaceutical industry through its employees who publish studies in scientific journals, says that the drug reduces mortality by 50%. The reality is that in this example only 1 out of 100 people benefits, *i.e.* the benefit is only for 1% of those who are medicated for four years, while all of them are at risk of damaging their health and their quality of life because of the side effects of the drug.
- ⑤ Concealment of the side effects of the drugs. In some studies, participants who have problems with a drug in the run-in period are not allowed to participate in the study, so the actual incidence of side effects is falsified. In general, researchers are not usually that interested on analyzing the incidence of all the possible side effects, again creating the illusion that the drug is safer than it really is.
- ⑥ Concealment of data that do not help selling drugs, which leads to the absurd, for example, of reducing cholesterol levels in elderly despite the fact that statistics says there are more deaths the lower cholesterol levels are^{153,154}.
- ⑦ As I have said, there has been an intense marketing campaign to demonize cholesterol, making doctors and the general population believe that to

reduce cholesterol in our blood is a goal in itself. There are drugs in the market that although they were shown to be effective for lowering cholesterol levels, they have never showed health benefits¹⁵⁵.

IS THIS FOR REAL?

In an article¹⁵⁶ published in 2010 by the Cholesterol Treatment Trialists' (CTT) Collaboration, which is part of the Clinical Trials Service Unit in Oxford, which has received hundreds of millions of pounds over recent years to conduct research on behalf of the pharmaceutical companies¹⁵⁷ (and, therefore, with clear interest in making data look favorable for the industry) one of the conclusions was that lowering LDL cholesterol by 39 mg/dl for a year has associated an absolute reduction of mortality of 0.2%. That means treating 500 people to delay one death to happen after the considered period instead of before that. 499 patients out of every 500 would not get more benefit than the side effects of statins. But the authors of the article tell us there is a reduction of 10% in the death risk (a 10% reduction in the mortality from any cause). Mortality was 2.1% in the medicated group, 2.3% in the placebo group. The absolute difference is 0.2%.

What does a patient understand when they hear their risk of death is reduced by 10%? I have asked that question from a few of my family members and nobody understands the real effectiveness of the drug. On the other hand if they are told that only 1 out of 500 medicated patients benefits each year, prolonging their life an unknown period, they surely understand. But, in the case of cholesterol, telling the truth does not help sell drugs.

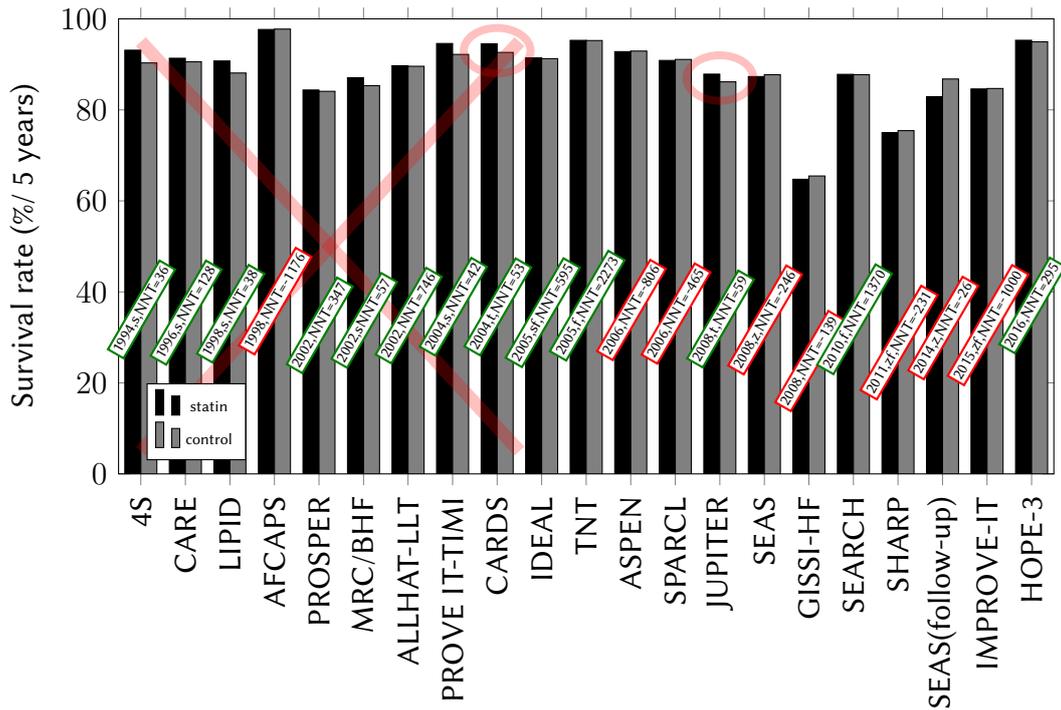
In my blog I posted¹⁵⁸ my review of a few scientific experiments that in my opinion make proclamations in favor of cholesterol-lowering drugs which are not backed up by their outcomes. Those studies are: JUPITER, SHARP, PROSPER, ASPEN, SEARCH, ALLHAT-LLT, AFCAPS, IDEAL, CARDS, SPARCL, TNT, MIR-ACL and J-LIT. In other articles¹⁵⁹ I did not see that the authors were misrepresenting the data in their conclusions, but the results of the cholesterol-reducing drugs were just as bad: AURORA, 4D, CORONA, GISSI-HF, PROVE IT-TIMI 22 and MRC/BHF.

According to those studies published after 2004, statins produce a postponement of death of just a few days, in the best cases¹⁶⁰.

My advice is that before you use cholesterol-reducing drugs, you should check the original data, which are the results published in scientific journals. And do not trust anyone's conclusions, especially those from the authors of the studies.

CHOLESTEROL-LOWERING DRUGS DO NOT SAVE LIVES

The following graph shows the rate of survival in some experiments with statins. I show in black the percentage of survivors in the experimental group, in grey the percentage of survivors in the control group. Note that after year 2004, the only study that shows some benefit in the survival rate was a study stopped ahead of time. The two studies highlighted with an ellipse were stopped early.



NNT: number needed to treat for 5 years so 1 of them delays their death after those 5 years, instead of dying in that period. f: control group also treated with statins. z: statin+ezetimibe. t: stopped early. s: secondary prevention

THE SECOND BATCH OF LIES

In the nutrition and health field, you not only have to watch out for the first lie: there are always more lies ready so the deception is maintained. In the case of statins, they tell us that although for primary prevention (people who had no prior cardiovascular events) statins do nothing, for secondary prevention they are effective. Still a lie. In secondary prevention, 83 people should be treated for five years so that one of them dies at an undetermined time after those five years, instead of dying before that moment¹⁶¹. To consider that this is an unequivocally effective treatment only shows that medical doctors are completely disconnected from reality.

RISK FACTORS

High blood cholesterol is one of the major risk factors for heart disease

National Institutes of Health

What does the sentence above mean? The message patients get is that a high cholesterol puts them at risk. But using the word “risk” is a hoax: it is used to make you think that there is cause-effect relationship, when the fact is that the available data are statistical/epidemiological, not physiologic. As far as we know, cholesterol is not a **cause** of disease, although a weak statistical association with disease might be found in part of the population.

“Risk” is a statistical term that means to what extent in the group of people who share a specific set of features is more likely to find a disease, compared with those who do not have that set of features. But this is not medicine nor it is physiology, it is just statistics.

There is no reason to think that reducing a risk factor is always beneficial for your health. That two parameters are statistically associated does not mean that one causes the other. For example, baldness is a cardiovascular risk factor¹⁶². Losing hair increases your risk, but that does not mean that a hair restorer is protective for your health.

They talk of “risk” to deceive people making us think that there is a danger in not using the drug. But it is not necessarily true.

PHARMACOLOGICAL MODEL OF MEDICINE

Medical doctors seem to be more sellers working for the pharmaceutical industry than health professionals. How did we arrive to the current situation in which medical doctors perceive their profession as prescribers of drugs?

On topics such as blood lipids the medical community has replaced knowledge with statistics. If a parameter, *e.g.* the LDL levels in blood, has a statistical relationship with a disease, they think it is justified to prescribe a drug to change that parameter, even when they do not know the real cause of the disease and even when they do not know if they are treating a symptom or the cause. And when the drug reduces cholesterol but shows no benefit for health they say it is a paradox. They have lost their way.

If, for example, you have non-alcoholic fatty liver disease the medical community looks for a drug that reduces intrahepatic fat¹⁶³, rather than consider what has caused this fat accumulation in the first place and, from that knowledge, advise the patient what diet or lifestyle changes are best suited to fix the problem.

Perhaps you believe that physicians do tell us what we have to change in our diet or lifestyle, but this is not true: the messages they give us almost always come from statistics (epidemiology) or industry’s propaganda, and, therefore, have neither scientific nor physiologic relationship with the disease (*e.g.* dietary cholesterol, saturated fat, whole-grains, etc.).

SPANISH ... ASSOCIATION?

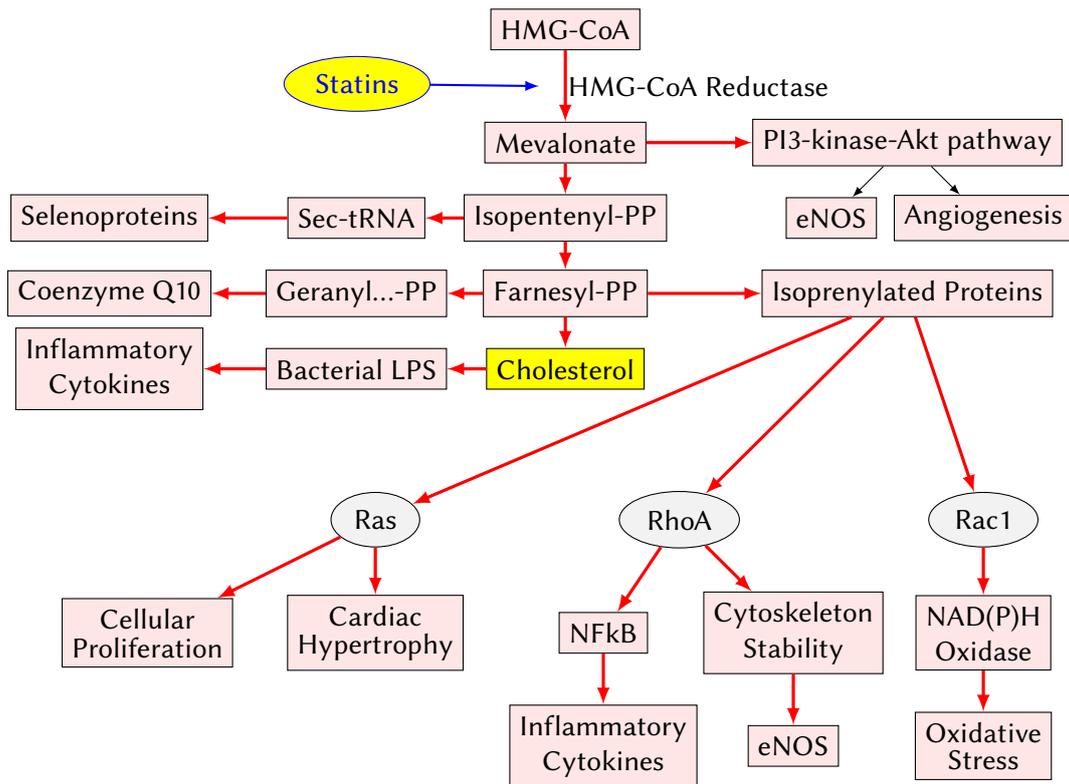
Quite probably you have never questioned what role do “scientific” associations or foundations that disseminate health information play. What is the point of their existence? Is our public health system unable of conveniently inform the public? (for sure, our public health system is doing a bad job of informing us, but that is a different issue)

Imagine, for example, that there is a European Association for the Study of the Fatty Liver, (which I think still does not exist, but this is a gap that the pharmaceutical industry can fill at any moment), what do you think would be its social role? The answer is obvious: the pharmaceutical industry wants to convey its messages to the population, but they lack credibility. And there are people who are willing to make this propaganda seem disconnected from the industry. One part has money to pay, but they lack credibility, and the other part has pockets that can be filled and is eager to participate in the scam. Next time you see the message from one of these associations in a pamphlet in the waiting room of a physician or as part of an article in a newspaper, I suggest you do a little research and look for the sponsors or collaborators of those associations or foundations, And there you will find the reason why these associations exists.

I am not saying that the funding source corrupts the message, what I am saying is that the funding source is the only reason why those associations exist. Conspiracy theories? No, this is the real world.

CHOLESTEROL-REDUCING DRUGS SIDE EFFECTS

Playing with the endogenous production of cholesterol is no joke. Notably, statins act by inhibiting an enzyme (HMG-CoA) in the mevalonate path¹⁶⁴, which is responsible for, among other things, cholesterol synthesis. Statins do not only reduce cholesterol, but they affect a whole set of important intermediate products and biochemical signals involved in diverse cellular functions.



For example, the deregulation of cholesterol can cause a malfunction of the β -cells of the pancreas, which in turn could explain a higher incidence of type 2 diabetes in people who are treated with statins^{165,166}.

In addition to diabetes, statins can cause muscle problems: myotoxicity in the form of myopathy, myalgia, myositis, or rhabdomyolysis¹⁶⁷⁻¹⁶⁹.

Some authors have expressed their concern about the possibility that statins increase the incidence of nonmelanoma skin cancer¹⁷⁰. This was suggested by the results from a pair of scientific experiments with statins, but, unfortunately, after those results contrary to the statins, in the following experiments the incidence of this type of cancer was not reported. A recent epidemiological article has found an increased statistical relationship between the use of statins and the incidence of this type of cancer¹⁷¹.

Statins may also generate cognitive problems in some people¹⁷².

I believe it is irrelevant how many patients suffer these side effects. Since the current proclamations of health benefit lack scientific support¹⁷³, in my opinion any side effect is excessive.

PATENTS AND SIDE EFFECTS

While a company holds the patent of a drug, they can sell it at a high prices. However, when its patent expires, the price is significantly reduced and it is much less profitable for pharmaceutical companies¹⁷⁴.

A fraudulent way to prevent that from happening is to change the drug to continue doing exactly the same thing, but being different enough as to justify a renewal of the patent¹⁷⁵. They are the so-called “me-too” drugs. For the patient

there is no benefit in this change of drug, but evidently there is a greater benefit to the company. In the case of statins (a type of drugs used to lower cholesterol levels), their patents are expiring. They are not as profitable as before. This is why the industry is no longer saying that statins are safe and free of side effects. They are ready to sell the new generation of cholesterol-reducing drugs, the PCSK9 inhibitors, and the message they are interested to emphasize right now is that statins do have side effects, but that for those people at a greater risk of suffering those side effects there is a new drug¹⁶⁹. A new drug with its patent in force and, therefore, much more expensive than statins. It was predictable that the same side effects of statins that were despised as infrequent, e.g. to cause diabetes or severe muscle problems, now are considered relevant: the more patients that have problems with statins, the more patients that can be changed to the new drug and the greater the benefit.

To date, these new drugs, the PCSK9 inhibitors, have only shown to reduce cholesterol¹⁷⁶. That is not the same as reducing mortality and it is possible that their side effects will be not known until, a few years from now, their patents expire¹⁷⁷.

HOW DOES FOOD AFFECT YOUR LIPID PROFILE?

Do you really need to know this? Do you think that eating “food” your lipid profile is going to be bad? I think that you do not have to be an engineer to deduce how to eat well. In any case, if you are concerned about your lipid profile, here you have a few results published in the scientific literature:

- ① A low-fat high-carbohydrate diet makes your type of LDL particles worse, increasing the amount of those that are believed to be related to cardiovascular disease^{77,85}. Although LDL cholesterol levels may increase slightly with a high-fat low-carbohydrate diet, that is not necessarily a bad sign.
- ② A low-fat high-carbohydrate diet increases triglycerides levels^{74,178,179}.
- ③ A low-carbohydrate diet improves HDL levels⁹⁸.
- ④ Seed oils may change the LDL particles to a profile that is believed to be atherogenic¹⁸⁰⁻¹⁸².
- ⑤ If you reduce your intake of saturated fat a worsening of HDL and triglycerides levels can be expected^{84,90}.

I am not talking about the saturated fat in cakes or biscuits. I mean the saturated fat in real food that has saturated fat, exclusively. And that fat is not bad for our health^{86,89}.

Not-food has to be avoided, regardless of its macronutrient distribution.

Apart from the points above, my conclusion is still the same I told you before: eating “food” is not dangerous for our health. There is no need to read scientific studies in order to know that.

WHAT DO I DO IF I HAVE HIGH CHOLESTEROL?

If a physician prescribes you a cholesterol-lowering drug, you should ask him/her how many people with your same characteristics (age, health status, sex, physical condition, current medication, etc.) must be treated, and how long, to delay the death of one of those people beyond the duration of the study (*i.e.*, that person dies at an unknown time after the study instead of before the end of the study). Tell him/her that you do not need the data right away, that you can wait until your next visit. If your doctor gives you the scientific studies that demonstrate the effectiveness of the treatment, use those data to make a decision. If, on the other hand, the physician takes offence because you ask him for the data you need to make an informed decision, that will open your eyes about the unscientific basis of cholesterol-reducing recommendations.

Even if having a high cholesterol were the cause of health problems, something about which I have serious doubts, the benefit provided by cholesterol-reducing drugs is minimal. It is not an insanity to ignore one's lipid profile and just focus on following a healthy diet and doing regular physical exercise. To continue to eat "not-food" and to take cholesterol-reducing drugs, is not one mistake: these are two mistakes.

CAP. 10 FAT-SHAMING AS MOTIVATION

Obese people have a willpower problem: they cannot stop eating and they follow sedentary lifestyles. Moreover, they do not realize that to be obese is a health risk. Or they get that but they do not care. But you are smart and you do know that obesity is a health risk and you want to help them by telling them to stop pretending to be a victim and do nothing to improve their health. People should stop excusing them because what they need is effort and sacrifice, not laziness and complacency.

Does this narrative make sense to you? Do we have a bigger understanding than people who are overweight or obese? Can we see things that they do not see? Do we know what they have to do if they want to lose their excess weight? Is self-acceptance the same as not caring about your health?

Obesity can lead to health problems but the humiliating messages to people who have excess weight are so powerful and harmful, that a relevant percentage of the obese population would rather die a few years sooner, lose a leg or become completely blind, than be thought of as obese¹⁸³. Despite this reality, some people believe that everything is as simple as that obese people do not strive. Does this really make sense?

If it were so simple, don't you think I would have already done it? All those comments didn't provide a solution, all they did it was make me sad and depressed, and make me eat even more

Anonymous

IMAGINE...

Imagine that there is a homeopathic treatment for obesity and that that remedy is promoted by all the health professionals in your country, but **the treatment has failed as many times as it has been tested** in scientific experiments. And in real life hardly anyone goes from being obese to have a normal weight. However, those outcomes are ignored by the experts and the general population is not told about them, while at the same time they insist that the solution of this problem is as simple as applying the treatment they recommend. Would you find it reasonable that the promoters of homeopathy humiliated and fat-shamed obese people, urging them to take responsibility for their health arguing that the problem is that do not care to apply the treatment?

Surreal as it may seem, this is exactly what is happening now with obesity. Obese people are harassed and humiliated for not applying a treatment, the infamous "eat less and move more" that it is not only based on pseudoscience, it has NEVER been proved to be effective for weight loss. Not in a single scientific experiment.

A PROBLEM OF ARROGANCE

Let me start by saying that during my 25 years of being overweight, the last thing I needed was to be INFORMED that I was overweight

Anonymous

Obese people:

- ① Need to be reminded that they have a weight problem, because do not realize, or they do not care.
- ② Need to be reminded that they have to try, because they do not know that, or they do not care.
- ③ Need to be told that obesity is a health risk, because they do not know that, or they do not care.

Is that what you see? You are smart and you see everything while fat people are so dumb that they can't see even the obvious? In addition to lazy and gluttonous, are they also idiots?

IDIOCY AND IGNORANCE FUEL FAT-SHAMING

The message that justifies the prejudices against the obese is, basically, the energy balance pseudoscience, which can be summarized in these two ideas:

- ① We get fat because we eat too much and/or move too little.
- ② With a balanced diet, portion control and moderate physical exercise, it is possible to lose weight and keep the reduced weight in the long run.

It might sound reasonable, but in addition to a fraud, this is the hurtful message: if the obese knows how to stay slim and also knows how to lose weight, not achieving a reduced weight is their fault and their responsibility. If they had willpower, they would not be in that situation.

But, as I have already said, these beliefs, those that lead the smartasses to think that obesity is a problem of willpower, are no more than pseudoscience. The first idea is blatantly stupid. The second one, despite its stupid origin, could have worked, but it did not: it has a record full of failures in scientific weight-loss experiments. These ideas define their promoters as ignorant idiots. But they are also aggressors with victims.

Fat shaming: A term made by obese people to avoid the responsibility to actually take proper care of their body and instead victimize themselves [sic] by pretending they're discriminated like an ethnic group

urbandictionary.com

I insist that this is key:

Where is the scientific evidence that supports that the treatment (low calorie diet and exercise) that obese people are told to follow is effective for weight loss?

According to the scientific evidence, what percentage of the obese people will lose a significant amount of weight, let us say 20 kg, and maintain their reduced weight in the long term, let us say 5 years, with this treatment? Specifically, what feature do they have that guarantee that they will indeed lose weight with this treatment? I insist: I am asking for scientific evidence, not hunches. It is not enough to have the belief that the method works, especially when the method we are talking about has no more scientific basis than pure human stupidity. When our messages hurt those of us who have excess weight, beliefs are not enough basis to justify our actions.

Another important question is: do we believe that weight gain is a personal decision? Why do we believe that a person who perceives that he is getting fatter, knows what he has to do to stop and reverse this process? What measures, scientifically proved, guarantee that those measures will work if they are applied? Any

I will not be FAT again

scientific evidence in this regard? The truth is that for decades obesity experts have been telling people that the energy balance equation is the law that governs our body weight and that fighting obesity is as simple as reducing our caloric intake and following a less sedentary lifestyle. What is the evidence that supports this message? Do I seem to be playing the victim? I believe that we obese people, ex-obese in my particular case, have received and are receiving misinformation about how to eat to be healthy and how to control our body weight. For sure, we are also guilty of that.

Fat people are fat because they are ill-informed about what makes them fat, and this needs to change, for everyone's sake

Anonymous

THERE IS NOTHING INNOCENT IN THE "ADHERENCE IS KEY" MESSAGE

The emphasis on messages that are absolutely devoid of scientific basis, as is the case of "weight loss is a question of having full adherence to a diet, any diet", only helps to perpetuate prejudices. The corollary is clear: if you do not get rid of your excess weight the reason is you are not able to maintain a diet. You lack the required willpower and you are to blame.

The damage is much more serious than it seems, when you consider that the aggressor is not the only one who (presumably) ignores that the message is a fraud: the victims also believe they know the cure for their problem, so they do not look for other solutions and they blame themselves for not losing their excess weight.

I wasn't ignorant just because I was overweight. I knew I needed to eat right. I knew I should exercise. I knew it all. Doing it was another thing

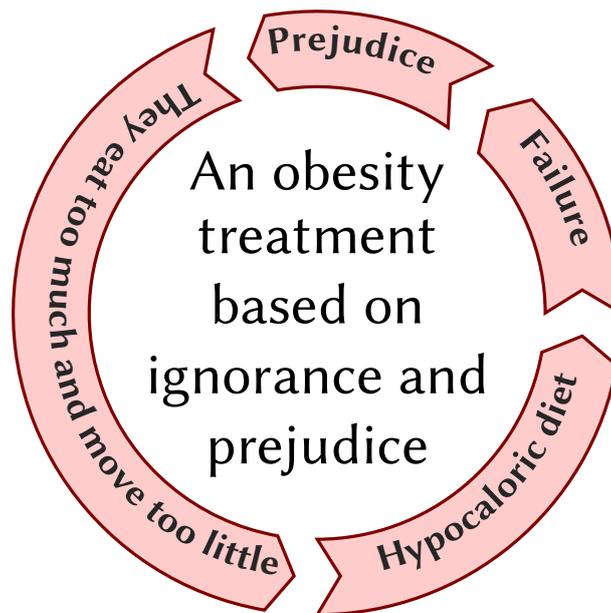
Anonymous

Even people who say they are against fat-shaming, paradoxically tell the obese what are the effective and healthy strategies for weight loss. What strategies do they know that are effective for weight loss?

It is reasonable to speculate that, if one day we find a weight loss treatment based on diet and exercise, following those dietary and exercise guidelines will require willpower. We can all agree that in this hypothetical case, willpower will play a role in the process. But, how do we explain making comments now on the alleged lack of willpower of the obese people?

THE VICIOUS CIRCLE

Since we know that the problem is that obese people lack willpower to stop eating, it is obvious that the solution must be to eat less. The solution almost always fails and in the experts' eyes the failure of the method just confirms that the obese lack willpower to adhere to a diet in the long term. Their prejudices are confirmed true.



People high in prejudices might be less responsive to outgroup member's needs and feelings and be less likely to understand their intentions¹⁸⁴.

ARE SELF-ESTEEM AND SELF-ACCEPTANCE DANGEROUS?

Is obesity bad? No way! They even say we have to accept the diversity of bodies.. uff danger!

Anonymous

This is the normalization of obesity using self-esteem as an excuse. Yeah! That's how it is

Anonymous

Some time ago we could see a short video on twitter in which a woman advocated that society should adapt to the existence of people with big bodies. A radical message, no doubt about that. This woman smiled and talked with confidence. The reaction to the video was to accuse her of playing the victim and not

I will not be FAT again

accepting her responsibility in her problem. If you are fat, to smile and to show self-esteem is a provocation, because it certainly means you lack desire to solve your problem. If you were responsible you would be sad and would apologize for being alive.

Our study clearly shows that weight discrimination is part of the obesity problem and not the solution

Jane Wardle

Should we fight self-esteem and self-acceptance? Is fat-shaming a reasonable way of motivating a person to lose weight? If we pretend that obese people do not exist, if we ignore them, if we make their life hell, will obesity disappear? In which contexts has shaming proved to work for motivation? At work? With students? In sport competitions? Such aggressions not only do not help, they probably aggravate the problem¹⁸⁵⁻¹⁹⁰. For example, by itself stress can cause weight gain by altering cortisol levels¹⁹¹.

In any case, I insist: why do we believe that obesity is a problem of lack of responsibility or lack of willpower? It is not the first time in the history of medicine that ignorance and prejudices are used to assign the cause of a disease to the behavior of the patient. We are just as stupid and ignorant as our ancestors.

WE NEED LITTLE EVIDENCE TO CONFIRM OUR PREJUDICES

I presume that all of us have read or heard comments that blame the victims for their obesity, like the ones below:

- ① I see that thin people do not eat too much
- ② People are not aware of how much they eat
- ③ People quit diets
- ④ I know people who lost weight by eating less
- ⑤ Fat people consume products that we all know are fattening

The scientific evidence, which clearly says that the promoted treatment does not work, is ignored while observations that represent the lowest level of evidence are treated as proof. To confirm our prejudices anything is enough. Literally anything.

THE DANGERS OF MAKING EASY FOR THE OBESE TO GIVE EXCUSES

Saying out loud that “eat less and move more” has never worked is dangerous. Obese people are desperate to find an excuse to stop striving, and this message

gives them what they seek. We must hide this information and go on encouraging a method that has never proved to be useful for weight loss. We must promote frustration, torment, and make them believe they are to blame for their problem.

I am not making this up: this is exactly what we are told by some nutrition “experts”. Of course, explaining physiology is also seen as dangerous and reprehensible, because some obese people could understand that the reason why they cannot lose weight is something different from eating eat too much for their energy expenditure, the only truth of the calorie counters.

We should not make the mistake of telling obese people that the cause of their problem is genetic or metabolism and that, therefore, it is not under their control

Anonymous

The existence of an adipocytes-based autonomous cause for weight regain may be taken as an excuse to abandon measures needed to maintain the reduced weight

Edwin C. M. Mariman

He is talking about those measures that have systematically failed as a method to keep the reduced weight. But do not tell this to the obese people because they would use it as an excuse to stop taking those measures.

I DO NOT KNOW IF BEING SLIM IS WORTH THE EFFORT

If I can not eat a sandwich when I watch a match with my friends, I do not know if I want to be slim

Anonymous

In my opinion, doing something that you know hurts your health, is a symptom of addiction. Since in food addictions a processed product, *i.e.* added to our natural diet, is often involved, it seems reasonable to argue that the problem does not lie in the person. Human food does not generate addiction: the problem comes from the consumption of products that do not belong to our diet.

I disagree with the comment from this person. My actual way of eating is full of reward and I am convinced it is healthy. When we find a diet that keep us healthy and thin, in my opinion the change of habits is worth the effort. I imagine that in some cases, such comments can be a way of avoiding to acknowledge that they can not lose weight. These would be people who try it once and again, and since they never succeed and that are led to believe that the fault is theirs (“had

I maintained the diet I would have kept the reduced weight”), they avoid the torment about what they think is a personal failure by convincing themselves that they do not want to lose weight.

Apart from addiction, what else can lead someone to believe it is not worth the effort? In my opinion, the problem is that older people, around 50 years old in this case, are used to life habits that they see as their way of life. Furthermore, certain products are an important part of their social life. To stop eating the things they so much like, those that are probably the basis of their social life, is seen as a failure that has negative implications not related to health. If no one in your social circle is doing the same, and the official bodies are saying that the excesses of one day can be compensated by eating less another day, what reason will they find to give up the beer or the sandwich? They cook without oils, they take away the fat and everything is fine. It is very easy to justify that you do not need to change anything.

We have grown up believing in the energy balance theory, which allows us to eat whatever we want as long as we compensate the caloric excess. The result has been an obesity epidemic. My impression is that as long as such pseudoscience continues to be promoted from official institutions, many people who have excess weight will still believe that it is possible to be slim without giving up anything. It is an easy choice because it is what the official sources say, and they want to go on eating as they are used to. As long as those official messages put sticks in the wheels, I cannot see reasons to blame those who follow that advice, although I believe they are wrong.

In any case, I insist that we are talking here about people who say they do not know if they want to do something, to lose weight, that they do not know how to do. To focus on their motivations, after decades of deception about the causes of the problem and the real options that they have, legitimizes the deception.

CONCLUSIONS

If we do not have a cure for obesity, does this mean that there is no reason to stop eating crap? I did not say that nor did I say anything alike. What I am saying is that the general population is being misled on the causes and treatments for obesity. And those lies have grave consequences for those people who suffer from obesity.

If we want to solve the problem of obesity:

- ① We must find a treatment for obesity that works for real (of course, proved with scientific evidence, not with quackery).
- ② This treatment has to be promoted from public institutions (and in the meanwhile they should stop promoting the energy balance pseudoscience).
- ③ People who do not want to apply the treatment and those for whom the treatment does not work should be treated with respect.

I am not saying that we fail in regard to the third point, I say that **none** of the three points above is fulfilled^{192,193}. I also say that the misinformation in the first two points is the main reason why the third one is not fulfilled.

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