The Bitter Truth About Sugar by Robert Lustig (Full Transcript)

Robert H. Lustig, a Professor of Clinical Pediatrics at University of California, San Francisco (UCSF), here details and explores the real truths about sugar that you perhaps never heard about much before. We thought it might be useful for our readers. So we decided to do a full transcript on his about 90-minute YouTube video called "Sugar: The Bitter Truth". Hope you find it useful and informative. Please use the "The Bitter Truth About Sugar" slides below while reading this transcript...

The Bitter Truth About Sugar by Robert Lustig Slides [PDF file]

Introduction

I'm going to tell you tonight a story and this story dates that about 30 years. This story has a little bit of something for everybody. It has a little bit of biochemistry, a little bit of clinical research, a little bit of public health, a little bit of politics, a little bit of racial innuendo. The only thing it's missing is sex. But well, we can see what we can do about that too.

By the end of the story, I hope I will have debunked the last 30 years of nutrition information in America. And I would very much appreciate if at the end of the talk you would tell me whether or not I was successful or not.

Atkins versus Japanese diet

Okay. So in order to get you in the mood as it were, let's start with a little quiz. What do the Atkins diet and the Japanese diet have in common? Anybody? Well you have the answers right – yes, never mind, that's right. You have the answer right there.

So the Atkins diet of course is all fat, no carb. The Japanese diet is all carb, no fat. They both work. So what do they share in common? They both eliminate the sugar fructose. With that, think about what it means to be on a diet and what macro-nutrients you are eating and which ones you're not. And then we'll go from there and I'll try to explain how this all works.

So you've all heard about the obesity epidemic. Here are the numbers. These are the Anne Haines database body mass index (BMI). Everybody knows what that is now. Histograms marching ever rightward as time has gone on… this was what was projected for 2008 in blue. We had so far exceeded and surpassed this - it's not even funny - from 2003. The reason I show this is not just to show that the obese or getting obese, or of course that's true, but in fact, the entire curve has shifted. We all weigh 25 pounds more today than we did 25 years ago, all of us.

Now it is often said that obesity is the ultimate interaction between genetics and environment. But having said that our genetic pool did not change in the last 30 years. So tonight we're going to talk about the environment rather than genes.

Obesity epidemic

Now in order to talk about the environment we need to talk about what is obesity, and of course, you're all familiar with the basic concept of the first law of thermodynamics, which states that the total energy inside a closed system remains constant. Now in human terms, the standard interpretation of this law is the following: If you eat it, you better burn it or you can store it. Now who here believes that? Oh come on, you all do.

I used to believe that. I don't anymore. I think that's a mistake. I think that is the biggest mistake and that is the phenomenon I'm going to try to debunk over the course of the next hour, because I think there's another way to state the law, which is much more relevant and much more to the point. Before I get there, of course, if you believe that these are the two problems: calories in, calories out, two behaviors – gluttony and slough. After all you see anybody on the street, he's a glutton and slough. You know Tommy Thompson said it on the TV show, "we just eat too damn much".

Well, you know if that were the case, how do the Japanese do this? Why are they doing bariatric surgery on children at Tokyo Children's Hospital today? Why are the Chinese? Why are the Koreans? Why are the Australians? I mean, all these countries who have adopted our diet all suffer now from the same problem, and we're going to get even further in a minute.

There is another way to state this first law. And that is, if you're going to store it, that is biochemical forces that drive energy storage – we will talk about what they are

in a few minutes - and you expect to burn it, that is normal energy expenditure for normal quality of life, because energy expenditure and quality of life are the same thing. Things that make your energy expenditure go up, make you feel good -- things that make your energy expenditure go down, like starvation, hypothyroidism, make you feel lousy. And how many calories you burn and how good you feel are synonymous.

So if you're going to store it, that is an obligate weight gain set up by a biochemical process and you expect to burn it, that is normal energy expenditure for normal quality of life, then you're going to have to eat it. And now all of a sudden these two behaviors, gluttony and slough are actually secondary to a biochemical process, which is primary. That's a different way to think about the process and it also alleviates the obese person from being the perpetrator but rather the victim, which is how obese people really feel, because no one chooses to be obese. Certainly no child chooses to be obese.

Oh, you say, oh yes, sure I know some adults who don't care. You know Rossini, the famous composer, he retired at age 37 to a lifetime of gastronomic debauchery. Okay, maybe he chose to be obese. But the kids I take care of an obesity clinic do not choose to be obese. In fact, this is the exception that proves the rule. We have an epidemic of obese six-month olds.

Now if you want to say that it's all about diet and exercise, then you have to explain this to me. So any hypothesis that you want to proffer that explains the obesity epidemic, you've got to explain this one too. And this is not just in America, the six-month old obese kids but these are around the world now. So you're your minds and let's go and figure out what the real story is.

Calorie intake & leptin

Now let's talk about calorie intake, because that's what today is about. We're going to talk about the energy intake side of the equation. Sure enough we are all eating more now than we did 20 years ago. Teen boys are eating 275 calories more. American adult males are eating 187 calories more per day. American adult females are eating 335 calories more per day. No question. We're all eating more. Question is why? How come? Because it's all there, you know what it was there before. We're all eating more.

There is a system in our body which you've heard about over the last couple of weeks called leptin. It's a hormone that comes from your fat cell, tells your brain "You know what, I've had enough. I don't need to eat anymore. I am done and I can burn energy properly". Well, you know what, if you're eating 187 or 335 calories more today than you were 20 years ago, your leptin ain't working, because if it were, you wouldn't be doing it whether the food was there or not.

So there's something wrong with our biochemical negative feedback system that normally controls energy balance. And we have to figure out what caused it and how to reverse it, and that's what tonight is about. But nonetheless there are 275 calories we have to account for. So where are they? Are they in the fat? No, they are not in the fat. 5 grams 45 calories out of the 275, nothing. In fact, it's all in the carbohydrate. 57 grams, 228 calories, we're all eating more carbohydrate. Now you all know back in 1982, the American Heart Association, the American Medical Association and the US Department of Agriculture admonished us to reduce our total fat consumption from 40% to 30%. Everybody remember that? That's how Entenmann's fat-free cakes came into being.

So what happened? We did it. We've done it. 40% down to 30% and look what's happened to the obesity metabolic syndrome, non-alcoholic fatty liver disease, cardiovascular disease, stroke prevalence, all jacked way up as our total fat consumption as a percent has gone down. It ain't the fat people. It ain't the fat. So what is it? Well, it's the carbohydrate. Specifically, which carbohydrate? Well, beverage intake - 41% increase in soft drinks, 35% increase in fruit drinks, fruit aids, whatever you want to call them. Just remember down here, one can of soda a day is 150 calories, multiply that by 365 days a year and then divide that by the magic number of 3500 calories per pound, if you eat or drink 3500 calories more than you burn, you will gain 1 pound of fat. And that's the first law of thermodynamics. No argument there. That's worth 15.5 pounds of fat per year. One soda a day is 15.5 pounds per year. Now you've all heard that before. That's not news to you.

The question is, how come we don't respond? How come leptin doesn't work? How come we can't stay energy stable? That's what we're going to get to.

Coca-Cola conspiracy

So I call this slide very specifically the Coca-Cola conspiracy. Anybody here work for Coke? Pepsi? Okay, good. All right. So this over here - 1915 the first standardized bottle of Coca-Cola out of Atlanta. Anybody remember this bottle? I remember this bottle because my grandfather in Brooklyn took me on Saturday afternoon down to the local soda shop on Avenue M and Ocean Avenue and every Saturday afternoon I have one of these. I remember very well.

Now if you drank one of those everyday assuming of course that the recipe hasn't changed, because after all only two people in the world know the recipe and they're not allowed to fly in the plane at the same time, right, you know that? Assuming the recipe hasn't changed, if you drank one of those everyday for a year, 6.5 pounds, that will be worth 8 pounds of fat per year.

Now in 1955 after World War II and sugar became plentiful, again it wasn't being rationed, we have the appearance of the 10-ounce bottle - the first one that was found in vending machines. You probably remember that one as well. Then in 1960 the ever ubiquitous 12-ounce can worth 16 pounds of fat per year, and of course today this over here is a single unit of measure, 20 ounces, anybody know how many servings are in that bottle? 2.58 ounce servings, that's right. Anybody know anybody who gets 2.58 servings out of that bottle? That's a single serving. So that will be worth 26 pounds of fat per year if you did that everyday and then of course over here we have the 7-Eleven Big K, Thirst Buster, Big Gulp whatever you want to call it, 44 ounces worth 57 pounds of fat per year and if that wasn't bad enough, my colleague Dr. Dan Hale at the University of Texas, San Antonio tells me that down there they got a Texas sized Big Gulp. 60 ounces of Coca-Cola, a Snickers bar and a bag of Doritos all for $0.99. If you did that everyday for a year, that would be worth 112 pounds of fat per year.

So why do I call it the Coca-Cola conspiracy? Well what's in Coke? Caffeine. So what's caffeine? It's a mild stimulant, it's also a diuretic, makes you pee free water. What else is in Coke? We'll get to the sugar in a minute. What else? Salt - 55 mg of sodium per can, it's like drinking a pizza. So what happens if you take on sodium and lose free water, you get thirstier. So why is there so much sugar in Coke? To hide the salt. When was the last time you went to a Chinese restaurant and had a sweet and sour pork? Everybody remember new Coke, 1985, more salt more caffeine. They knew what they were doing. That's the smoking gun. That's why it's the Coca-Cola conspiracy.

Soft drinks - the cause of obesity?

Well it depends on who you ask. If you ask the scientist for the National Soft Drink Association, he will tell you there is absolutely no association between sugar consumption and obesity. If you ask my colleague Dr. David Ludwig, remember I'm Lustig, he is Ludwig. He does what I do at Boston Children's Hospital. Someday we're going to open a law firm. Each additional sugar sweetened drink increase over a 19-month follow-up period in kids increased their BMI by this much and their odds risk ratio for obesity by 60%. That's a prospective study on soft drinks and obesity.

If you look at meta-analyses – a conglomeration of numerous studies subjected to rigorous statistical analysis – 88 cross-sectional longitudinal studies regressing soft drink consumption against energy intake, bodyweight, milk and calcium intake, adequate nutrition all showing significant associations and some of this being longitudinal, this came from Kelly Brownell's group at Yale. I should comment a disclaimer - those studies that were funded by the beverage industry showed consistently smaller effects than those that were independent. Wonder why?

Now how about the converse - what if you take the soft drinks away? So this was the fizzy drink study from Christchurch England James [Adolph] British Medical Journal where they went into schools and they took the soda machines out – just like we did here in California. We haven't seen the data yet but they went and did it for a year. So the prevalence of obesity in the intervention schools stayed absolutely constant, no change whereas the prevalence of obesity in the controlled schools where nothing changed continued to rise over the year.

Soft drinks and Type 2 diabetes?

So how about type 2 diabetes? Are soft drinks the cause of type 2 diabetes? Well this study from JAMA in 2004 looked at the relative risk ratio of all soft drinks, cola, fruit

punch and found a very statistically significant trends of sugared soft drinks, fruit aids etcetera causing type 2 diabetes and you know we've got just as big a problem with type 2 diabetes as we do with obesity for the same reasons. This was a sugar sweetened beverage (SSBs) against risk for type 2 diabetes in African American women, looking here at sugar sweetened soft drinks, just the downward arrow shows that there was a significant rise as the number of drinks went up. You can see that here whereas orange and grapefruit juice interestingly did not. So, two different studies, two different increases in type-2 diabetes relative to soft drink consumption.

So what's in soft drinks?

Well, in America it's this stuff, right? High fructose corn syrup. Everybody's heard of it, right? It's been demonized, something awful so much so that the corn refiners industry has launched a mega-campaign to try to absolve high fructose corn syrup of any problems which we'll talk about in a moment. But the bottom line is this is something we were never exposed to before 1975. And currently we are consuming 63 pounds per person per year - everyone of us. 63 pounds of high fructose corn syrup. That's America.

What is high fructose corn syrup?

Well, you'll see in a minute. It's one glucose, one fructose. We'll talk about those at great length. One of the reasons we use high fructose corn syrup is because it's sweeter. Here is sucrose. This is cane or beet sugar, standard-table sugar, you know, the white stuff. And we give that an index in sweetness of 100. So here is high fructose corn syrup. It's actually sweeter. It's about 120. So you should be able to use less, right? Wrong. We use just as much. In fact, we use more.

So here is lab fructose over here, crystalline fructose, and they are starting to put crystalline fructose into some of the soft drinks. They are actually advertising it as a good thing. And that's got a sweetness of 173. So you should be able to cut that way back, right? They are not.

Lactose down here, milk sugar, that's not sweet at all. And glucose I should point out over here 74, it's not particularly sweet and we're going to get to that at the end and what goes on with glucose. But anyway there's why we use it: it's sweeter. It's also cheaper as I'll show you.

Here's high fructose corn syrup. One glucose, one fructose. Notice the glucose is a 6-membered ring. The fructose is a 5-membered ring. They are not the same. Believe me, they are not the same. That's what this whole talk is about. It's how they are not the same.

And here is sucrose. And they are just bound together by this ether linkage. We have this enzyme in our gut called sucrose. It kills that bond in 2 seconds-flat and you absorb it. And basically high fructose corn syrup sucrose, it's a non-issue. It's a wash. They are the same. And they know that they are the same - the soft drinks companies and the corn refiners. Because here are their missives. This comes from the corn refiner association: " Obesity research shows high fructose corn syrup metabolizes and impacts satiety similar to sugar." Indeed it does.

Hunger and satiety profiles, energy intake, following ingestion of soft drinks. Bottom line: research supported by the American Beverages Institute and the Corn Refiners Association. They are correct. There is absolutely no difference between high fructose corn syrup and sucrose. So much so that the Corn Refiners Association in attempt to capture market share came out with this entire ad campaign. You probably saw it on the back page of the New York Times. It was on TV. It's everywhere.

"My hairdresser says that sugar is healthier than high fructose corn syrup." “Wow? You get your hair done by a doctor?" I didn't know I could cut hair. If you all want to see all of them there are a whole bunch and you can go to www.sweetsurprise.com and see how you are being hoodwinked? But indeed this is true. High fructose corn syrup and sucrose are exactly the same. They are both equally bad. They are both dangerous. They are both poison. I said it: poison. My charge before the end of tonight is to demonstrate that fructose is a poison. And I will do it. And you will tell me if I was successful.

Nonetheless here's Center for the Science in the Public Interest (CSPI) and the Corn Refiners Association. Everybody remember last year when Gavin Newsom floated the soda tax last February? Governor Paterson of New York has since floated one and other people are starting to talk about it. So why are they saying this? Well, they are saying "Obesity is

a problem. Kids are drinking soda. Let's tax it." So they are talking about soda like it's empty calories. I'm here to tell you that it goes way beyond empty calories.

The reason why this is a problem is because fructose is a poison. It's not about the calories. It has nothing to do with the calories. It's a poison by itself. And I am going to show you that. Nonetheless I just want to read you this paragraph here: "We respectfully urge that the proposal be revised as soon as possible to reflect the scientific evidence that demonstrates no material differences in the health effects of high-fructose corn syrup and sugar." I agree. Here is the important sentence. “The real issue is that excessive consumption of any sugars may lead to health problems." I agree. That's exactly right. Not "may", "does".

So here is the secular trend in fructose consumption over the past 100 years. Before we had food processing, we used to get our fructose from fruits and vegetables and if we did that today, we would consume about 15 grams per day of fructose - not sugar, fructose. Sugar would be 30 grams, would be double. We're just talking about fructose today.

Prior to World War II, before it got rationed again, we were up to about 16 to 24, about 20 grams. So a small increase from the beginning of the century to World War II. Then in 1977, just as high fructose corn syrup was hitting the market, we had increased that, we had basically doubled up to 37 grams per day or 8% of total caloric intake. By 1994 we were up to 55 grams of the stuff per day. Remember if you want to do sugar, then double the number. So that's 10.2. So you can see that more and more of our caloric intake, a higher percentage is being accounted for by sugar every single year. So it's not just that we are eating more. We are eating more sugar. And for an adolescent today we are up to almost 75 grams, 12% of total caloric intake. 25% of the adolescents today consume at least 15% of their calories from fructose alone. This is a disaster. An absolute unmitigated disaster. The fat is going down. The sugar is going up. And we are all getting sick. Now let me show you why.

Perfect storm

How did this happen? Why did it happen? This is where the politics comes in. This is the perfect storm. And it was created from three political winds that swirled around all at the same time to create this perfect storm. So the first political wind. Everything bad that ever happened in this country started with one man. And it's still being felt today.

So Richard Nixon, in his paranoïa back in 1972, food prices were going up and down, up and down and I'll show you that on the next slide. And he was worried that this was actually going to cost him the election. So he admonished his secretary of Agriculture Earl Rusty Butz to basically take food off the political table, to make food a non-issue in presidential elections. Well, the only way to do that was to make food cheap. So he was out to find all methods to be able to decrease the price of food. Remember Nixon's war on poverty? This we are suffering from it today. That's what this is.

Second political wind: the advent of high fructose corn syrup. This was invented in 1966 at Saga Medical School in Japan by a guy named Takasaki who is still alive. As far as I am concerned, this stuff is Japan's revenge for World War II. Except of course that they are suffering from it now themselves. Like everything, you know it always comes back to haunt you.

And it was introduced to the American market in 1975. So what do you think happened to the price of sugar when this thing hit the market? Here is what happened. The US producer price index of sugar going up and down and up and down…… this is not good. Stability is at 100%, if it stays nice and stable at 100%, that's what you want, if you’re a politician, up and down.

Here is where corn sweeteners entered the market 1975-1980 and you can see that since then the price of sugar has remained remarkably constant, and it did so not just in the US but also on the international stage. Here is the London price doing the same thing and when you look at the difference in price between sugar and high fructose corn syrup, you can see that high fructose corn syrup is about half the price. So in other words, it's cheap. So high fructose corn syrup is evil, but it's not evil because it's metabolically evil; it’s evil because it's economically evil because it's so cheap that it's found its way into everything. It’s found its way into hamburger buns, pretzels, barbecue sauce and ketchup. Almost everything. Somebody e-mailed me the other day and told me they went into their local grocery store and went through every single loaf of bread on the shelf and

out of 32 types of bread on the shelf, only one of them did not have high fructose corn syrup in it.

So we are being poisoned by this stuff and it has been added surreptitiously to all of our food. Every processed food. And the question is why? Well, you will see why in a minute. So the core refiners like to point out well, you know it's just been a substitution. As the high fructose corn syrup has gone up, the sugar's gone down. We're just replacing like gram for gram. Well, not exactly because here's 73 pounds of sugar per year. This is from the Economic Research Service of the US Department of Agriculture. 73 pounds up to 95 pounds by 2000 and there is something missing from this slide. Juice is missing because juice is sucrose, right? And juice causes obesity.

So this is a study done by Miles Faith, a prospective study in inner-city Harlem toddlers, and the number of juice servings per day predicts the change in BMI score per month in these inner-city Harlem toddlers. Now where do these inner-city Harlem toddlers get their juice? From what, from where, from whom? From WIC. Anybody heard of WIC - Women, Infants and Children - a government entitlement program set up under Nixon to prevent failure to thrive. They did. This is the equal and opposite reaction.

So let's add juice in. And so most fructose items when you put it together, now we are up to 113 pounds on this graph, and I just heard from Brian Williams of NBC News after the most recent study came out, that was in the Journal of Clinical Investigation that we're actually up to 141 pounds of sugar per year. 141 pounds of sugar per year.

Now do you think that this might have some detrimental effects on you? It hasn't stopped you. That's why we need to talk about this. The juice consumption increases the risk for type 2 diabetes.

The third political storm. That swirling around to create this disaster, this mega typhoon. That thing that happened in 1982, the USDA, American Heart Association, American Medical Association all telling us we had to reduce our consumption of fat. Now why did they tell us that? To stop what? To stop heart disease, did we? No, we didn't. In fact, this worked the exact opposite. We've only created more.

Now how did this come to be? Why did they tell us to stop eating fat? Well in the early 1970s, we discovered something in our blood called LDL, low density lipoproteins. You've heard of that, right? Is it good or bad? Not so bad. We will talk about it. In the mid-1970s, we learned that dietary fat raised your LDL. So if dietary fat is A and LDL is B, we learned that A led to B. The dietary fat definitely increases your LDL, no argument. It's true.

And then finally in the late 1970s we learned that LDL correlated with cardiovascular disease. So let's call cardiovascular disease C, so we learned that B led to C. So the thought process, you know by some very smart nutritionists etc. said well if A leads to B and B leads to C, then A must lead to C, therefore no A, no C. This was the logic.

Now anybody see any problems with that logic? That's right, premise is incorrect. And I will tell you why the premise is incorrect, because this suggests that this is all transitive, but in fact, only the contrapositive is transitive. So it's not no A, no C, it's no C, no A. So the logic isn't even right. It's faulty logic here. So this doesn't work on any level.

So I am going to show you why this doesn't work but before I show you why it doesn't work, I am going to show you that this was a battle roiled back in the 1970s. This was not a simple thing. There were people lined up on both sides of the story. So this over here is a book. 1972 it came out, and it was called Pure, White and Deadly - it's all about sugar - written by a British physiologist, nutritionist, endocrinologist by the name of John Yudkin. Now I never knew John Yudkin, he's passed away. But I read this book about a year ago and without even knowing it I was a Yudkin acolyte. I was a Yudkin disciple. Every single thing that this man said in 1972 is the God's honest truth. And if you want to read a true prophecy you find this book, it's not easy to find but if you go find this book and I'm telling you every single thing this guy said has come to pass. It's astounding. I am in awe of this guy.

Seven Countries Study

But on the other side, we had this guy over here. His name was Ancel Keys. Ancel Keys was a Minnesota epidemiologist, very interested in the cause of cardiovascular disease and he

performed the first multivariate regression analysis without computers. This is where you take a whole lot of data. You normally would just run a few computer programs, but basically the object is to try to figure out what causes what and to try to factor out other things and determine what the contributions of various things all at once are to an outcome that you're looking for.

So he was interested in cardiovascular disease and so what he did was he did this study along with other people around the world called the Seven Countries Study. A very famous, front page of Time Magazine in 1980. So here is the data on the Seven Countries study. We have the US, Canada, Australia, England, Wales, Italy, Japan, and here is percent calories from fat on the X-axis and here we have coronary disease death rate on the Y axis and so you would say look at that, it's very obvious, isn't it? Sure. Percent calories from fat correlates very nicely with coronary disease. Except a one little problem. Japan and Italy? So how much sugar did they eat? Didn't I tell you the Japanese diet eliminates fructose. They never even had it till we brought it to them after World War II. Italy, aside from Xylitol, what else they got? They have a lot of pasta, it's a lot of glucose but no fructose. There is no sugar in the Italian diet other than the occasional sweet, which they moderate. They are very careful about moderating and they cost a lot.

But here we got England, Wales, Canada, Australia, US, we are sugarholics. Aren't we? We're also fatoholics. So in fact, the fat migrated with the sugar. So here this is from Keys's own work. Page 262, if you want to pick up the 500-page volume, and I am just going to read you the one paragraph that talks about this. "The fact that the incidence rate of coronary heart disease was significantly correlated with the average percentage of calories from sucrose in the diet is explained by the inter-correlation of sucrose with saturated fat". In other words, donuts, and wherever there was the fat, there was sucrose too, because these guys here eat doughnuts. Partial correlation analyses show that with saturated fat constant there was no significant correlation between dietary sucrose and the incidence of coronary heart disease.

Okay. When you do a multivariate linear regression analysis you have to do it both ways. You have to do holding fat constant showing the sucrose doesn't work and then you have to hold sucrose constant and show that fat still works. You see that anywhere. He didn't do it. He didn't do the thing that you need to do to do a multivariate linear regression analysis. Now this was done before computers. We can't check the work. He is dead. He died in 2004.

So we're left with a conundrum. Do we believe this? Do we believe this study because we based 30 years of nutrition education and information and policy in this country on this study? And as far as I'm concerned, it has a hole as big as the one in the USS Cole.

2 types of LDLs – Good guys & bad guys

Let's keep going. Remember I told you LDL may be not so bad. Well here's why, because there really isn't one LDL there are two – there are two LDLs. Here is one over here; it's called pattern A, or a large buoyant LDL. So everybody knows that LDL correlates with cardiovascular disease and that's true. And I am not going to argue that, that is true, but it's not this one - pattern A LDL. These guys are so light they are buoyant; they float. So they get carried through the bloodstream and they don't even have a chance because they are so big and they are so buoyant, they don't even get underneath the edge of the endothelial cells in the vasculature to start the plaque formation process.

But over here we have this other guy called pattern B or small dense LDL. You see the difference. These guys are dense, these guys don't float. These guys are small, they get underneath the edge of the surface of the endothelial cells and they start the plaque formation. And this has been shown by numerous investigators now: the small dense LDL is the bad guy. Okay, now when measure LDL in the bloodstream, when you do a lipid profile, you measure both of them together, because it's too hard to distinguish the two. So when you can get an LDL, you're getting both LDLs – the neutral one and the bad one.

Now how can you tell whether your LDL is the neutral one or the bad one? What you do is you look at your triglyceride level in association with it. Because the triglycerides tell you which one it is. So here is pattern A over here - big large buoyant LDLs. And you will notice that the triglycerides are low and your HDL is high. That's what you want; you want a low triglyceride, high HDL because that's the good cholesterol. You want high

good cholesterol.

Over here you have pattern B. Here you have high triglyceride, low HDL. That’s the bad buy. That's the guy you don't want to be, because you’re going to die of a heart attack. No question about it. Triglyceride to HDL ratio actually predicts cardiovascular disease way better than LDL ever did. Point is when you measure LDL you measure both.

So dietary fat raises your large buoyant. What do you think raises your small dense? Carbohydrate. So what did we do in 1982? We went on a high carb diet which was supposed to a low-fat diet. So here's the low-fat craze took America and the world by storm, because the content of low-fat home-cooked food that you cook by yourself in your house, you can control the content of fat. But when you process it -- low-fat processed food it tastes like cardboard. So the food companies knew that. So what did they do? They had to make it palatable. So how do you make something palatable that has no fat in it? You add the sugar. So everybody remember SnackWells. So 2 grams of fat down, 13 grams of carbohydrate up for them being sugar so that it was palatable. Well, we've just shown you that that's the worst thing you could do and that’s what we’ve done, and we’re still doing it today.

So when you find a mistake, what do you do? You admit the mistake and you right the shift. We haven’t admitted the mistake and we haven't righted the shift. So we've had our food supply adulterated, contaminated, poisoned, tainted on purpose and we’ve allowed it and we’ve let it through the addition of fructose for palatability, especially because they decrease fat but also as essentially browning agent which actually has its own issues, because why brown so well with the sugar in it actually is what's going on in your arteries, because that's causing what we call protein glycation and cross-linking, which is actually contributing to atherosclerosis. So it works on your steak on the grill; it works in your arteries the same way. And removal of fiber also.

Removal of fiber in diet

Now why did we remove fiber from our diet? We as human beings, walking the earth 50,000 years ago used to consume 100 grams to 300 grams of fiber per day. We now consume 12. Why? What did we do? We took the fiber out. So why did we take the fiber out? Well it takes too long to cut, takes too long to eat and shelf life. So people ask me what’s the definition of fast food – fiber less food. I dare you other than a salad, I dare you to go to any fast food restaurant and find anything on their menu that actually have to cook that has more than 1 gram of fiber in it, because there isn’t any. And that's on purpose, because they take the fiber out, because that way they can freeze it, ship it around the world and cook it up fast and not only it’s a fast cooking, but it's fast eaten which also causes us satiety issues. Bottom line we have a typhoon on our hands.

And then finally the last issue was the substitution of trans fats which are clearly a disaster. But those have been going down because we know that those are problems. So we've actually gotten rid of most trans fats, not completely, but most. So this is it. This is what we've done over the last 30 years.

Some Fructose Facts

Okay. Now to the biochemistry. How many of you here have taken biochemistry? About 25%. I am going to show you a lot of reactions in excruciating detail. If you've studied biochemistry you will have an anaphylactic reaction. If you haven't studied biochemistry, you will fall asleep. So what I am going to suggest that you all do is just let me do my thing to show you that at least it works and just count the arrows. Just let me do my thing and let me show you why fructose is not glucose. Because what the liver does to fructose is really unique, and you've got to understand that to understand everything I've just told you.

So number one - fructose is seven times more likely than glucose to do that browning reaction - the thing that happens on your grill happens in your arteries for the same reason. You can actually see the color change too. Fructose does not suppress the hunger hormone. There is a hormone that comes from your stomach called Ghrelin and you've heard about already. So if you pre-load a kid with a can of soda and then you let them loose at a fast food restaurant do they eat more or do they eat less? They eat more. They just took on 150 calories, yet they eat more. Reason: this fructose doesn't suppress the hunger hormone Ghrelin, so they eat more.

Acute fructose ingestion does not stimulate insulin, because there's no receptor for

fructose – no transport fructose on the beta cell that makes insulin. So the insulin doesn’t go up. Well if insulin doesn’t go up, then leptin doesn't go up and if leptin doesn't go up, your brain doesn't see that you ate something. Therefore you eat more.

And finally, I am going to show you liver hepatic fructose metabolism is completely different between fructose and glucose - completely different and I'm going to show you before the day is out that chronic fructose exposure alone - nothing else - causes this thing we call the metabolic syndrome. Everybody knows what the metabolic syndrome is. This is the conglomerate of the following different phenomena: obesity, type II diabetes, lipid problems, hypertension and cardiovascular disease. Those are all clustered together called the metabolic syndrome. I am going to show you how fructose does every one of us.

I want to show you the difference between glucose and fructose in a way that will be glaringly apparent. So let’s consume 120 calories in glucose, two slices of white bread. What happens to that 120 calories? Now you eat that 120 calories, 96% or 80% of the total will be used by all the organs in the body. 80% off the table. Why, because every cell in the body can use glucose. Every bacteria can use glucose. Every living thing on the face of the earth can use glucose because glucose is the energy of life. That's what we were supposed to eat.

24 of those calories or 20% will hit the liver. So let’s watch what happens to those 24 calories. Here they go. So the glucose comes in through this transporter called Glu2. Out here the glucose is going to stimulate the pancreas to make insulin. The insulin is going to bind to its receptor and it’s going to take this substrate over here called IRS-1 insulin receptor substrate 1, that’s not important right now, don't worry – and it’s going to tyrosine phosphorylated and this would be tyrosine IRS-1 which is now active. That’s going to stimulate the second messenger here called AKT.

Now what a AKT does is it stimulates this guy down here SREBP-1 - sterol receptor binding protein number one. This is the thing that ultimately gets fat mechanics going. So you will see in a minute. So one of the things SREBP-1 does is it activates this enzyme here called glucokinase, which takes glucose to glucose six phosphate. Now glucose six phosphate can’t get out of the liver. The only way to get glucose six phosphate out of the liver is with hormones - glucagon or epinephrine. That's the way you can get it out. So now the glucose is fixed in the cell but it’s only 24 calories worth. So it’s not a big bowls of it.

Now the glucose six phosphate, almost all of it is going to end up going over here to something called glycogen. Now glycogen is the storage form of glucose in the liver; because glycogen is easy to fish the glucose out with glucagon and epinephrine. So my question to you - granted this is a physiology question - is how much glycogen can your liver store before it gets sick? The answer is any amount. Unlimited. We have carb loaders who run marathons. We have kids with the disease where they can’t get the glucose out of the glycogen called glycogen storage disease type 1a or von Gierke's disease. They got livers down to their knees that are so big, their hypoglycemic like all get out because they can't lift the glucose out of their liver. But they don’t go into liver failure. Because glycogen is a non-toxic storage form of glucose in the liver. So the whole goal of glucose is to replete your glycogen. So this is good; this is not bad. This is good.

Now a little of that glucose is going to fall down here, it’s going to get metabolize down to the stuff here called pyruvate and the pyruvate is going to enter your mitochondria. You might remember mitochondria are the parts of your cell that actually burn the energy. They make the stuff that lets you live called ATP - ATP adenosine triphosphate; that’s the energy of life. So the pyruvate comes in, gets converted to something called acetyl CoA, gets metabolized by this called the Kreb's Cycle, TCA cycle and you throw up ATP and carbon dioxide which you breathe off.

So this stuff over here acetyl CoA gets burned off in the TCA cycle. Now maybe you won't burn all of it off. And so some of it may exit as citrate, and the citrate then leaves the mitochondria through a process known as the citrate shuttle and then that citrate can then be broken down by these three enzymes, which are all subservient to this SREBP-1. This is ATP citrate lyase, acetyl-CoA carboxylase, fatty acid synthase. They are not important. The only thing to know is these three enzymes together turn sugar into fat. This is called de novo – meaning new – lipogenesis - fat making - this is de novo

lipogenesis. So you take the citrate which came from the glucose, and you end up with something called Acetyl-CoA, which then gets packaged with this protein here and you end up with something called VLDL - very low density lipoprotein. Is it good or bad? Bad. VLDL is bad because that's one of the things that causes heart disease. It's also a substrate for obesity.

So you don't want to make much of this. But the point is you started with 24 calories, maybe a half a calorie will end up as VLDL. So that little Japanese guy with a little hat, working out in the field, eating rice for the next 90 years, can he die of a heart attack at age 90? Sure, but that's not so that. If you make it to 90, you are doing all right because of that VLDL coming from glucose. Glucose made a little bit VLDL. That serves as a substrate for adipose deposition into your fat cell here triglyceride. In addition, because the insulin went up in response to the glucose, your brain sees that signal and it knows that that is supposed to shut off further eating. In other words, hey, I am busy metabolizing my breakfast; I don't need lunch. And so you have a nice negative feedback loop between glucose consumption, the liver, the pancreas and brain to keep you in normal negative yin yang energy balance. This is good. This is not dangerous. This is what's supposed to happen.

Ethanol is also a carbohydrate

So let's now talk about a different carbohydrate. Let's talk about my favorite carbohydrate. Maybe yours too. Ethanol. Ethanol is a carbohydrate. Here is the structure: carbon, hydrogen, oxygen. It's a carbohydrate, but we all know that ethanol is a toxin, a poison.

So here is acute ethanol exposure. CNS depression, vasodilatation, hyperthermia, tachycardia, myocardial depression… here is fructose, nothing – doesn't do any of those because the brain doesn't metabolize fructose. Alcohol get metabolized in the brain to cause all of those things but fructose doesn't. So fructose is not an acute toxin, ethanol is. We control ethanol, don't we? We have something called Bureau of Alcohol, Tobacco and Firearms. We have all sorts of things, we tax ethanol. We do all sorts of things to limit consumption of ethanol. The Nordic countries, all the liquor stores are state run in an attempt to try to set the price of ethanol high enough so as to discourage consumption for public health reasons. We have 1500 years of alcohol control policy in this world to draw on in terms of how to limit consumption, because ethanol is a toxin, and we know it.

So let's take 120 calories and ethanol. So 24 calories right off the top. The stomach and intestine have something called the first pass effect, so 10% off the top and kidney muscle and brain will consume the other 10%. So there goes 20% or 24 calories right off the top. 96 calories of the 120 calories are going to the liver. Now how many was it for glucose? It was 24 calories. So four times the substrate is going to hit the liver and there's the rub. This is a volume issue. We're going to show you how.

So the ethanol comes in - passive diffusion, there's no receptor for it, no transporter. First thing that happens is ethanol gets converted to this guy over here called acetaldehyde. Anybody know anything about aldehydes? Like formaldehyde. Are aldehydes good for you or bad for you? They are bad. Because what do they do – the can cause cancer. They cross-link proteins is what they do. So if you cross link enough proteins in your liver, what do you think happens to it? You get something called cirrhosis. So this guy over here is bad and it generates something called reactive oxygen species (ROS).

Reactive oxygen species damage proteins in the liver and the more alcohol you drink the more of this stuff you get. So this is one of the reasons why alcohol is bad. Now acetaldehyde will come down here to something called acetate. Acetate will enter the mitochondria just like pyruvate did before, will get converted to acetyl-CoA and participate in the TCA cycle just like before to generate energy so that alcoholics don't die from lack of energy; they got energy. Everything else they don't have. They are going to have a whole lot of citrate, because they have 96 calories that have to get metabolized. How many calories made it to the mitochondria with glucose? About half, because most of it went to glycogen. So we've got a big citrate. And so the big citrate is going to get metabolized all the way to VLDL by the SREBP-1. So you're going to get a lot of VLDL, and this is the dyslipidemia of alcoholism.

So the liver is going to try to export this VLDL out so that it doesn't get sick, because

when fat builds up in the liver that's not good for you. Some of it is going to exit as free fatty acids and these free fatty acids will take up residence in the muscle and you get something called muscle insulin resistance. So insulin resistance, that's a bad thing, that makes your muscles and your liver not work so well and can cause all sorts of other problems like diabetes.

Some of the acetyl-CoA won't even make it out and will precipitate as a lipid droplet. So there's your alcoholic steatohepatitis. This acetyl-CoA and this ethanol and this reactive oxygen species can start this enzyme and activate it. It's called c-jun-N-Terminal Kinase 1 or JNK 1 and it really is junk 1 because it is the bridge between metabolism and inflammation. So when you generate JNK 1 you do bad things to your liver, which I'll show you when we talk about fructose.

So let's talk about fructose. Fructose is sweet. We like it a lot. We like it in everything. We like it in our bread, we like it in our pretzels, we like it everywhere we look. So let's consume 120 calories in sucrose, a glass of orange juice. So two slices of white bread, a sodar Maker's Mark, a glass of orange juice, all the same 120 calories but three different substrates. Let's see what happens to the fructose?

So number one - glucose - remember sucrose is half, glucose half fructose. So 60 of the calories of the 120 calories are glucose, 12 calories are going to make it into the liver, 48 calories out here for the rest of the body. So the same 20:80 split we had before with glucose. But all 60 calories of fructose are going to be metabolized by the liver. Why? Because only the liver can metabolize fructose. So what do we call it where when you take in a compound that's foreign to your body and only the liver can metabolize it and in the process generates various problems, what do we call that? We call that a poison. So let me show you how it's a poison.

So let's watch the fructose. So the fructose comes in through this transporter now – before it was Glu2, now it's Glu5 – no insulin, remember because fructose does not stimulate insulin. Fructose then gets metabolized by this guy over here called fructokinase to form something called fructose 1-phosphate. In the process ATP has to give up 1-phosphate to ADP because the phosphate has to come from somewhere, so comes from here.

Now before we had 24 calories that had to be phosphorylated, now we have 72 calories that have to be phosphorylated. So we have three times the substrate and there's the rub. It's a volume issue for right now. so we are going to lose a lot of phosphate, aren't we? So there is a scavenger enzyme in your liver called AMP Deaminase 1 [Adenosine monophosphate deaminase] to rescue the phosphates off the rest of the ATP molecule and that takes ADP down to AMP adenosine monophosphate down to IMP Inositol monophosphate and finally to the waste product uric acid. Anybody ever heard of uric acid? What is it? It's a waste product, goes out in your urine, causes what disease? Gout. It also causes another disease called hypertension. Let me show you how.

Because uric acid - it turns out - blocks the enzyme in your blood vessels called endothelial nitric oxide synthase, and that's the enzyme that makes the stuff called nitric oxide NO and that is your endogenous blood pressure lowerer. That keeps your blood pressure low. So when you can't make it your blood pressure goes up. So this just shows that fructose consumption increases gout in adults. This is a study that came out last year showing that fructose consumption increases the risk for gout showing that uric acid is going up. This is a study done by our pediatric renal fellow Stephanie Winn just published in Journal of Pediatrics, showing that this is in [Anne Haynes] database in the adolescents showing that sugar sweetened beverages as it goes up, your uric acid goes up, and not only does your uric acid go up, but here is your sugar sweetened beverages and here is your systolic blood pressure going up.

Now here is a study done by [Dan Pack] at the University Texas, San Antonio where he took obese adolescents with hypertension and he gave them the drug allopurinol and allopurinol is the drug that you treat gout with to lower the uric acid and look what happened to the blood pressure systolic diastolic went down, showing that in fact, uric acid is an important part of hypertension. We have a hypertension epidemic in this country. Here it is – it's the sugar.

The fructose will get metabolized down to pyruvate. The pyruvate will enter the mitochondria just like before throwing off a lot of citrate and here is a little trick,

that fructose does that glucose doesn't. Because these two can reform this stuff over here called fructose-1, 6-bisphosphate, which can then reform with glyceraldehyde to form this guy over here called xylulose 5-phosphate and I will get to xylulose 5-phosphate in a moment. But I want to point out this asterisk – that's there to remind me to tell you something - that's there to remind me to tell you that this is why the sports drink companies put high fructose corn syrup in the sports drinks, because if you are glycogen depleted – in other words, if you just ran a marathon and you have no glycogen left in your liver because you burned it all, and you've taken a sports drink with high fructose corn syrup, you can replete your glycogen faster than with glucose alone. That's true.

So for elite athletes a high fructose corn syrup containing sports drink actually makes sense, and so indeed sports drinks have high fructose corn syrup. The question is who is drinking the sports drinks? Any elite athletes you know – who is drinking the sports drinks? The kids. Why are they drinking it? Because it's cool right, because it's cool and it tastes good. Okay so before we go on, I'll just want to now digress for a moment.

1967, University of Florida patents Gatorade. 1970, the Florida gators win the NCAA Championship in football. Gatorade makes a big splash. Big deal. Anybody ever tastes the original Gatorade? What does it taste like? It tasted horrible. It tasted like something that you might find coming out of you instead going into you. It tasted awful.

1992, Pepsi buys Gatorade and they say how we're going to market this well? So what did they do? They added the high fructose corn syrup. So now who drinks it? Right, fat kids. Not even skinny kids. Fat kids drink it. So there is a problem here. I am going to show you how that works.

So xylulose 5-phosphate just to show you – so if you take a rat and you glycogen deplete him by making him run on an exercise wheel and then you re-feed them with starch or with sucrose, the xylulose 5-phosphate goes way up with the sucrose. So you get more xylulose 5-phosphate through this pathway here going over here. So why do we care about xylulose 5-phosphate? Well here is why, because it stimulates this guy over here called PP2A which then activates this transcription factor here - carbohydrate response element binding protein, which then activates what three enzymes, new fat making, de novo lipogenesis. So here is the citrate, we got lots of that and how – here we've got acetyl COA, which is the way into fat which then gets packaged to the VLDL and now you've got the dyslipidemia of obesity of fructose consumption, which has been known for many years.

So here's normal medical students, if you call them normal, taking in a glucose load - almost none of it ends up as fat. Taking in a fructose load, same number of calories, 30% of it ends up as fat. So when you consume fructose you're not consuming the carbohydrate; you're consuming fat. So everybody talks about a high-fat diet, well high sugar diet is a high-fat diet. That's the point. That's exactly the point.

This is a study where they gave acute administration of fructose and you can see the triglycerides going up compared to the controlled. Here is normal medical students again six days of high fructose feeding, triglycerides doubled, de novo lipogenesis went up five times higher and here's free fatty acids, which then cause insulin resistance doubled - six days. So here's the dyslipidemia of fructose consumption, we are not done. If some of the fat won't make it out of liver just like with ethanol, and now you've got a lipid droplets, now you've got this nonalcoholic steatohepatitis. So this is the work that we did in our clinic looking at sugar sweetened beverage consumption against the liver enzyme marker ALT, alanine aminotransferase, which tells you about fatty liver, and sure enough here's sugar sweetened beverages against ALT and you can see a nice linear relationship in Caucasians. African-Americans, it's a different relationship, and that's a whole another story all by itself.

So there is the lipid droplet of nonalcoholic steatohepatitis. Some of it will come out a free fatty acids and populate the muscle, will also tell the insulin to go up higher. Remember that JNK-1. So here is what JNK-1 does. So the acetyl COA and the fructose can all activate JNK-1 and what JNK-1 does is remember – when we use glucose, this IRS became tyrosine IRS-1 and that was good. Well JNK-1, what it does is it's serine phosphorylates IRS-1 and serine IRS-1 is inactive. So now the insulin can't even do its job in the liver. So now you have liver insulin resistance as well. That's going to make the pancreas work that much harder generating higher insulin levels which raise your blood pressure even further, cause further fat making, cause more energy to go into your fat cell - there's

your obesity – and finally our research has shown that the higher the insulin goes less well your brain can see it's leptin. And so there you've got continued consumption because your brain thinks it's starving and it's been shown on many different ways that fructose consumption changes the way your brain recognizes energy, all in a negative fashion so that you basically think you're starving. Your brain gets the signal that you're starving even though your fat cells are generating a signal that says hey, I'm full like all get out.

So the high insulin generates the obesity because this is that – remember the first law of thermodynamics - the biochemical force generating the energy storage as the primary phenomenon. If you're going to store it and you expect to burn it, then you're going to have to eat it. So here's the store it. Normally that would make leptin and the leptin should feed back and turn everything off but it doesn't because the insulin gets in the way and high-fat diet gets in the way. Also hyperinsulinemia stops the the leptin from acting on that nucleus accumbens and so you get an increased reward signal so that continues your appetite, continues more fructose, more carbohydrate, generating more insulin resistance and you can see you generate a vicious cycle of consumption and disease and no stopping.

So here we are hypertension, inflammation, hepatic insulin resistance, hyperinsulinemia, dyslipidemia, muscle insulin resistance, obesity and continued consumption. Looks like metabolic syndrome to me. So here are the phenomena associated with chronic ethanol exposure. Hematologic disorders, electrolyte abnormality, hypertension……here is fructose. 8 out of 12. Why? Because they do the same thing, because they metabolize the same way, because they are the same, they come from the same place. How do you make ethanol naturally? Right, you ferment sugar; hasn't changed, because it has all the same properties because it's basically taken care of by the liver in exactly the same way and for the same reason, because sugar and ethanol are the same. Every which way you turn.

So here's our clinic intervention. This is what we do in our clinic. It's as simple as you can imagine. We write this on the back of a matchbook - it's just as simple as you can make it. We have four things we teach the kids to do and the parents. Get rid of every sugar liquid in the house; bar none. Only water and milk. There is no such thing as a good sugar beverage. Period. Eat your carbohydrate with fiber. Why? Because fiber is good. Fiber is supposed to be an essential nutrient.

We follow our patients every three months. So here is my question – does it work? Yes, it worked. This is BMIG score time from initial visit; that works. We were interested in what made it work and what made it didn't work, so we did a multivariate linear regression analysis – the thing that made it not work. Sugared beverage consumption. The more sugared beverages the patients drank at baseline, the less well a lifestyle intervention worked for all the reasons I just showed you.

Why is exercise important in obesity?

So why is exercise important in obesity? Because it burns calories. Come on - 20 minutes of joggings, one chocolate chip cookie. You can't do it. So why is exercise important? I will tell you why, here is why. Number one, it improves that skeletal muscle insulin sensitivity, because your insulin actually works better at your muscle, which then brings your insulin levels down which is good for you. Number two, it's your endogenous stress reducer; it's the single thing that actually stress reduces. If you stress reduce what do you think your appetite does? Goes down, because stress and obesity go hand-in-hand for all sorts of reasons which are beyond the scope of this lecture today. And then finally, remember that de novo lipogenesis, remember those three nasty enzymes.

What if you've burned the stuff off before you made the fat? That's what exercise does, because it makes that TCA cycle run faster, so you don't get the citrate leaving the mitochondria, so it doesn't get turned into fat so it doesn't precipitate and cause all the problems you just saw. That's what they mean by a higher metabolism, yes. But it has nothing to do with burning of calories. That is the stupidest reason that I've ever heard of for exercise. You got to be joking me. You can't do it. I mean one big Mac, you know you got a mountain bike for 10 hours, are you joking?

Why is fiber important in obesity?

So why is fiber important in obesity? So this is my motto in clinic. When God made the poison he packaged it with the antidote. Because fructose is a poison, I think I've

hopefully shown you that. But wherever there's fructose in nature, there is way more fiber. There you see a piece of sugarcane, it’s a stick. You can’t even chew the damn thing. You have to suck the stuff out like that right. I mean how many calories you think you’re going to get out of a piece of sugarcane. They actually did studies on the sugar plantations back in the early 1900s. All of the workers were healthy and lived longer than the sugar executives who got processed product. How about that? Wonder why?

So eat your carbohydrate with fiber; that's why we say that. That includes sugar, that's why fruits are okay, because number one, it limits how much fructose you are going to take in, and number two, it gives you an essential nutrient which you needed in the first place and you get some micronutrients along with it so that you actually can – your liver works healthier. So here is what fiber does. Number one, it reduces the rate of intestinal carbohydrate absorption. Now sometimes that's bad. I will tell you when that's bad. That’s bad when you're at a formal function. Because what happens if you reduce the rate of carbohydrate absorption in your gut, the bacteria get to it. As far as I'm concerned in life you've got two choices – it’s either fat or fart. It increases the speed of transit of the intestinal contents to the ilium and that raises this hormone over here called PYY which goes to your brain and tells you the meal is over; that's your satiety signal. So when you add fiber to your diet, you actually get your satiety signal sooner because the food moves through faster. And then finally it also inhibits the absorption of some free fatty acids all the way to the colon and then those get chopped up into little itty bitty fragments called short chain fatty acids and those actually suppress insulin as opposed to long chain fatty acids which stimulate insulin. So there are all whole bunch of reasons why fiber is good.

Anybody ever heard of the Paleolithic diet? Go home and read up on it on the Internet. The Paleolithic diet - basically if you read everything as it came out of the ground off with no cooking, you would cure diabetes on a dime. It takes about a week, because you’re getting that 100 to 300 grams of fiber I mentioned before. Because fiber is good for you, and the more the better – type 2, not type 1.

Fructosification of America

So now comes the fun part - the racial innuendos and all the political stuff - the fructosification of America and of course the world. Another quiz. Can you name the seven food stuffs at McDonald's that don't have high fructose corn syrup or sucrose? Hash browns, for the same reason, it’s all starch and fat. Chicken McNuggets, I was shocked - no sucrose or high fructose corn syrup in Chicken McNuggets. But as the of circuit court judge in New York called them, they are still a McFrankenstein creation but nonetheless no fructose. Sausage – there is no fructose. Diet coke, coffee if you don’t add the sugar, and iced tea if you don’t add the sugar. By the way, the Chicken McNuggets have a disclaimer – because no one eats the Chicken McNuggets without the dipping sauce. And there's a whole bunch of high fructose corn syrup in the dipping sauce.

So who is really drinking this? We talked about this before, Gatorade AM. So this is an attempt by Pepsi to capture market share on the juice market. Do you think there are any elite athletes who actually drink this stuff? You’ve got to be kidding me. Okay, this is for kids. So this really blew my socks off. This was my daughter when she was in second grade 2 years ago Mariam Lustiq, brought these two cartons of milk home for me and said, “Dad, you’re not going to believe this?” Second grade. So here is the calories in Berkeley Farms 1% low-fat milk 130 calories, 15 of them are sugar, because it’s lactose, which is okay. And here's Berkeley Farms 1% chocolate milk, 190 calories, 29 g of sugar all fructose corn syrup. It’s like a glass of milk plus a half a glass of orange juice, and that’s what we’re giving to our kids.

What about WIC? We talked about 112 pounds of orange juice that the kid down in Salinas was drinking, what about WIC? So remember what we started with. We have an epidemic of obese six month olds. So could this be the reason? So here's a can of formula, 43.2% corn syrup solids, 10.3% sugar. It’s up baby milkshake. Soda, Coca-Cola is 10.5% sucrose, formula is 10.3% sucrose, any difference? And there is a huge literature that's now coming of age that shows that the earlier you expose kids to sweet, the more they're going to crave it later. Plus there's a new literature that shows the more sugar the pregnant woman drinks or eats during the pregnancy the more that gets across the placenta and actually causes what we call developmental programming changing the kid’s adiposity

even before the kid is born and driving this whole epidemic even further.

So we will close in a few minutes. But I just want to point out what's the difference. Here we've got a can of Coke, here we've got a can of beer. I'm not picking on Schlitz or anything, any beer you want. So 150 calories each, no difference in terms of total calories; percent carbohydrate 10.5% from sucrose here, except this high fructose corn syrup but who cares - 3.6% alcohol. Here's the breakdown: 75 fructose, 75 glucose for the Coke. 90 alcohol, 60 maltose for the beer. Remember the first pass GI metabolism takes 10% of the alcohol off the table. So when you actually compute the number of calories hitting the liver, which remember was the big difference between glucose and fructose - remember 72 versus 24 and started the whole thing into motion as terms of what happens that's bad – bottom line no difference.

So we have something called beer belly. Well, welcome to soda belly that's what America is suffering from - no ifs, ands or buts, that's what it is. Now you wouldn't think twice about not giving your kid a Budweiser but you don't think twice about giving your kid a can of Coke? But they are the same. In the same dosing, for the same reason, through the same mechanism, fructose is ethanol without the buzz. So fructose is a carbohydrate. Yes, it is. The fructose is metabolized like a fat and I have just shown you that 30% of any ingested fructose load ends up as fat. So when people talk about high-fat diets doing bad things – no, what they're really talking about is high fructose diet, and that's what Ancel Keys was looking at.

So the corollary to that is in America at least and around the world too, a low-fat diet isn't really a low-fat diet, because the fructose or sucrose doubles its fat, it's really a high-fat diet. That's why our diets don't work and fructose just like ethanol, for the same reason through the same mechanism and the same dosing is also a toxin.

Last, what can we do about it? Can we do anything about it? How about the FDA? Do you think they can do something about it? After all, aren't they supposed to regulate our food? Aren't they supposed to regulate what they can put in food? Weren't they supposed to regulate tobacco? Now they are actually.

So I want to just show you what the tobacco company thinks of all this? So this is actually from the US UCSF Legacy Tobacco Documents Library that Stan Glantz runs right across the street. Stan is a good guy. And he showed me – "Under the regulations governing food additives, it is required the additives be safe, defined as reasonable certainty by competent scientist that no harm will result from the intended use of the active." Now does fructose meet that standard? Well the FDA says that fructose – high fructose corn syrup has what's known as GRAS status - generally regarded as safe. Now where did that come from? Nowhere. It came from nowhere. It came from the notion that well, fructose is natural, it's in fruit, must be okay. Yes, well tobacco is natural too but it's not. Ethanol is natural, but it's not.

Keeping ongoing – "a food shall be deemed to be adulterated if it bears or contains any poisonous or deleterious substance which may render injurious to health". Fructose fits that description but not with the prevention of chronic diseases even though its own regulations explicitly postulate a connection between such products and such diseases. In other words, FDA will only regulate acute toxins, not a chronic toxin. Fructose is a chronic toxin. Acute fructose exposure did nothing, because the brain doesn't metabolize fructose. The liver does. And the liver doesn't get sick after one fructose meal. It gets sick after a thousand fructose meals. But that's how many we eat.

So the FDA isn't touching this. The USDA isn't touching this, because if the USDA touched this, what would that mean? That would mean an admission to the world that our food is a problem. So what do you think that would do? There are three things in this country that we can still sell overseas: weapons, entertainment and food. Cars? Computers? I don't think so. I mean can anybody think of anything else that another country wants of ours? All right you get the picture. So the USDA doesn't want to know about this, because this is bad news. And so who runs the food pyramid? The USDA – it's the fox in charge of the henhouse, because their job is to sell food. And who is eating it? we are. So

Conclusion

So in summary, fructose and I don't care what the vehicle is, it's irrelevant, sucrose or high fructose corn syrup I don't care – fructose consumption has increased in the past 30 years coinciding with the obesity epidemic. A calorie is not a calorie, and the

dietitians in this country are actually perpetrating this on us, because the more you think a calorie is a calorie, the more you think, well then if you ate less and exercise more it would work. It doesn't. All of these studies show it doesn't work. Here is why it doesn't work because a calorie is not a calorie. Fructose is not glucose. We know a calorie is not a calorie because there are good fats and bad fats. There's good protein and bad protein. There's carbohydrate and bat carbohydrate. And glucose is good carbohydrate. Glucose is the energy of life. Fructose is poison. You are not what you eat; you are what you do with what you eat and what you do with fructose is particularly egregious and dangerous.

Hepatic fructose metabolism leads to all the manifestations of the metabolic syndrome – hypertension through that uric acid pathway; de novo lipogensis, dyslipidemia, hepatic steatosis through that DNL pathway, those three enzymes, the new fat making pathway; inflammation through JNK-1, hepatic insulin resistance because of the serine phosphorylation of IRS-1; obesity because of the VLDL transport to the adipocyte and leptin resistance promoting continuous consumption, basically starving your brain, making you think you need more. Fructose ingestion interferes with obesity intervention as we showed in our clinic. The more soft drinks, the less well diet and exercise actually worked.

Fructose is a chronic hepatotoxin for the same reason that alcohol is. The only difference is alcohol is metabolized by the brain, so you get alcohol effects. Fructose is not metabolized by the brain, so you don't get those effects, but everything else it does is the same. But the FDA can't and won't regulate it. It’s up to us. I'm standing here today to recruit you. That’s a famous saying here in San Francisco, right?