THE HOMESTEAD
RATHBURNIE ESTATE NATURE REFUGE,
1166 MT.STANLEY ROAD
KINVILLE. Q. 4306.
Upper BrisbaneRiver Valley.

10th June, 2008.

The Chairman,
Senate Committee on Alcopops Legislation,
Parliament House,
CANBERRA.
A.C.T.

Dear Committee Members,

SUBMISSION.

Accompanying my recent Submission to the 2020 Summit on A LONG-TERM NATIONAL HEALTH STRATEGY - INCLUDING THE CHALLENGES OF PREVENTATIVE HEALTH, wherein in Section (a) Items 6, 7 and 8, I stresssd the strategies necessary to prevent chronic health problems and their ultimate high costs to the community and associated health provisions,

is the enclosed TRANSCRIPT ON "THE OBESITY EPIDEMIC" of a Health Report broadcast by A.B.C. Radio National on 9th July, 2007 and 3rd Decembe , 2007, which I suggest be photocopied and made available to all Committee Members for perusal.

Whilst the proposed impost on Alcohol Pops is being described as a control on Binge drinking and its resultant outcomes, I urge Committee Members to peruse this Transcript which shows the HEALTH HAZARDS science behind the addition of soft drinks or fruit juices to diets - including the addition of these components to alco-pops.

(Mrs.)V.D.BURNETT.
TRUSTEE - ESTATE G.C.BURNETT (DECD.)
(A.I.F. CORPS SIGNALS QX.898)

Encl. Submission to 2020 Summit.
Transcript "The Obesity Epidemic". A.BC. Radio National Health Report.

Mrs.V.D.Burnett (aged 80)

SUMMIT TOPICS FOR SUBMISSIONS # (please limit comments to 500 words per topic)

A long-term national health strategy – including the challenges of preventative health, workforce planning and the ageing population

The *Australia 2020* Summit will examine:

a. How we invest to help prevent chronic and acute health problems

b. How we plan to ensure all Australians continue to have access to the very best of modern medical technology including pharmaceuticals

c. How we meet the emerging regulatory challenges of modern medical technology

d. The use of electronic infrastructure to facilitate efficient and effective patient care

e. Strategies to preserve Australia's internationally unique blend of public and private health services

f. How Australia best plans for the future demands on our medical workforce.

(a) HOW WE INVEST TO HELP PREVENT CHRONIC AND ACUTE HEALTH PROBLEMS.

1. Ban the addition of **FRUCTOSE** to food and drinks to control its effects on obesity, diabetes and liver problems. (*)

2. Ban the use of **TRANS-FAT** (transformed fat) added to food and cooking oils for longer shelf-life, which causes damage to good and bad cholesterol. (**)

3. Impose a consumption tax on toasted and sweetened breakfast cereals and yoghurt thereby encouraging natural Muesli and natural yoghurt. Also a consumption tax on refined white bread to encourage wholemeal and whole grain breads to reduce incidence of diverticulitis and colon cancer.

4. Restore provision of free milk to all school children to provide calcium for brain and bone development.

5. Ensure school children receive a free nutritious lunch with education about the dangers of snack foods and soft drinks containing trans-fat and fructose.

6. Restrict drivers under the age of 21 to single occupancy of vehicles – peer group pressure as well as drugs and alcohol cause loss of young lives the country can ill afford.

7. Close liquor outlets at midnight.

8. Impose high taxes on alcohol to reduce consumption.

References:

(*) ABC Medical Report, 8.30am Radio National

(**) Harvard Medical School Report on web.

"The Obesity Report" – Dr Robert Lustig – Professor of Pediatric Endocrinology – University of California, SAN FRANCISCO.

ABC Radio National Health Report http://www.abc.net.au/rn/health report/stories/2007/2104024.htm

"Trans-fat" - Harvard School of Medicine hhtp://www.hsph.harvard.edu/nutritionsource/fats.hmtl

V.D. Burnett

The Health Report

on ABC Radio National

The obesity epidemic

3 December 2007

A researcher in the United States claims that the reason for the obesity epidemic is more than just the calories we eat and the lack of exercise. It's a substance that food manufacturers are widely using.

This program is a repeat and was first broadcast on 9th July, 2007.

Hide Transcript

Transcript

This transcript was typed from a recording of the program. The ABC cannot guarantee its complete accuracy because of the possibility of mishearing and occasional difficulty in identifying speakers.

Norman Swan: Good morning and welcome to the Health Report. Today another chance to hear a controversial program we played a little while ago. It's about a theory with scientific evidence behind it as to why the obesity epidemic is perhaps worse than it should be. The food industry, especially in the United States, hates the message you're about to hear and while certain manufacturing practices mentioned don't happen in Australia, as you'll hear, some in fact do.

The question is whether there's stuff in our food which makes us even fatter than our calorie excess would suggest. It's about how a carbohydrate may be behaving like a dietary fat. One of the key people pushing this idea is Dr Robert Lustig who's Professor of Pediatric Endocrinology at the University of California, San Francisco.

Robert Lustig: I'm very interested in what's happened over the last 30 years that has fomented this obesity epidemic. And of course everyone says well, that's because you're eating too much, and you're exercising too little and of course that's true. But the question is what about our physiology allows this to happen, we have some built-in negative feedback mechanisms that are supposed to stop us from gaining too much weight but clearly they are not working. The question I've been interested in now for the last ten years is what is actually blocking that signal to the brain to tell our bodies to eat less and exercise more? Clearly something is getting in its way.

Over the course of those ten years I've done numerous experiments in people and have come up with the notion that this is actually one of the main functions of the hormone insulin. Insulin's job is to store energy, insulin's job is to make you gain weight.

Norman Swan: Transports sugar from the blood into cells.

Robert Lustig: Exactly, that's insulin's job. Let's take a diabetic off the street, blood sugar is 300 --

in Australian terms that would probably be something in the order of 15. We give them a shot of insulin, the blood sugar goes down to 100, that would be something like 4 or 5, the question is where did the sugar go? It went to the fat for storage. That's insulin's job, insulin's job is to take sugar from the blood and put it into fat for storage, more insulin -- more fat. Well all these kids who are walking around who are massively obese now have extraordinarily high insulins. The question is when your insulin is high and you're storing energy you make another hormone, and that hormone is called leptin, and leptin is supposed to go to your brain and tell your brain that you've eaten enough.

Norman Swan: It's produced by fat cells in fact.

Robert Lustig: That's right, it's produced by fat cells, it circulates in the bloodstream, binds to specific receptors in the hypothalamus, the area of the brain that controls energy balance and it's supposed to turn eating off. In addition it also raises the tone of an area of your brain called the sympathetic nervous system which is designed to actually help you burn energy. So by reducing food intake and by increasing the burning of energy you're supposed to stay in balance -- but clearly these kids are not in balance.

So the question is, could insulin actually be interfering with that leptin signal, and that's what we've ultimately shown by actually suppressing insulin with a drug.

Norman Swan: So what have you done?

Robert Lustig: We took these kids who developed massive obesity after brain tumours; these kids have a tumour in the area of the brain which controls energy balance, the most common of which is called a cranial pharyngioma, and once these kids are treated, that area of the brain is now dead, it cannot see leptin. When you can't see leptin your brain is starving, and so what it does is it increases your food intake because you need to eat more -- even though there's plenty of leptin, you can't see it, so it's like it wasn't there -- and it also reduces your sympathetic nervous system in order to actually make you feel lousy and to burn less energy.

Because energy expenditure, energy burning and quality of life are the same thing, anything that raises your energy expenditure makes you feel good; for instance coffee, for two hours, and then you need another one. Anything that reduces your energy expenditure, like for instance hypo thyroidism as an example, makes you feel lousy. So when you can't see your leptin your brain thinks you're starving, you feel crappy, you certainly don't want to exercise and you're going to eat more.

So we see these children with brain tumours who can't see their leptin and we asked the question -- could we somehow influence this disastrous feedback cycle? What we did is we gave a drug called Octreotide and we knocked down their insulin levels with this medicine and all of a sudden, not only did these kids stop eating, they started exercising spontaneously, they just did it. Two kids started lifting weights at home, one kid became a competitive swimmer, one kid became a manager of his high school basketball team, running around collecting all the basket balls.

Norman Swan: So you're postulating that insulin was having an influence on the brain itself.

Robert Lustig: Right, by getting the insulin down instead of the energy that they were eating being forced to fat, the energy that they were eating could now be burned by muscle, could now be burned by the rest of the body, made them feel better.

Norman Swan: It could help to explain why leptin has been such a disappointing hormone, that in fact it's much more complicated than leptin, if only it was just leptin but in fact leptin's pretty lousy at controlling appetite itself, even when you've got it.

Robert Lustig: That's right, all the studies giving leptin to obese people have basically been failures, and the reason is because you have this thing called leptin-resistance, you can't see your leptin. If you could see your leptin you wouldn't be fat, in fact leptin-resistance and obesity are actually the same thing. So the question is, what causes the leptin- resistance, what causes you to not be able to see your leptin? Well these brain tumour kids, we know what it is, they've got death of that area of the brain. The question is what's wrong with the rest of us?

What we did was we actually dropped insulin in otherwise normal, healthy obese people, using the same mechanism.

Norman Swan: Adults or children?

Robert Lustig: This was adults, and we ended up with the same answer, we were able to get them to stop eating, in fact they stopped eating carbohydrate on a dime, they went from 900 calories a day in carbohydrate intake to 350 calories a day in carbohydrate intake, they stopped snacking between meals, they stopped drinking soft drinks. We didn't tell them to do this, they just did it, they didn't need to do it. Their insulins went down, they felt better, they started exercising and they lost weight and continued and kept losing weight.

If you look at all of the drugs that are out on the market today, they all cause some weight loss and then at the four month time point that's it, you can't lose any more, you hit the negative plateau and you can't go any further. And the reason that you hit this negative plateau is because your leptin has finally gotten down to a point where your brain is now starving. When we got the insulin down not only did the leptin keep going down but it kept going down even further. They kept losing more weight, they kept feeling better, they kept exercising and we were able to not have a negative plateau, we lost even more weight over the course of the year.

Norman Swan: So how come anti-diabetic drugs which effectively do that, they reduce insulin-resistance, get your insulin levels down -- how come they don't universally cause weight loss, in fact some of them can cause weight gain?

Robert Lustig: It depends on which one, in fact Metformin is an insulin sensitiser, it does get insulin levels down and we've shown that it's actually a very good promoter of weight loss,

especially in insulin-resistant children. If you look at the adult data it's a relatively mediocre response. The question is why does it work in some patients to cause weight loss and not in others, and that's a very complicated answer. I think it has to do with how insulin- resistant you are when you take the medicine.

It is true that there are some anti-diabetic drugs that cause you to gain weight, they are called the glitazones one is called rosiglitazone or piaglitazone.

Norman Swan: They are the new generation of anti-diabetic drugs?

Robert Lustig: That's right and the reason they cause weight gain is because they actually cause pre-adipocytes, that is fibroblasts that are not adipocytes themselves yet...

Norman Swan: So actually these are the cells that become fat cells?

Robert Lustig: Yes, almost adipocyte stem cells if you will, and they actually cause them to differentiate into adipocytes giving you a larger tank to store energy, and when you do that you actually can clear energy better and that makes you insulin-sensitive for a time. But only at the expense of increasing the size of the tank.

Norman Swan: So why are people still searching for weight-loss drugs if what you're saying is all you need to do is to depress insulin and the technology exists to do that?

Robert Lustig: Well it's not that easy. It's actually quite difficult. Not everybody has a disorder that's amenable to an insulin antagonist. Only about 20% of adults have the disorder called insulin hyper secretion that is responsive to this drug called Octreotide. 80% are insulin-resistant and Octreotide does not work in them at all. Metformin will work in them but only to a certain point because it's not the perfect drug either and it has other side effects.

Norman Swan: There's a non-drug that does it which is exercise, particularly resistance exercise, building up your muscles.

Robert Lustig: Exactly, in fact exercise is the best treatment. The question is why does exercise work in obesity? Because it burns calories? That's ridiculous. Twenty minutes of jogging is one chocolate chip cookie, I mean you can't do it. One Big Mac requires three hours of vigorous exercise to work that off, that's not the reason that exercise is important, exercise is important for three reasons exclusive of the fact that it burns calories.

The first is it increases skeletal muscle insulin sensitivity, in other words it makes your muscle more insulin sensitive, therefore your pancreas can make less, therefore your levels can drop, therefore there's less insulin in your blood to shunt sugar to fat. That's probably the main reason that exercise is important and I'm totally for it.

The second reason that exercise is important is because it's the single best treatment to get your

cortisol down. Cortisol is your stress hormone, it's the hormone that goes up when you are mega-stressed, it's the hormone that basically causes visceral fat deposition which is the bad fat and it has been tied to the metabolic syndrome. So by getting your cortisol down you're actually reducing the amount of fat deposited and it also reduces food intake. People think that somehow exercise increases food intake, it does not, it reduces food intake.

And then the third reason that exercise is important, which is somewhat not well known, but I'm trying to evaluate this at the present time, is that it actually helps detoxify the sugar fructose. Fructose actually is a hepato-toxin; now fructose is fruit sugar but we were never designed to take in so much fructose. Our consumption of fructose has gone from less than half a pound per year in 1970 to 56 pounds per year in 2003.

Norman Swan: It's the dominant sugar in these so-called sugar-free jams for example that you buy, these sort of natural fruit jams.

Robert Lustig: Right, originally it was used because since it's not regulated by insulin it was thought to be the perfect sugar for diabetics and so it got introduced as that. Then of course high fructose corn syrup came on the market after it was invented in Japan in 1966, and started finding its way into American foods in 1975. In 1980 the soft drink companies started introducing it into soft drinks and you can actually trace the prevalence of childhood obesity, and the rise, to 1980 when this change was made.

Norman Swan: What is it about this, it's got more calories than ordinary sugar weight for weight hasn't it?

Robert Lustig: No, actually it's not the calories that are different it's the fact that the only organ in your body that can take up fructose is your liver. Glucose, the standard sugar, can be taken up by every organ in the body, only 20% of glucose load ends up at your liver. So let's take 120 calories of glucose, that's two slices of white bread as an example, only 24 of those 120 calories will be metabolised by the liver, the rest of it will be metabolised by your muscles, by your brain, by your kidneys, by your heart etc. directly with no interference. Now let's take 120 calories of orange juice. Same 120 calories but now 60 of those calories are going to be fructose because fructose is half of sucrose and sucrose is what's in orange juice. So it's going to be all the fructose, that's 60 calories, plus 20% of the glucose, so that's another 12 out of 60 -- so in other words 72 out of the 120 calories will hit the liver, three times the substrate as when it was just glucose alone.

That bolus of extra substrate to your liver does some very bad things to it.

Norman Swan: Dr Robert Lustig who's Professor of Pediatric Endocrinology at the University of California, San Francisco. And you're listening to a Health Report special here on ABC Radio National on how food manufacturers by adding fructose to our foods, either from corn syrup as in the United States or added sucrose as in Australia, may actually be making the obesity epidemic even worse, starting with damage to our liver cells, the hepatocytes.

Robert Lustig: The first thing it does is it increases the phosphate depletion of the hepatocyte which ultimately causes an increase in uric acid. Uric acid is an inhibitor of nitric oxide, nitric oxide is your naturally occurring blood pressure lowerer. And so fructose is famous for causing hypertension.

Norman Swan: High blood pressure. And what you're saying here is that the liver cell itself gets depleted of this phosphate and then you've got this downstream reaction.

Robert Lustig: That's right. And so when you have excess uric acid you're going to end up with increased blood pressure and we actually have data from the NHANES study in America, the National Health and Nutritional Examination Survey in America which actually shows that the most obese hypertensive kids are making more uric acid and have an increased percentage of their calories coming from fructose.

Norman Swan: Are they getting gout as well?

Robert Lustig: Well not yet. They will.

Norman Swan: So what you're saying in fact is that whilst we are clearly eating too much, we're passively eating too much of the wrong thing, that the food manufacturing industry is putting stuff in which is fuelling the epidemic?

Robert Lustig: Absolutely, we're being poisoned to death, that's a very strong statement but I think we can back it up with very clear scientific evidence.

Norman Swan: There's clear scientific evidence on this fructose pathway in the liver?

Robert Lustig: There's clear scientific evidence on the fructose doing three things that are particularly bad in the liver. The first is this uric acid pathway that I just mentioned, the second is that fructose initiates what's known as de novo lipogenesis.

Norman Swan: Which is fat production.

Robert Lustig: Excess fat production and so VLDL, very low density lipoproteins end up being manufactured when you consume this large bolus of fructose in a way that glucose does not, and so that leads to dyslipidaemia.

Norman Swan: And that's the bad form of cholesterol.

Robert Lustig: That's correct. And then the last thing that fructose does in the liver is it initiates an enzyme called Junk one, and Junk one has been shown by investigators at Harvard Medical School basically is the inflammation pathway and when you initiate Junk one what happens is that your insulin receptor in your liver stops working. It's phosphorylated in a way that basically inactivates it, serine phosphorylation it's called and when your insulin receptor doesn't work in your liver that means your insulin levels all over your body have to rise. And when that happens basically you're

going to interfere with normal brain metabolism of the insulin signal which is part of this leptin phenomenon I mentioned before. It's also going to increase the amount of insulin at the adipocyte storing more energy. And you put all of this together and basically you've got a feed forward system of increased insulin, increased liver fat, liver deposition of fat, increased inflammation, you end up with non-alcoholic fatty liver disease. You end up with your inability to see your leptin and so you consume more fructose and you've now got a viscious cycle out of control.

In fact fructose, because of the way it's metabolised, is actually damaging your liver the same way alcohol is. In fact it's the exact same pathway, in fact fructose is alcohol without the buzz.

Norman Swan: So this is the obesity related fatty liver disease that people talk about?

Robert Lustig: Exactly.

Norman Swan: Some people say, I've heard obesity experts say, well it's surprising that will all the ready availability of food that we're not fatter. In other words that we are actually controlling our appetite pretty well given that we've probably been evolutionary designed to eat anything that goes, and there's anything that goes all around us, so why aren't we actually fatter? It's not so much why is there an obesity epidemic, why isn't is worse, is what people say and therefore you don't need to postulate fructose, it's just the fact that we've evolved in the savannah to eat in times of plenty.

Robert Lustig: I've heard those same concerns you know, why, if we have so many calories why aren't we fatter. Well there are a few reasons why that might be. I do want to mention that the American food industry produces 3,900 calories per capita per day. We can only eat 1,800 calories per capita per day. In other words the American food industry makes double the amount of food that we can actually use. Who eats the rest? We do, through this mechanism, they actually know that by putting fructose into the foods that we eat, for instance pretzels -- why do you need fructose in pretzels, why do we need fructose in hamburger buns?

Norman Swan: Are you postulating here a fructose conspiracy, the way the tobacco industry had a nicotine conspiracy?

Robert Lustig: Well I can't call it a conspiracy per se. I certainly know, and they certainly know that they sell more of it when they add the fructose to it. That's why it's in there, otherwise why would it be in there? Do they know that this is actually harmful? That's what I don't know. There's no smoking gun, ultimately we found the smoking gun for smoking, you know we found the documents. I'm not prepared to say that about the food companies. I do not know that they know that they are hurting us. However, they definitely know they sell more, and it temporally coincides with the advent of fructose being added to our diet.

Norman Swan: And of course you could argue that it's going up because they are responding to the market and they've got sugar-free, fat-free etc. etc.

Robert Lustig: Well in fact fat-free doesn't help, if anything as the fat content of our foods has gone down, and it has gone down, it's gone from 40% to 30%, in fact our obesity prevalence has gone way up. So that's not the answer.

Norman Swan: This is because they're adding carbohydrates and sugars to it to replace the fat.

Robert Lustig: Absolutely, in fact fat does not raise your insulin but certainly sugar does. And fructose has been bandied about...because after all it doesn't raise your insulin directly because there's no fructose receptor on your beta cell in your pancreas. So people say well it doesn't raise your insulin, but in fact it does because it's a chronic effect not an acute effect. This has nothing to do with one fructose meal, this has to do with a year's worth of fructose meals, or a lifetime's worth of fructose meals, because as you become insulin resistant, which fructose clearly does and has been shown by many investigators not just me -- that interferes with that leptin signal which causes you to eat more.

Norman Swan: Insulin-resistance increases your insulin levels because your pancreas pumps out more to get the insulin working.

Robert Lustig: Exactly, especially since your liver is not responding to it because of that effect on the serine phosphorylation of the insulin receptor. So that's going to cause you to make a whole lot more insulin, that's going to interfere with your leptin, that's going to make you eat more so the whole thing just keeps going out of control.

Norman Swan: One way of proving this would be to put you on a fructose free diet, has anybody done that?

Robert Lustig: Well no one's done it yet. In fact we're trying to do that, in fact we're actually going to be working with the Atkins Foundation here in America to actually do a fructose withdrawal experiment to try to actually answer that question directly.

Norman Swan: Well given that you're not going to come to harm by reducing the fructose in your diet -- somebody who's listening to this -- what's the ingredient on the packet, or the jar, or the back of the tin that tells you there's fructose in there because it won't always say fructose will it?

Robert Lustig: Well high fructose corn syrup, it should say that, now in Australia for instance the sodas don't have high fructose corn syrup they have sucrose. Well sucrose is half fructose. You know a lot has been made over this high fructose corn syrup being particularly evil. In fact high fructose corn syrup is either 42% or 55% fructose, the rest is glucose. Well sucrose is 50% fructose the rest is glucose. In fact high fructose corn syrup and sucrose are equally problematic.

Norman Swan: Basically table sugar.

Robert Lustig: Table sugar -- that's right. We were not designed to eat all of this sugar, we're supposed to be eating our carbohydrate, particularly our fructose, with high fibre. Well the fact is we

have 100 pound bags of sugar that go into the cakes, and the donuts.

Norman Swan: So we don't need to get obsessed on fruit sugars, it's sugar itself, sucrose.

Robert Lustig: Absolutely, it's sugar in general. So people say oh does that mean I can't eat fruit? No, let's take an orange -- an orange has 20 calories, 10 of which are fructose and has high fibre. A glass of orange juice has 120 calories, it takes 6 oranges to make that glass of orange juice and there's no fibre. You tell me which is better for you, so by all means eat the fruit, just don't drink the juice. Juice is part of the problem and there's plenty of data, not just mine. Miles Faith had an article in *Pediatrics*, December 2006 showing that in toddlers, in inner city Harlem in New York, in toddlers the number of juice servings correlated with the degree of BMI increase.

Norman Swan: Where does this fit, I mean people at the University of Sydney who've pioneered the glycaemic index, the idea that you get some foods which actually boost your blood sugar very quickly and some which are slow. They kind of argue that it doesn't actually matter terribly much what kind of sugar it is, it just depends on how fast your insulin is going to go up. Where does what you're saying fit into the glycaemic index story?

Robert Lustig: In fact glycaemic index is half the story, the other half of the story is the fibre. Here's the way it works -- carrots, let's talk about carrots for a minute. Carrots are very high glycaemic index, what is the definition of glycaemic index? It's how high your blood sugar goes if you eat 50 grams of carbohydrate in that food, that's what glycaemic index is. So if you eat 50 grams of carbohydrate in carrots your blood sugar goes up very high and so that would be a high glycaemic index food. Fructose is a low glycaemic index food because fructose does not stimulate insulin, it's all of these calories but it doesn't stimulate insulin. So in fact a soda has a glycaemic index of 53 which is low. So you'd say oh wait a second, carrots are bad for you and a soda is good for you? Because glycaemic index is not the whole story, in fact what you really want to talk about is a related concept called glycaemic load.

Glycaemic load is glycaemic index times the amount of food you'd actually have to eat to get the 50 grams of carbohydrate, so in carrots you'd have to eat the entire truck in order to get that. Well you can't do that, you wouldn't do that, so in fact carrots, even though they are high glycaemic index are actually low glycaemic load. Carrots are fine, there's nothing wrong with carrots. On the other hand fructose, I mean a soda, there's a lot wrong with it but you wouldn't see it in just looking at glycaemic index.

Norman Swan: So glycaemic index plus common sense?

Robert Lustig: Well it's glycaemic index plus fibre. Fibre turns any food into a low glycaemic load food. In fact we are supposed to eat our carbohydrate with fibre, that's the key. Processed wheat is white, when you go out into the field it's brown but by the time it gets to your bakery it's white. What happened? Well the bran was stripped off, well the bran is the good part, the bran is what we're supposed to be eating.

Norman Swan: I've often wondered, I've heard of some processed stuff and the evil of the food industry etc. but explain to me a conundrum -- why Asians are thin, or have been traditionally thin and for centuries they've eaten processed rice, they've eaten white rice, they don't like brown rice and I don't blame them.

Robert Lustig: Not a problem, I can explain it very simply. If you look at the Atkins diet, the Atkins diet was no-carb, high-fat, no-carb and it worked. We look at the Japanese diet, high-carb, no-fat, it also worked. When you put them together you get something called McDonalds and clearly that doesn't work. So the question is what is it about the Japanese diet, even though they eat all of this white rice, that still allows this phenomenon to be OK? And the answer is very simple -- it's called fructose, because fructose is really not a carbohydrate. If you look at the metabolism, the liver metabolism of fructose it is just like a fat, it doesn't stimulate insulin, just like fat. It causes all this de novo lipogenesis.

Norman Swan: Fat production

Robert Lustig: Fat production within the liver, it causes deposition of fat within the liver, it's actually like alcohol and alcohol is like a fat. So here's a carbohydrate that's acting like a fat. So outside of the Japanese diet, when you eat a low fat diet what are you eating? Snackwell -- and what did they do? They added sugar because otherwise it would be unpalatable. So in fact a low fat diet's not really a low fat diet, a low fat diet containing fructose is really a high fat diet and that explains what's going on. So a Japanese diet yes, they're eating a lot of white rice but they are also eating a lot of fibre in all of their vegetables and they are not consuming any fructose. There is no fructose in the Japanese diet whatsoever, but there is now, and childhood obesity has doubled in Japan in the last ten years whereas adult obesity hasn't moved.

Norman Swan: And the reasons?

Robert Lustig: Because the adults are eating like they used to and the children are eating like we do in America.

Norman Swan: So do you check your home garage floor for brake fluid every morning, I mean you can't be the most popular person with the food industry?

Robert Lustig: Well I'm not, I am not, very much so. The Corn Refiners Association and the Juice Products Association have been on my tail, but the fact of the matter is the science is clear, the science is there and the science has to drive the policy.

Norman Swan: So what about the regulators?

Robert Lustig: Well we're trying to work with them, we are trying to do something about it. They are not moving very fast. In fact you may be aware of the International Obesity Task Force that met at the Sydney meeting in October and they came out with something which they called the Sydney

principles. The Sydney principles involved marketing and advertising to children and trying to get rid of that, and they basically said that you have to do something about this and it has to be statutory in nature, it has to be regulated, it has to be a law. In fact in Europe 52 health ministers from the World Health Organisation from all the different European countries got together in Istanbul in August and agreed that marketing to children had to stop. Well in fact that is not happening in America.

Norman Swan: Nor is it in Australia.

Robert Lustig: Well probably not, but I just met with the commissioner of the Federal Communications Commission, Miss Deborah Taylor Tait, and she mentioned that she expected that the food companies would police themselves, that regulation would not be necessary. In fact I said, excuse me but I disagree. In fact in 1978 the US Federal Trade Commission had an entire congressional hearings on marketing and advertising to children and the food companies actually lobbied congress to actually have that killed. And they knew why, they knew what they were doing then, and they are going to do it again because it's not in their best interest. They couldn't increase their profits by 5% a year if they didn't advertise and market to children.

Norman Swan: Dr Robert Lustig is Professor of Pediatric Endocrinology at the University of California, San Francisco.

References:

Robert H Lustig Childhood obesity: behavioral aberration or biochemical drive? Reinerpreting the First Law of Thermodynamics. *Nature Clinical Practice, Endocrinology & Metabolism* Review,August 2006;2;8:447-457

Robert H Lustig, MD The 'Skinny' on Childhood Obesity: How Our Western Environment Starves Kids' Brains. *Pediatric Annals*, November 2006;35;12:899-907

Elvira Isganaitis,Robert H. Lustig Fast Food, Central Nervous System Insulin Resistance, and Obesity. *Arterioscler Thromb Vasc Biol.* 2005;25:2451-2462

Guests

Dr Robert Lustig
Professor of Pediatric Endocrinology
University of California
San Francisco

Presenter

Norman Swan

Producer

Brigitte Seega

Radio National often provides links to external websites to complement program information. While producers have taken care with all selections, we can neither endorse nor take final responsibility for the content of those sites.

Mon 8.30am
repeated **Mon 8pm**
Presented by **Norman Swan**

Health Report | Radio National | Programs A-Z