Dr. Ted Naiman - Hyperinsulinemia –

What Insulin Does in Your Body (LCC 2016)

I am Ted Naiman, I am a family practice doctor from Seattle

and I've been treating patients with low-carb diets for almost 20 years now.

which is crazy, yes, that does mean I became a doctor at the age 5,

which I think it's some sort of record.

So my topic today is hyperinsulinemia.

By end of this talk I want everybody here to know exactly what insulin does in your body,

I want everybody have a really good feel for what insulin is there for.

I want you to know what factors influence insulin, what raises and lowers insulin.

I want everybody to know what hyperinsulinemia means to our health,

how we get hyperinsulinemia and how you can reverse or prevent it.

So it's all going to be about insulin today.

I have a ton of stuff to go through and this is going to go way too fast,

and I apologize in advance - it's going to go way too fast.

Here we go, let's dive right in.

100 years ago Sir Edward Schafer - this guy theorized insulin.

He realized that if you take the pancreas out of a dog, it dies of type 1 diabetes,

if you put the pancreas back and it lives.

And he knew that the pancreas was crucial

for controlling fat and glucose and carbohydrates.

And he theorized this and he called this substance that the pancreas made - insulin.

A couple of years later it was identified.

These early pioneers of insulin realized that insulin had two simultaneous actions.

First of all it has this excitatory or stimulatory action,

meaning insulin did something.

And what it did was uptake of glucose into the cells

and stimulate the formation of fat.

At the same time insulin stopped something, it had an inhibitory role.

And what insulin stops is the breakdown of stored energy,

inhibits lipolysis, which is a breakdown of stored fat

and inhibits glycogenolysis, which is a breakdown of stored glycogen.

Now doctors came along and said, "Look, insulin lowers blood sugar a lot!

Well, that's convenient, we've got a bunch of patients out here with high blood sugar."

So doctors started to prescribe insulin for anyone with high blood sugar.

Now, when you have a hammer, everything looks like a nail.

And doctors had this insulin hammer.

The average healthy pancreas makes 24 units of insulin a day

and doctors started to prescribe 50 units of insulin a day,

100 units of insulin a day, 300 units of insulin a day,

500 units of insulin a day...

And we ushered them what will for ever be known as "The Black Age of endocrinology".

From about 1960 to 2000.

I'm quoting from a textbook from the Black Age of endocrinology,

"The basic action of insulin is to facilitate glucose entry into the cells,

primarily skeleton, muscles and hepatocytes"

This this kind of wrong, we had this flawed concept

that you needed insulin to shove glucose into your cells

and that type 2 diabetics had all this glucose stacking up in their bloodstream,

just because they needed more insulin to shove the sugar into their cells.

And this was wrong and terrible and awful.

Here is a cartoon from the Black Age of endocrinology.

"I'm learning to manage my type 2 diabetes with insulin."

And I'm just eating a lot of crap and sucking down a gallon bottle of insulin.

And this is basically how we function for a half a century.

Now we're living in a much more enlightened age of insulin,

at least most of us do, hopefully.

This is a beautiful graph showing the smoothing simultaneous action of insulin

on an adipocyte.

He can see that insulin simultaneously stimulates lipogenesis,

or the formation of fat,

and it inhibits lipolysis or the breakdown of fat - this is what insulin does.

What we now know is that it is the inhibitory action of insulin

that is far more relevant to human physiology,

and it's the inhibition of lipolysis, which is the more important action.

This is the primary role of insulin -

inhibition of lipolysis and the preservation of stored energy.

Here is what insulin does -

Insulin is a nutrient sensor,

it detects energy coming in from outside your body

and it communicates to other cells, "Hey, don't break down stored energy,

because we have energy coming in from the outside."

And that wouldn't make sense, that would be wasteful.

Your body is very thrifty and went through a lot of trouble to store that energy.

You don't want to burn it, if energy is coming in.

Here is a picture of how it works -

there's my doughnut, there's my nutrient sensor cell.

It says, "I think we ate a doughnut, there's energy coming in.

"Stop lipolysis, we're not going to break down our stored energy,

because there is energy coming in."

So I am quoting from modern textbooks on action of the insulin.

"The overall action of insulin on the adipocyte

is to stimulate fat storage and inhibit mobilization" - that's what insulin does.

"Insulin not only promotes fat storage, but it also restrains fat mobilization."

Storing, not burning, that's what insulin does. blah, blah, blah, whatever.

Primary function of insulin - insulin is a nutrient sensor.

It's there to preserve stored energy.

And at the end of this lecture, there's going to be a final exam

and it's going to be one question long.

I'm going to ask everybody, "What is the primary function of insulin?"

And you will say, "It detects nutrients coming in, it preserves stored energy.

The primary function is the inhibition of lipolysis - this is what insulin does."

Let's talk about anabolic versus catabolic just for a second.

Anabolic reaction, you take smaller molecules,

you add energy, you make a bigger molecule.

Catabolic obviously, you take a bigger molecule,

you break it down, you get smaller molecules and energy back.

Examples of anabolic reaction, you chain together glucose, you add energy,

you get glycogen, the storage form of glucose.

You chain together fatty acids with the glycerol backbone,

you add energy, you get triglycerides, the storage form of fat.

You chain together amino acids, add energy, you get protein.

Now, insulin is anabolic and at the same time is anti-catabolic.

That's what the insulin does.

When you perform anabolism and you take smaller molecules

and you chain them together and add energy, you physically make things larger

and that's where we get the term anabolic steroids for bodybuilders.

Of course, insulin is anabolic and anti-catabolic

and it physically makes things larger.

When you perform catabolism, which is the opposite,

you take a larger molecule, you break it down into smaller molecules

and get energy back,

you are physically making things smaller and that's what catabolism is doing.

Now if you were a type I diabetic, you don't have any insulin...

You know maybe what an auto-immune disease is -

destroys the beta cells in your pancreas and you don't have any insulin.

And when you don't have insulin, you go through uncontrolled catabolism.

You are just burning off all your mass into energy.

Here is a picture of a type 1 diabetic patient.

On the left is before insulin, where is insulin deficient.

On the right is with insulin.

And you can see that this poor patient

is literally melting their entire body down into energy.

And they have uncontrolled catabolism or accelerate starvation.

This is the primary role of insulin, it prevents catabolism,

or the breakdown of stored energy.

This is what insulin does.

Now this is a little bit weird, but bear with me, I like this analogy.

I have on the screen here a definition of a battery.

A battery is a container consisting of one or more cells

in which chemical energy is converted into electricity

and used as a source of power.

A battery has two basic states, it can be charging or discharging.

Now your human body is basically battery-powered.

That's how we have energy to function

without being connected to an external source of power.

You literally have chemical energy in your body

that you convert to an electrical gradient into mitochondria

and you use that to make UTP, and that's how we function.

You've got basically two battery systems in your body.

You have your primary battery, which is fat,

you have a secondary like a turbo boost, like a nitro mode, which is glycogen.

You only have about 2000 cal of glycogen in your body.

That's enough to power you for maybe one day or 90 minutes of high-intensity exercises.

But the average person has at least 40 times that much stored in fat,

maybe 100 times or more stored in fat.

So we use that for most of our activities -

if you're walking, talking, even lightly jogging, you're mostly powered by fat.

But if you're suddenly sprinting for your life from a saber-tooth tiger,

you tap into glycogen, you burn maximum fat and add glycogen to that.

And it's like jet fuel or nitro boost.

So we burn glycogen in emergencies for tons of glucose.

And that's how you get maximum output.

Now why is fat preferred by your body?

I got the carbon chains here, the glucose and left, fatty acid on the right

and it turns out that fat is 25% more efficient than glucose on a carbon-for-carbon basis.

Also of course fat has more energy density, 9 cal/ gram, versus 4 cal/gram.

Fat also burns more cleanly with less exhaust.

So there's a lot of reasons why your body prefers fat,

but we also like our glucose on top of that for emergencies.

It's really good if you're running for your life.

So here's my battery-powered human.

I've got my primary fat battery,

I've got my turbo boost nitro mode glucose for glycogen battery

and now I eat a T-bone steak and my insulin level goes up,

because there is energy coming in from the outside

and insulin is like the little charge symbol on your cell phone

that you've plugged into the wall now and you're charging.

I'm quoting from a biochemistry textbook,

"Signaling an abundance of exogenous energy..."

Exogenous is coming in from the outside, like an exoskeleton.

"...adipose tissue fat breakdown is suppressed and its synthesis is promoted."

Now you are in storage mode, you're just plugged in to the wall,

you are storing energy.

So there's a Fed State and a Fasted State.

And in the Fed State, there's my turkey.

In the Fed State, insulin is high

and you are partitioning all your calories into storage.

"Okay, now I'm recharging my batteries."

In the Fasted State, insulin is low and now I'm freely discharging my batteries,

I'm leaving off of stored energy, I am unplugged from the wall.

Now, I know what you are thinking.

You're thinking, "Isn't insulin there to lower your blood sugar?"

Well, no, insulin is not there to lower your blood sugar.

This is not the primary job of insulin.

And let me explain, this is really important, see this little cute airplane here?

This airplane is your blood sugar.

What happens if the altitude at a certain point drops to zero?

Well, the plane crashes and everybody dies.

The same thing happens to your body if your blood sugar goes to zero.

So that can't happen, you're going to die instantly.

That won't happen, that doesn't happen,

your body is such an amazing survival machine...

It won't happen, you have all the systems in place to keep your blood sugar up.

You've got glucagon, which is always there, always raising your blood sugar.

Your blood sugar is never going to go too low.

If something were to happen to glucagon,

you've got five other emergency backup hormones -

epinephrine, cortisol, ACTH, growth hormone and thyroxine.

Your blood sugar is not going too low. Right?

Yes, insulin sort of halfheartedly lowers blood sugar as part of its storage capacity

it's just that up taking glucose in your cells,

so it can store it as glycogen or fat.

It doesn't really want your blood sugar to be that low.

Let's face it, if your blood sugar is too high,

who cares you're going to live another 20 or 30 years and die of kidney failure?

But you have plenty of time to pass on your genetics

and have offspring and fulfill your role to evolution,

so it's not that a big of deal.

So insulin is not really there to lower your blood sugar, it's not its primary job at all.

There's this counter pressure with glucagon raising the blood sugar and insulin lowering it.

But insulin is going to lose every time,

because the default has to be high blood sugar.

You can let your blood sugar be too low.

Let's look at some actual insulin curves.

So I've got here is actual curves, real data, insulin and glucose.

You've got glucose in red, insulin in blue and you can see

that they basically mirror each other and you've got spikes at mealtime.

And then notes that it takes about 12 hours after your last meal

for your insulin to slowly, gradually drift all the way back down the baseline.

That's going to be important later when we'll talk about intermittent fasting.

Here is another insulin curve -

you see spikes at mealtime, breakfast, lunch, dinner.

And again it takes about 12 hours for insulin to get back down to baseline.

Let's look at the glucose index on the left and insulin index on the right.

This is how high these things go up with the different micronutrients,

carbs, protein, fat.

You get a big spike of glucose and insulin from carbohydrate,

so this is the big story here.

Now on the glucose side, protein and fat... nothing - flat as a pancake.

On the insulin side, protein gives you about half a spike

and fat is much, much smaller.

Here is another study, you've got red arrows pointing to an oral glucose tolerance.

It says you drink 100 g of glucose,

check your blood sugar on the top insulin level on the bottom

and you get huge spikes from carbs.

The green arrows point to an oral fat tolerance,

which is where you drink 40 g of fat

and you see nothing happening on the glucose side

and just the tiniest little blip on the insulin side on the bottom.

Now this beautiful graph was made by Marty Kendall,

who is a really smart engineer guy in Australia.

He has this awesome blog optimizingnutrition.com

I don't know if anybody here has heard of it, but it's really cool, you should check it out.

He has done a ton of number crunching with the insulin index of foods.

And he has graphed out a bunch of foods here.

You can see that the big story with insulin index of foods is carbohydrate.

The main thing raising insulin index is carbs.

At the same time the primary factor of giving foods a lower insulin index

is the fat content.

Here is a study where they took a bunch of different foods.

They graphed them all out and they looked to see

what factors raise and lower the insulin index of foods.

What tends to give you a lower insulin index of foods is fat, protein and fiber.

So your steak and your salad is going to have a lower insulin index.

Your carbs and sugars are going to raise insulin index,

so your pop tarts are going to have a really high insulin index.

Okay, this is where it gets really interesting.

Everything before this was boring and stupid, but this is great.

To this you'll have to pay attention to.

We're going to look at glucose and insulin curves

in normal people and obese people.

Check it out.

There's a glucose curve, normal in green, obese in red,

you see spikes as mealtimes...

And actually that's not interesting now, they are exactly the same.

Who cares? This is pointless.

This is interesting - insulin.

Look at normal versus obese,

you've got normal in green, you've got obese in red.

And wow! Holy crap! Can I say holy crap?

Okay, sorry, holy crap... Can I say it twice?

The obese person is twice as high at baseline fasting

and it's double, triple, quadruple after eating.

It's a huge big deal, major difference.

It turns out there's nothing you can measure metabolically

that is this difference between lean and obese - that's the insulin levels.

Let's take a closer look at the rate of insulin secretion.

You've got normal in green, obese in red.

And wow!

Again obese persons, twice as high at baseline fasting

doubled, tripled, quadrupled AUC after eating.

So there's a huge big deal, this is major, this is enormous!

Now once you know, like everybody in this room now knows,

the primary function of insulin is inhibition of lipolysis.

You've got to ask yourself whether these obese people were ever burning fat.

Well that would be never, that's why they are fat.

And when do these people partition all their energy into storage?

And that will be all the time.

And then if you can't tap into stored body fat,

how hungry are you going to be a couple of hours after your last meal?

You are going to be really hungry all the time.

And if you can't tap into stored body fat,

what are you going to be burning mostly on the cellular level,

I mean your mitochondria?

You mostly are going to be burning glucose.

And what you've got right here

is the entire behaviors of obesity explained in one graph.

Always burning glucose,

always hungry for carbs, craving carbs for glucose.

Hungry all the time, eating frequently, low energy,

because you're constantly partitioning all your energy into storage,

always storing fat and never burning fat.

This explains the entire phenomenon of obesity in one slide.

It's huge big deal, very important, this is crucial.

I mean everybody has to know what's going on with insulin.

Here's another study - you've got glucose anabolism on corner.

Lean versus obese exactly the same.

Who cares? Blood sugar is worthless.

Look at insulin, see peptide, the rate of insulin secretion.

Massive differences between lean and obese.

This is a really, really big deal.

This is a study just in overweight people, not obese.

But look at insulin in normal versus overweight.

There's a big, huge difference there.

And if you look at respiratory quotient,

these overweight people have no metabolic flexibility,

they are burning mostly glucose all the time.

Okay it turns out you can draw a graph

of 24-hour insulin secretion AUC and body mass index.

And it is a perfectly straight line.

And this is so important,

because your fat mass is 100% determined by insulin.

Insulin is the most important thing

you could possibly be looking at when it comes to obesity.

I love this graph, this is so important.

This is another beautiful graph that I absolutely love.

Let's look at fasting insulin levels, these are fasting insulin levels.

On the far left, the tiny little bar you've got a normal lean person.

Next to that, in yellow, you've got an obese person with normal blood sugar.

And yeah, their insulin levels are four times higher than the normal lean person.

Now you've got an obese person with impaired fasting glucose,

their blood sugar's too high.

Now you've got a really type 2 diabetic.

And there's an advanced type 2 diabetic.

And, holly cow, the higher you're fasting insulin level,

the further along the phenotype you are to obesity and diabetes

and you can really explain the twin epidemics of obesity and diabetes.

100% with insulin - this is the whole story right here.

Insulin is crucial, we have to be looking at insulin.

This is everything, this is basically everything in obesity and diabetes - insulin.

Another fascinating thing happens -

the longer you've been obese, the higher and higher your insulin levels go.

It's almost exponential.

In this graph you've got ration of obesity in years on the X axis,

insulin just goes up and up and up.

It's because the more insulin resistant you are, the more insulin you secrete

and that makes you even more insulin resistant.

This is a downward spiral straight from hell.

Can I say "hell"?

Oh, okay, sorry... yeah, straight from hell.

Here is another study,

the longer you've been obese, the lower your insulin sensitivity.

This is just a horrible downward spiral.

This brings us to the mechanism of insulin resistance.

How do you get insulin resistant?

Turns out the more insulin you're exposed to, the more insulin resistance you get.

And this is not unique specialty of insulin,

this is every hormone in your body.

If you are exposed to any hormone, you can get resistance to it.

Actually if you are exposed to anything your body you will get resistance to it.

That's how your body works.

Let's talk about olfactory fatigue.

This is a phenomenon where if you are in a room with a really bad smell,

at first it's overpowering, you stay in there a couple of hours, it's not so bad,

you stay in there all day, you can't smell it at all.

Now if you leave the room for a long time and then you come back in,

well, then you can smell it.

This is adaptation and everything in your body works this way.

You go from a dark room into bright sunlight, you can't see until you adapt.

You go from bright sunlight into a dark room, you can't see until you adapt.

All your sensors work this way, all your hormones work this way,

all your neural processes work this way,

all your biochemical processes work this way.

Your whole body just adapts to stuff.

We have studies that prove this -

insulinoma patients, they have a tumor that makes insulin.

They get insulin resistant.

Here is another study, insulinoma patients,

anyone with high insulin gets insulin resistance.

If you give insulin in a pulsatile fashion,

you stay more sensitive to it than if you give it continuously.

You can take rats or humans and just give them higher and higher doses of insulin

and literally make them insulin resistant.

And so, we doctors have been doing that

throughout the Black Age of endocrinology.

So this really happens, this is not something made up by Dr. Fung.

This is exactly how your body works, this is real, this is happening.

Let's talk about hyperinsulinemia and what it means to your health.

Hyperinsulinemia is really just a metabolic syndrome.

And everybody here is familiar with metabolic syndrome, hopefully.

Abdominal obesity, high triglycerides, low HDL, high blood pressure

and eventually high glucose.

But I want everybody to know that high glucose

is the very last thing you see when you have hyperinsulinemia.

In this graph only 10% of people with hyperinsulinemia have high glucose.

The other 90% have a ton of metabolic damage.

So glucose is the worst thing to track.

Is literally the last thing...

We saw these other graphs, lean versus obese...

Blood sugar was the same. Who cares?

What percentage of US adults have hyperinsulinemia?

Well, this is a graph of the US adults with hyperinsulinemia

and it's like 70% or 80%.

This is a huge epidemic, this is a really, really big problem.

That brings us to the work of Dr. Joseph Kraft.

Who the heck is Dr. Joseph Kraft?

Well, he is this really cool, really old pathology dude

who started 40 years ago

collecting oral glucose tolerance tests with insulin levels.

And he did it in 14,000 patients.

When you do a glucose tolerance test, you drink 100 g of glucose,

your check your blood sugar afterwards.

If your blood sugar is normal, then you are normal,

if your blood sugar is too high, then you are pre-diabetic or diabetic.

And when you do that on a standard population the way Kraft did,

you see that 2/3 of people have normal blood sugar

and 1/3 of people have high blood sugar, they are pre-diabetic or diabetic.

Well, Dr. Kraft took a step further and he looked at insulin levels with these tests

and he saw that a few people had normal insulin,

but way more people had this crazy hyperinsulinemic response.

And when he broke it down, he saw that basically 80% of people had hyperinsulinemia

and only 20% of people were normal.

And he called this diabetes in situ, which is pathology term for early diabetes.

And this correlates really well with the 80% of people

we know in the US have one or more symptoms of hyperinsulinemia.

So this is just a huge epidemic, this is basically everybody.

How do you know if you have hyperinsulinemia

without drinking 100 g of glucose

and checking your blood sugar hour after hour for five hours?

You could just look at your fasting insulin level.

Fasting insulin is actually pretty good.

The average in this country is 8.8, that's way too high.

It should be less than five, you really want a 2 or a 3.

But fasting insulin level correlates fairly well with insulin sensitivity.

It's a pretty good test, I kind of like it.

Now for free, you can measure your waist-to-height ratio.

Waist-to-height ratio is almost amazingly perfect one-to-one for insulin sensitivity.

This is an awesome measurement and it cost nothing.

You measure your waist at the belly button,

abdomen fully relaxed,

you divide it by your height - your weight should be half your height.

So your waist-to-height ratio should be 0.5 or less.

If it's higher than that, not only you're hyperinsulinemic,

not only have insulin resistance, but you're going to die a lot faster.

You can draw a graph of years of life lost versus waist-to-height ratio

and it's just this crazy exponential monster.

It turns out there's nothing you can measure on human

that's more correlated with strata of dying than waist-to-height ratio.

And that's because it's one-to-one with insulin sensitivity.

Awesome measurement!

What else correlates well with insulin?

Well, triglycerides go up nicely with insulin and HDL goes down nicely at the same time.

So I really like triglycerides to HDL ratio, this is an awesome measurement.

I like triglycerides to HDL ratio, I like waist-to-height ratio

and I like fasting insulin levels.

And those are the best three ways to tell if you are hyperinsulinemic or not.

Now if you have high insulin, there are two pathways you can be on.

You can have high blood sugar, and you are pre-diabetic or diabetic,

and then you are resistant for all these diseases of high glucose

that were familiar with the diabetics, diabetic retinopathy, nephropathy, neuropathy.

Only 10% of people have high blood sugar

and the other 90% have completely normal glucose.

But there's still a risk for this crap ton of diseases you get from high insulin...

Hypertension, stroke, polycystic ovarian syndrome,

fatty liver, cancer, sleep apnea, obesity of course.

So all this stuff is going to happen to 90% of people,

before their blood sugar is ever abnormal.

Cardiovascular disease can be driven equally well

from high insulin, high glucose or both.

So that's a huge, big deal.

You can have tons of disease and damage from high insulin

with totally normal blood sugar.

Let's look at blood pressure - it turns out you can draw a graph,

blood pressure versus insulin it's fairly linear.

How does this occur? - Well, insulin causes your kidneys to retain salt and water.

And that jacks up your blood pressure.

This is also why people with hyperinsulinemia have problems with swelling,

their feet and ankles swell, their rings fit tightly.

This is why if you are hyperinsulinemic and you go on a low-carb diet

and slash your insulin level,

you'll lose 10 pounds the first week because you pee out a gallon of salted water.

It also drops your blood pressure a lot by the way.

Cardiovascular disease, if you have hyperinsulinemia,

your risk is at least 2.5 times higher.

Here is the Paris Perspective Study.

They will take quintiles of fasting insulin

and the risk of dying of heart disease annually

just goes up and up with each quintile of insulin.

Dr. Kraft has quoted the same,

"Those with cardiovascular disease not identified with diabetes

are simply undiagnosed."

Because in his experience, everybody with vascular disease had high insulin.

And I agree with this 100%, that's exactly what I have seen my entire career.

Here is the Helsinki Policemen Study, really good study, 22 years of data.

Quintiles of insulin and dying of heart disease goes up in a straight line.

Hypersinulinemia was a major independent cause risk factor.

Here's another good study on insulin resistance and coronary disease in non-diabetics.

They look at tertiles and quintiles of insulin.

Sure enough the higher your insulin level, the more vascular disease you have.

This is a great study, they look at the hyperinsulinemia and heart disease.

Of course, high insulin was the major risk factor.

Look at these low insulin people, they have way less heart disease than everybody else.

And a fascinating thing came out of this study.

If you look at the Apolipoprotein B or a particle count,

you see the huge difference

and particle counts are really, really high insulin levels,

but if you look at someone with even half way normal insulin,

it's less than 12 on the left side there,

particle count completely collapses as a risk factor, it just goes away.

This is why some doctors think particle count is the Holy Grail of heart disease

and other smarter doctors, like Dr. Gerber,

realize this is really not that important.

So there you have it.

This is a great study that did a huge mathematical analysis

of all the variables in heart disease and realized that yes,

"insulin resistance is likely the most important single cause of CAD."

Thank you.

This is a great study, this is basically the study of the Black Age of endocrinology.

They gave higher and higher doses of insulin to type 2 diabetics

to drive their sugar really low.

And guess what - they just died more, increased all cause mortality,

major adverse cardiac events and cancer.

So if you trade lower sugar for injecting more insulin,

you're literally going to die faster,

it's really worse for you to have high insulin and lower sugar

than it is to have a higher sugar and lower insulin.

This is really a big deal.

This is a great study, they followed 200 people for 10 years.

They looked for age related disease endpoints -

hypertension, cancer, heart disease, diabetes, stroke.

Divided them into tertiles of insulin.

The higher your insulin, the more of all the stuff you had.

Turned out there were no events in the most insulin sensitive tertiles,

that's probably the one I would like to be in.

Let's talk for one second about insulin-like growth factor, IGF-1.

Insulin-like growth factor...

Insulin and IGF-1 go hand in hand.

Everybody with high insulin has high IGF-1.

Like the name implies, it's a growth factor for all your tissues,

it grows things like fertilizers.

This is a graph of high data over the last 150 years for every country in Europe.

You can do this anywhere in the world, we're all growing about 1 cm a decade.

We've grown 6 inches in the past 150 years average height.

This is because we have more calories, more carbohydrates,

more energy density, more food availability.

We have high insulin levels and we have higher IGF-1 levels.

This is great, right?

We're all going to be in the NBA.

No, actually it's not so good for your health.

It turns out your cancer risk goes up when IGF-1 goes up.

Here is a graph of cancer risk versus height in the Million Women Study.

And the taller you are, the more cancer you get.

Here is another study - for every increase in height of 4 inches

the risk for every cancer we've got gets skewed to the right.

Here is a French study, lifespan versus height.

The taller you are, the shorter your lifespan... Sorry, Dr. Eenfeldt.

Here is a study in baseball players, the taller you are the shorter your lifespan.

This is not that great.

Look at cancer.

The pink line is the fifth quintile of insulin,

the black one is one through four.

And people with the highest insulin are dying of cancer

a decade before everybody else.

It's kind of bad, this is kind of a big deal.

Alzheimer's disease is an epidemic, it's on the rise,

it's going to bankrupt every industrialized nation on earth in the next 50 years.

And of course it has to do with hyperinsulinemia.

There's a prospective study, they followed people along.

The higher insulin level is, the more cognitive decline you have.

Period.

Insulin was a major risk factor for Alzheimer's.

Here is another study.

Your risk for Alzheimer's is more than doubled if you have hyperinsulinemia.

Here is another study, it looks at your retinal vessel abnormalities

and cerebral atrophy, brain shrinkage and cognitive decline.

The higher your insulin level, the more of all of these bad stuff you had.

Polycystic ovarian syndrome, most of us know by now

that PCOS is basically just pure insulin resistance in women.

And look at this graph of insulin secretion and oral glucose tolerance test

in PCOS versus controls.

Just tons of insulin flying around.

There's another study -

hyperinsulinemic women had the largest ovaries and the most polycystic ovaries.

Now men get their own version of this.

Men with hyperinsulinemia have up regulation of aromatase activity,

which means you convert all your testosterone to estrogen.

So men with hyperinsulinemia have low testosterone, high estrogen, gynecomastia

and this grows the hell out of your prostate,

you get tons of prostate growth and prostate cancer.

I've got study after study that just comes right out and says

that the primary cause of enlarged prostate is insulin.

This is basically the primary event in prostate enlargement and prostate cancer.

On erectile dysfunction does insulin resistance play a part?

Yes, of course it does.

Skin findings in hyperinsulinemia - acne, hair loss, hirsutism,

skin tags, huge odds ratio between skin tags and hyperinsulinemia.

7.5, that's like "smoking causes lung cancer" territory right there.

Other skin findings related to hyperinsulinemia - seborrheic keratosis,

the waxy, warty brown things on old people.

Acanthosis nigricans, start discoloration, all these things.

Psoriasis, of course, people with psoriasis are more insulin resistant than controls.

Now everybody knows you can get a chronic kidney disease from high blood sugar.

Diabetes is number one cause of kidney failure,

but you can also get a nasty chronic kidney disease

from just hyperinsulinemia with normal sugar.

This is the mechanism that I'm not going into, but suffice it to say

that high insulin will give you chronic kidney disease.

It will also increase your risk for kidney cancer.

Autoimmune disease, here is a graph...

Rheumatoid arthritis versus insulin resistance, straight line.

Lupus versus insulin resistance, straight line.

Everybody knows that high sugar will give you a peripheral neuropathy.

Diabetics get numbness in their toes and feet.

But you can actually get the same peripheral neuropathy

just from high insulin with normal glucose.

We see that in insulinoma patients again.

You can literally create peripheral neuropathy in lab animals,

by just giving them a bunch of insulin.

Intraocular hypertension - this is glaucoma.

Look at glaucoma with hyperinsulinemia versus those without.

Tinnitus or tinnitus, that's ringing in your ears...

Hyperinsulinemia has a huge association with tinnitus,

because it's a peripheral neuropathy of your inner ear.

If you go on a low-carb diet

you are five times more likely to improve your tinnitus than people who don't.

That's kind of interesting.

Vertigo, huge odds ratio between vertigo and hyperinsulinemia, 4.66.

If your sugar is high as well, it's another 2.5 times.

Idiopathic recurrent pregnancy loss is miscarriages associated with high insulin.

Sarcopenia, the higher your insulin level, the less muscle mass you have.

That sucks.

Bone strength - people with the highest insulin levels have the weakest bounds.

That's really interesting.

Tendinopathy, huge association with tendinopathy and hyperinsulinemia.

Achilles tendinopathy in this study.

Carpal tunnel syndrome -

look at the carpal tunnel people with hyperinsulinemia versus controls.

Asthma - we've known for ever

that there's a huge epidemiological association between hyperinsulinemia and asthma

and now we have a biological basis for that, we know how it works.

Sudden sensorineural hearing loss is associated with insulin resistance.

Autism and neurodevelopmental disorders -

autism is this horrible epidemic right now,

is like 1 in 61 children or something.

And women with hyperinsulinemia are four times more likely to have kids with autism.

There's nothing else this high in association.

It's a disaster, I hope somebody's researching this problem.

Longevity - it turns out the lower insulin level, the longer you're going to live.

That's kind of nice.

Let's switch it up for a second here

and talk about how to create insulin resistance with your diet.

Okay, you want to eat refined processed empty calorie carbs and fats together,

like a doughnut - it's the best way to do it.

You want empty calories so you can eat all day with no satiety

and you want carbs and fats together,

because the carbs spike your insulin and it's faster and easier

to store fat that's already in your blood stream,

rather than making it from scratch via de novo lipogenesis, that's really slow.

So when we design obesogenic rat chow for lab animals

and we want to make them hyperinsulinemic

and make them obese and make them diabetic, because they are all the same thing,

we give them a whole bunch of refined processed carbohydrates, sugar or cornstarch,

we mix them with the refined processed empty calorie fat,

like vegetable oil or lard,

and you've got obesogenic rat chow.

You can do the same thing to humans with a doughnut.

Sugar consumption is huge in hyperinsulinemia.

Here is a graph over sugar consumption for the past 200 years.

It's ridiculous.

Sad but true,

"but eight years old children today have eaten more sugar

than an adult on century ago ate in their entire lifetime."

That's really terrible.

Now let's talk about how you minimize your exposure to insulin,

which minimizes insulin resistance.

We've already looked at the insulin response to different macronutrients.

Huge spikes from carbs, half that from protein, smaller from fat.

Well, you could start by eliminating digestible carbohydrates,

like grains, sugars and starches.

Everybody in this room knows you have no need for these things in human diet.

It's stupid, right.

These glucose producing foods are pointless.

So if you eliminate those you immediately lower your insulin exposure a lot.

Here is a graph from just a standard low-carb diet.

You've got control groups in black, you've got low-carb in red.

Low-carb immediately slashes your insulin exposure in half.

Here's another study, after one day of low-carb diet

your insulin AUC is less than half of what it was.

Here is a real-time common data in a type 1 diabetic.

Standard diet on the left, low-carb on the right.

And immediately your insulin requirements drop by more than half.

Here is a high carb meal versus a low-carb meal in lean versus obese.

The obese person...

Got cut in half, sorry!

The obese person has tons of insulin after the high carb meal,

but low-carb meal really levels the playing field between lean versus obese.

This is why low-carb diets are so great for obese people.

I mean really, really levels the playing field in terms of insulin between lean and obese.

You could go on a paleo diet, we have studies that prove

that paleo diets significantly reduce AUC for insulin.

I would caution people to be careful with paleo diets though,

because plant foods have way more sugar in them than they used to.

So it's paleo to eat sweet potatoes and bananas all day,

but that's probably way too much plant sugar,

because your Paleolithic foods didn't have that much sugar in them.

So watch out.

Now if you're looking at stored body fat, it's 95% fatty acids,

it's 5% glycerol backbone, which your body makes sugar out of.

There is a way of eating that mimics living off of stored body fat

and has the lowest insulin requirement of any day you can think of.

And that's a ketogenic diet, some people here may have heard that term before.

Or even read a book about it, I don't know... Jimmy?

This is your basic ketogenic dietary-meter.

Eating meat, eggs, nuts, green vegetables, low sugar fruits.

All you're really skipping is the grains, sugars and starches,

this ridiculous glucose producing foods that nobody should be eating anyway.

Not everybody thinks ketogenic diets are a fad,

but I am pretty sure your hunter and gatherer ancestors were in ketosis all the time,

especially during ice age, which is a half million years...

Especially during the wintertime, anytime we didn't have a ton of carbohydrates.

So now I have a word of caution about low-carb ketogenic diets.

You can do it wrong, it's possible to do it wrong.

You could drink corn oil all day, just for an extreme example.

This would be low-carb, this would be ketogenic, but it's not good,

it's bad, it's nutrient poor.

So what you really want to do is minimize your energy exposure

by eliminating empty calories.

The real cause of hyperinsulinemia is empty calories.

That's both refined carbohydrates, grains, sugar and starches,

but also industrial seed oils, which are totally empty calories.

There's this idea of caloric restriction with optimum nutrition,

or the C.R.O.N. spectrum.

You've got at one end a really high energy density.

A low nutrient density would be drinking corn oil.

What you really want is the opposite end of that with a maximum nutrient density,

which gives you the least amount of energy exposure and empty calories.

And that's going to get your insulin level at the very lowest.

You could have bariatric surgery,

we have studies that prove that bariatric surgery really slashes insulin levels.

But I don't recommend that because it's a dangerous,

painful, expensive, $20,000 version of ketogenic diet plus intermittent fasting,

you might want to try that first.

Let's talk about intermittent fasting for a second.

Most people are eating breakfast, lunch, dinner, snacks, snacks.

There are always in the fed state, they're never in the fasted state.

Their insulin is always high, they're always partitioning calories into storage

Here is a standard meal set up, breakfast, lunch, dinner in a 12 hour window.

I recall from way back our first slide that takes 12 hours after you eat

for your insulin level to be down the baseline.

When is this person ever at baseline insulin and resetting their insulin sensitivity?

Maybe first thing in the morning for about 30 seconds.

So you could shrink your eating window, we call it time restricted eating.

You can take any obese or diabetic mammal, a rat, a mouse, a dog, a cat, a human.

Shrink their access to food from 24 hours/day down to 8 hours/day,

you will immediately lower insulin level, improve insulin sensitivity,

lower fat mass, lower blood sugar, reverse diabetes.

The easiest way for humans to do this is just extend your overnight fasting,

skip breakfast, push your first meal out for five hours,

eat lunch and dinner in an eight hour window.

Really good for insulin sensitivity just spending 16 hours in a low insulin state.

The ultimate form of this is the Warrior Diet, popularized by Ori Hofmekler in his book.

You fast or under-eat all day,

then you eat a giant meal at the end of the day.

This makes insulin really pulsatile, which is what it's supposed to happen.

Big spike of insulin, then you don't eat for a really long time.

Insulin gets really, really low - you reset insulin sensitivity.

We have tons of data now on alternate daily fasting,

which is basically eating once a day

and you can literally cure almost any type 2 diabetic by just eating once a day.

It's phenomenal for insulin sensitivity.

As hunter/gatherers, this is probably the way we were supposed to function.

You didn't just roll out of bed and eat a huge breakfast.

You were hunting and gathering in a fasted state.

Got a bunch of food together, eat as much as you could

and then you maybe didn't eat again for a long time, I love this pattern.

There is a bunch of health benefits from fasting.

You'll have to wait for Jimmy's book for all of those.

This is a great study, carb restriction versus fasting.

If you restrict carbs, you get about 72% of benefits of fasting.

Here is a standard diet, low-carb diet, fasting for blood sugar response.

Low-carb is about 71% as good as fasting.

Insulin response, standard diet, low-carb, fasting...

Insulin response about 72% as good on low-carb versus fasting.

Exercise - exercise is huge.

The higher physical activity, the lower your insulin level.

Here is another study - aerobic exercise in 12 weeks

improved insulin sensitivity by 35% in lean people

and 59% in obese people.

Here is a strength training study in obese youth.

And in just a couple of weeks they improved their insulin sensitivity significantly.

It turns out the more muscle you have, the lower your insulin level. Period.

Muscle is awesome.

The more muscle you have, the lower your insulin level.

This is another study on exercise

and they figured out that more is better in every way.

The higher the volume, the higher the intensity,

the higher the duration, the lower your insulin level goes

and the higher your insulin sensitivity.

And it turns out that all the benefits you get from fasting

are just sped up with exercise.

So low-carb is good, fasting - slightly better,

exercise - slightly better than that.

I put everything together on one graph here

and you've got in red everything that raises insulin.

You've got eating carbs, eating carbs with fat,

eating frequently empty calories.

And then in green you have everything that lowers insulin.

Carbohydrate restriction, intermittent fasting, high-intensity exercise

and of course nutrient density, maximizing nutrient density.

Now there are a bunch of people out there who say,

"I eat a high carb diet and I am thin and I'm insulin sensitive

and I reverse my diabetes with high carb..."

Those people think low-carb is dumb.

Now I just have this slide to point out that you can do all those things without low-carb.

But it's really not optimal.

So let's say I'm on a high carb diet.

Here is glucose coming in from my diet,

it's been stored as glycogen in my liver and my muscle.

Eventually my liver is full of glycogen, my muscle is full of glycogen.

I could just stop eating, I could use a bunch of willpower

and minimize my calories and just stop eating and I won't get insulin resistant.

That's true.

But if I continue to eat a bunch of glucose, because it's delicious,

what will happen next, my muscle's insulin resistant.

It says, "No more glycogen, we're full."

My blood sugar is still too high, so my pancreas pumps up more insulin.

Now my poor liver has to convert all that glucose into fat via de novo lipogenesis.

I've got fatty liver, I have high triglycerides, I have low HDL.

Now I'm fully insulin resistant, liver and muscle.

Well, fasting will deplete your liver of glycogen and fat

and restore your liver sensitivity to normal.

And high-intensity exercise will deplete your muscle of glycogen and fat

and restore your muscle insulin sensitivity to normal.

And that's how you can reverse insulin resistance

with fasting caloric restriction and high-intensity exercise,

even if you are still eating carbs and glucose.

So it's possible to do that.

Possible does not equal optimal.

So if you really want to be optimal, you want to do all these things.

This is my last slide, I don't want to run over.

I put all together here.

There are basically four things you want to do to minimize insulin resistance.

The first one - maximize nutrient density.

The real problem with all obesity, diabetes, hyperinsulinemia

is empty calories - you have to get rid of empty calories.

Just only eat super foods.

If your whole diet is just Wildcats spinach and pastured eggs

and Brazil nuts and salmon,

you're definitely not going to have any problems with hyperinsulinemia.

The second thing you want to do is minimize glycemic impact.

Carbs were helpful back in the day where we starved every winter.

You got carbs in the summertime.

These raised insulin on purpose,

it made you insulin resistant so you could eat as much as possible

and store up enough fat to live during the winter time.

Now we've got whole foods down the street.

So you don't have to spike your glucose and become insulin resistant

to survive the winter time - so stop doing it.

Keep insulin pulsatile, fasting and feasting.

You want to eat a bunch of food and then go a really long time without eating.

This is what we're supposed to do, it keeps insulin really sensitive.

And finally - exercise, high-intensity exercise.

You want as much muscle as possible, you want as many mitochondria as possible.

If you never exercise, you might just have one mitochondria in your whole body.

And I don't care how fat adapted you are and how low-carb your diet is,

if you only have one mitochondria to burn fat,

you're never going to burn enough fat.

So you do a push-up, now you've got two mitochondria.

You do another push-up, now you've got 4, and then 8, and then 16.

So you want as much muscle as you can get, as many mitochondria as you can get

and exercise does that for you.

So in summary, there are four things you want to do

to get your insulin as low as possible...

Nutrient density, low-carb, fasting and feasting - intermittent fasting,

and high-intensity exercise.

And I will leave you with the ketogenic diet pyramid.