Great turnout.
Thank you all for coming tonight.
It's a real pleasure for me to
introduce one of the superstars of the Paleo,
or ancestral movement, as we call it.
I know for many of you,
Robb needs no introduction tonight,
but for those of you who are new to his work,
let me tell you a few things about his remarkable resume.
Robb is a former powerlifting champion
of the State of California.
He's co-founder of
the NorCal Strength & Conditioning Gym,
which was rated one of
the top 30 gyms in the United States.
He's also the co-founder of the training and
nutrition journal called Performance Menu.
He is also, of course,
the New York Times bestselling author,
The Paleo Solution.
We'll have copies of the book available to
purchase in the sign afterwards at the reception.
I would invite all of you to come to the reception in
the terrace immediately after the talk.
Of course, Robb is also known for
his top ranked iTunes podcast,
which has more than six gazillion listeners every week.
Tonight, Robb will be speaking on the topic of Darwinian medicine, asking us, is there something to this evolution thing? With that, please join me welcoming Robb Wolf. How are you-all doing? Apparently, evolution is pretty damn popular.

I'm just surprised here.

Thank you-all for coming.

A huge honor to be here.

I just can't reiterate that enough.

I have a thank you slide at the end, but upfront, thank you.

To Professor Stapell, to the EvoS program, thank you to Dr. Mat Lalonde.

Is Mat here yet? He's going to be here.

Mat helped me a ton on several talks, this talk included, so thank you all.

Gosh, it's just been such an amazing ride with all this.

I first got exposed to this idea of evolutionary biology as it relates to health and wellness by dealing with a health concern that was probably going to kill me.

I had tinkered with my nutrition many years ago.

It started eating a high carb, low-fat vegan way of eating.

For me, it didn't work.
I had a host of problems, ulcerative colitis, high blood pressure, just all kinds of wacky stuff going on, and it was an idea of applying evolutionary biology, like applying an ancestral diet to my situation that ultimately saved my life, that was almost 15 years ago, that was 1998. It's led me down this road of doing a research fellowship with Professor Loren Cordain, opening a gym, and running the gym, and using the principles of evolutionary biology; sleep, exercise, nutrition, socialization, the deep need for socialization and community has been just indispensable to our success. It definitely has been the driver to the success of the book, the podcast. We just keep putting out this message of using this evolutionary template as a hypothesis generator, really. That's going to be the way that I couched the talk today, is using the evolutionary template to ask the right questions. When I start off here, I'm going to actually do a little diversion into another topic, into economics just a little bit as a springboard to get
into this evolutionary biology topic.
Right before I do that though,
anybody know the dude in the green? Anybody know that guy?
Aaron Rodgers won a Super Bowl a little while back.
When ESPN interviewed him,
they asked him what he was taken to the Super Bowl,
and he was taking a copy of
the Paleo Diet and a guitar, so that's cool.
Aaron is actually from Chico,
he's from the town where our gym is, so cool stuff.
We're going to start things off with a little bit of,
I'll call it soft science or
end up getting beat up by somebody
out of the humanities section.
But as a biochemist,
I'm still looking at,
even though I consider myself an armchair economists,
and I really like geeking out on that,
just the rigor that we can bring to bear on
different topics relative to physics, or engineering,
or chemistry, the humanities are much more complex,
so it's a little bit more difficult to pin down
a cohesive thought on the whole thing.
But I'm going to kick this off with
his concept of Moore's Law,
which Moore's Law is
this idea that was born in the 1960s,
that about every 18 months,
computer processors double in speed,
but half in price.
They get twice as fast and half as expensive.
If you look at the computer that you're using today,
it's much faster and much
cheaper than what it would have been five years ago.
This is a concept that we can apply across,
not just computing, but a whole host of things,
you type of technological innovation.
Typically, if we understand more about that innovation,
things both get better with
regards to quality of products and services,
and cheaper with regards to how much that costs,
both in real terms and in what's given to the consumer.
I guess, I just getting eaten up there.
We see the cost per unit computing
power versus actual computing power,
and we have this dramatic drop in the cost.
Back in 1980, a gigabyte of
processing would have cost about a million dollars,
so it's not that long ago.
I definitely remember that,
I remember being in school.
Some of you guys may be post 1980 and birth or something,
but it's not really that long ago.
Out here, over on this side,
we have a gigabyte of
processing being about a million dollars today,
a gigabyte of processing is less than $0.10.
It is so cheap that that gigabyte of
processing is worth more as an advertising medium,
than it is as the item that it was intended for.
This is that thing, again,
where we understand more,
the product gets better,
and things get cheaper.
This is, again, across all disciplines,
getting more into my wheelhouse,
DNA base pair sequencing,
when we start thinking about like
the Human Genome Project and
various genomic projects trying to
map the genome of different organisms.
In 2001, it was about
$10,000 to sequence one megabase of DNA.
Today, it's less than $0.10.
It's almost free, and this is actually
an artificially extended scale.
It's a logarithmic graph,
this really just drops.
It's like falling off of the cliff.
The reason for this is that
our understanding of genes, genomics, the technology involved in gene sequencing has exploded to such a degree that it is damn near free to sequence the genome of a critter, like we can sequence a human genome for about $1000. Maybe a little bit more, maybe a little bit less, but it's streaking towards being free for all intents and purposes. This is in contrast to our current health care expenditures. If we remain on the vector that we are doing, by 2050, we will be spending 300 percent of then projected GDP on healthcare. If that doesn't give you a gut check, it should. I have my firstborn child arriving in late April and it makes me just have like a watery stool standing up here saying that [LAUGHTER], so this is terrifying stuff. Now, when we talk about the hows and whys with this, folks will talk about demographic shift. Our population is getting older, yeah, it is getting older, and that is insignificant contribution to the increases in costs, healthcare costs more, period. It shouldn't. We know more, we understand more,
we're mismanaging things, we have
a market-based things that are not working,
and I don't want to get too
political about all that stuff.
But on the implementation side,
on the medical side,
we are making recommendations that are wrong.
The vast majority of that increase in healthcare cost,
almost all of it is attributable
to obesity and obesity-related conditions.
We know more about Biology,
we know more about genes,
but things are getting more expensive,
so just to recap that whole story,
when technology advances, good and
services improve, cost decrease.
We know more about Biology,
Genetics, Pathology than we ever have.
Costs are going up,
quality of care is going down, and disease rates,
especially related to obesity
and related diseases are increasing.
This is stupefying if you really overlay this
against any other technology,
getting minerals out of the ground is
cheaper today than what it was 20 years ago.
Because we know more about that process,
we are more efficient to that process,
so the healthcare increases and the
fact that we have disease rates increasing,
I would go out on a limb and
say that we might be doing something wrong,
and that limb would further extend in the thing that I
would argue that we're doing wrong
is that when we tackle medicine,
when we tackle health, we're tackling it from
a position that is not steeped in evolutionary biology.
When we look at a well-defined,
a well developed science like physics,
we have basic characteristics and
epistemological underpinning
built out of Newtonian physics,
and then quantum mechanics,
statistical mechanics, not that long ago,
just a little over a 100 years ago.
Things like the quantum electric effect,
the photoelectric effect, Brownian movement,
the way that molecules move in a solution,
we had no explanation for these processes.
It wasn't until the development of quantum mechanics,
that we had an explanation,
we had an epistemological structure to
overlay what we were seeing empirically,
what we were seeing in experimental science.
But up until that point,
most of the new physics,
most of the things that we were dealing with,
with regards to light interacting with matter,
gravitational effects and things like that,
we had no model for describing it.
Now, that may not seem significant,
but every time you use your cell phone and it
has some sort of a GPS synchronization with a satellite,
there's an adjustment in
the communication between the phone and
the satellite because we being near
the surface of the Earth or in a deeper,
more dense part of the gravity.
Well, that the matter of the Earth
creates relative to the satellite,
and there is a quantum effect
that needs to be adjusted for that.
Otherwise, we would be in the wrong spot
according to the satellite relative to the phone.
Every single day when we use a GPS phone and a satellite,
we are benefiting from
these fundamental principles of physics,
and if we didn't understand those fundamental principles,
something as fundamental as an iPhone would not work.
It would be impossible for the features
with which it provides to work.
Again, the reason why this stuff is working is because we have this foundational epistemological structure in physics, and chemistry, and engineering, we have this guiding tenet in biology. The guiding tenet is evolution via natural selection. My wife speaks Russian, so I'm going to do my best with this. Theodosius Dobzhansky, said that nothing in biology makes sense except through the light of evolution, so we're pretty clear on this and we've made a lot of advances in various elements of biological scientists, but we don't really have it in medicine. There is not yet a Darwinian medicine program, but shoe dietitians and physicians do not receive a formalized training in Darwinian medicine or evolutionary biology as part of their training, and I think that's a huge problem. That would be like physicists and chemists trying to do their work without quantum mechanics and statistical mechanics and all of that stuff. I think that we're operating in a world where we have lots of information,
but we don't have an organizing theory to put things together with.

This is kind of the orientation that I'm going to present as we go along here, it's always nice to ask the question too, or humans exempt from evolution, I would again go out on a limb and say, no, we're a part of nature, were a part of biology.

That is another thorny topic on this evolution piece because there are a lot of folks that don't think evolution applies to humans, so that can be a problem.

The early human timeline, we start seeing australopithecus, robustus, and gracilis about two million years ago, 2.5 million years ago, we have a long history of human and pre-human ancestors that seemed to transition from a more frugivorous and herbivorous diet to scavenging based economy, to a hunter-gatherer economy, and we definitely have the opportunistic omnivore in us.

We exploit a wide variety of resources and environments, but the lion's share of our time biologically is over here in
this Homo erectus Neanderthal.
That's when significant changes in our genomes,
significant changes in our neural structure
appear to have been formed,
and there's all kinds of interplay with that;
social groups, languages, sharing of information,
increasing nutrient density of the diet,
potentially providing
substrates to increase encephalization rates,
grow a bigger brain, there's
lots of stuff that goes into that.
But the basic idea here is that
organisms evolve to be very,
very successful at
the particular biome that they exist in.
That changes to that environment
could potentially be problematic.
It can also be advantageous.
You can find lots of examples in biology when, say,
like an evolutionary novel food is
presented to an organism and it's been official.
So we need to be careful with this.
I'd made this mistake before and I
will say something sloppily,
and say that an evolutionarily novel food is
bad or potentially problematic.
Olive oil is new.
It's evolutionarily novel.
It's great for us,
so it's good stuff.
So just because it's new
doesn't necessarily mean it's bad.
But my orientation with this is that,
if we have problems,
let's say that we have problems
with Type 2 diabetes, and obesity,
and systemic inflammatory diseases,
maybe we should look at
the ancestral genomic environment for a place to find,
to start looking for answers.
That's not necessarily the answer but
it's a place to start looking for answers,
and to use evolution as a hypothesis generator.
So that's where my orientation is with all these stuff.
With that in mind, if we
look at anthropological observations,
we know that contemporary hunter-gatherers and
ancestral hunter-gatherers were largely
free from modern diseases.
We didn't see much in the way of cancer,
diabetes, heart disease,
bone demineralization,
the transition to the agrarian life way.
Source and pretty remarkable changes in health;
a loss in height,
dental caries, increase infant mortality rates.
If you go over and talk to the anthropology department,
this stuff is all pretty well-known.
If you talk to nutritional sciences department,
it's completely unheard of.
Again, this isn't proof,
this is an interesting observation.
It's a place to maybe start
having a conversation about this.
But I liken this whole process
to what I call the Russian literature paradox,
which here it is.
Let's imagine we're having
discussion about Russian literature,
which would be almost as much fun
as probably talking about physics at this point.
But if we're having
a discussion about Russian literature, let's say,
I asked the group,
what's your opinion about the sentence structure and
syntax of mid 18th century Russian literature?
How many of you would be able to
posit an opinion about
mid 18th century Russian literature?
One dude. There's always got to be
one. There's always got to be one.
[LAUGHTER] Generally, not a lot of folks though, but the interesting thing with this is that when we start talking about human biology, and diets, and the changes from the hunter-gatherer life way to the agriculturalists life way and then the post-industrial life way, folks have lots of opinions despite the fact that they have no education on the topic. So it's a stupefying process for me that, again, this isn't proof of anything. The fact that hunter-gatherers lived a particular way, and that they may have been free of certain diseases, doesn't prove anything, but it's damn interesting to me. When we start seeing problems in our modern way of living, looking back, if we just simply asked, if we look into our past, do we see a lack of disease at different points when we were doing different things? If not, then that's a bad place to look, I guess. But when, in fact, we look at our ancestral lineage, we see both contemporary and archaic hunter-gatherers that don't really show the signs and symptoms of disease that we see today. So it's maybe a good place to start asking some questions,
and that question would start off, could changes in the ancestral environment cause pathogenic epigenetic shifts?

We have our genome, and then our epigenome, is the protein expression basically interacting with the environment. It's the interaction with the environment that manifests in who we are. So some way that we're interacting with the environment could change that is either adaptive or maladaptive, and we'll maybe look at some maladaptive changes that we have.

I gave a very similar talk to this one to a couple of hospital systems, and the way that I lead in with those was talking about exercise, because the exercise piece of the evolutionary biology picture is largely uncontroversial. People usually buy into it fairly easily, and then use that as the skinny end of the wedge to then throw the food through the door, and then shut the door and let them freak out about it afterwards.

[LAUGHTER] So throughout our ancestral life way,
we were very, very active.
We hunted and we gathered.
That was our base economy.
There was all kinds of interesting stuff;
reciprocity, altruism,
optimum foraging strategy, there's lots of theories that
tie elements of that hunter-gatherer life way together.
But the base unit is that throughout
the lion's share of human evolution,
we were quite active.
In the process of being active,
it wasn't just a matter of burning calories,
like you would throw a log into a fire,
but we were changing the way that
our genes turned on or off.
We changed the way that the proteome,
the gene expression products
were expressed in our bodies,
and we tended to see either an optimization,
a healthy scenario that
was usually tied in with activity,
or we're going to see some problems
when we have reverted to a condition in which we
literally can exist without
exerting much in the way of effort at all.
We can order food into our house,
we can work from home,
and we can do virtually no activity at all.
The supposition here is that there
might be some problems with that.
One of the best papers that I've
ever seen on this topic, Frank Booth;
exercise and gene expression,
physiological regulation of
human genome through physical activity.
This is just an amazing paper.
When we think about this,
when we remove exercise from the ancestral perspective,
we actually have a silencing of certain genes and
then an over-expression of
other genes as a consequence of sedentism.
So that might be good,
that might be bad but it's very, very different.
Most hominids expand about 200 kilo-joules per kilogram.
Currently, we're at about a 130.
There's a lot of interesting stuff in
the literature not really linking
exercise with improving obesity
and improving different things,
but there is lots of data linking exercise and health.
Before we talk about that,
a good question to ask is,
what is the normal ancestral phenotype?
We have our genotype which is our genes.
The phenotype is how we manifest those genes. When we look at the archaeological record, when we look at the anthropological record, the norm for our species is to be lean, strong, and healthy. That is the norm.

Even though we had a short average lifespan, which is one of the common counterpoints to this whole ancestral way of eating, even though we had an average short lifespan due to infections, and injury, and stuff like that, if individuals lived into their 60s or 70s, they tended to be very, very fit and healthy. We see this both in skeletal remains. We also see this in contemporary studied hunter-gatherers like the [inaudible 00:22:04] which is one of the guys on the side. Running the scale is actually Professor Stefan Lindeberg at the University of Lund in Sweden who spoke at the health symposium last year. Amazing guy, and is just one of the geniuses of this whole scene. So the way that gene silencing can become a problem when we decreased physical activity,
we can have a failure to manifest certain gene products,
which are basically proteins
that can either be beneficial,
negative, or something in between,
but it usually reaches some sort of a threshold,
and classic example of this is cardiomyopathy.
When we are active,
we have left ventricular hypertrophy.
The left ventricle of our heart increases in thickness,
increases in size, and it's a very beneficial adaptation.
They call the normal heart, the unathletic heart.
It's actually this small,
puny heart of just sitting
around doing nothing when in fact,
it's pretty clear that our phenotypic or
genotypic optimum is an active heart
that is enlarged and healthy.
But an interesting thing that happens is that,
if we are sedentary,
a certain percentage of the population
develops cardiomyopathy,
a pathological left ventricular hypertrophy
that will kill them eventually,
and the best explanation of that that I can give you,
is that when that heart signaling,
it's trying, I say trying,
I don't want to anthropomorphize genes,
but we'll say trying,
it's trying to find its way.

There's an expectation of being healthy,
of being an enlarged heart,
but without that signaling of exercise,
we get an inactivation of
healthy gene expression and
activation of pathological gene expression,
which in this case would manifest in cardiomyopathy.

Lots of cancers, lots of other disease states,
seem to come about from this.

There's a pretty good theory that
a significant amount of our glucose disposal,
the way that we store glucose,
should come about the exercise.

When we exercise, we have
a non-insulin mediatic glucose transport
that moves glucose into the muscles,
and so if we are active throughout the day,
we don't need to rely on our pancreas
to produce insulin all the time,
our activity level actually helps to
regulate our glucose disposal.

There's lots of things woven into activity, and usually,
people are pretty open to
the idea of hunter-gatherers being active,
that the ancestral life way was active,
but all hell breaks loose when you start talking about food.
So people will sign off on the idea that we hunted and gathered,
but then when you bring up what we actually ate when we hunted and gathered,
then there's all gnashing of teeth and problems that arise from that.
Characteristics of the ancestral diet, it was largely grain, legume, and dairy-free.
Now, that doesn't prove that grains, legumes, and dairy are problematic,
but it's again an interesting observation.
We keep pushing transient grain consumption back.
Like I think they found a spot that showed grain consumption maybe 100,000 years ago now.
But when we look at stable isotopes studies, what people were eating consistently day-to-day,
we can determine how much animal protein versus plant material folks were eating,
whether it was aquatic or terrestrial in origin, and when we look at that stuff,
we just don't see a ton of plant-based proteins being consumed.
When we consider optimum foraging strategy, there's some reasons for that.
But largely the ancestral diet was free of grains,
legumes, and dairy, local seasonal fruits and veggies, roots and tubers, lean wild meat, fish, fowl, anything that creeps or crawls, nuts, and seeds as available. Macronutrients varied with the season and locality. Even in equatorial regions, you have a coming on and a coming off of different fruiting seasons, animals will move in and out of an area, so it's definitely not a static nutritional profile. It's very dynamic. Changes over time, changes with location. Optimum foraging strategy is this idea and it's funny, how many folks saw the eye caveman gig? Did people see the eye caveman gig? I did the Discovery Channel show where they gave us some rudimentary survival skills and a bunch of stone tools that we had to make and we had to live for two weeks as hunter-gatherers, and you discover really quickly what an optimum foraging strategy is, it's not spinach and lettuce and stuff. If you can find tubers, those are really good, but it usually boils down to big game hunting and staring small animals.
The optimum foraging strategy is one of those pieces of evolutionary biology applied to any type of organisms, food procurement that really clears up a lot of hows and whys about what they could have eaten. Cattle and other ruminants spend all day eating and they tend to have fermented digestion, which is very different than the type of digestion we have. We do really well on nutrient-dense foods. Great paper to look at that American Journal of Clinical Nutrition, plant-animal substitution for ratios and macronutrient energy estimations of worldwide hunter-gatherers, Loren Cordain classic. Where I think that we use this template, we can use anthropology as an observational tool. We can ask some questions based on anthropology, about, what did people eat or not eat, what were the relative health conditions of folks, did agriculturalists gain or lose height, did agriculturalists have better or worse teeth, did they have more or less infant mortality? We can use that to ask some questions. But then we can use epidemiology and clinical interventions to start investigating certain hypotheses that we generate,
and then from there, we need to find
some molecular mechanisms to
tie the whole story together.
We should see some self-similarity,
whether we're talking about the anthropological story,
clinical interventions,
or proposed mechanisms of causation.
We should see consistency throughout this picture,
instead of the anthropological story looking very
different than what we're looking
at with regards to mechanism.
An example that I'll use with this are
the Kitavans and the guy on the left,
he's 100 years old,
102 years old, I think in that photo.
This is a very well-studied population.
I think that they've had Western anthropologists and
medical professionals living amongst them since 1929.
They perform autopsies, they weigh and measure
food that they hunt and gather and bring in.
They're a very well-studied population,
which again goes back to that Russian literature paradox.
Like we do in fact know a lot
about these types of folks and other folks.
They have a traditional diet built around yams,
taro, bananas, fish, pork, and coconut.
I liked the Kitavans because
depending on virtually anybody you talk to, these folks should be dead. They should just keel over and die. They eat a 15-25 percent animal protein diet.

If you're out of the T. Colin Campbell, China's study camp, these people should be just way by cancer, but they see virtually none. I mean, none. Their primary fat is saturated fat.

If you're out of the American Heart Association camp, these people should be keeling over and dying from cardiovascular disease. They have none.

As an aside, 75-80 percent of the adults in the Kitava are seen smoking. Percolate on that for a second. [LAUGHTER]

If you're in the lunatic fringe, low carbohydrate Paleo camp. They eat a 60 percent carbohydrate diet, so they should be fat, diabetic, and keeling over and dying from metabolic derangement. If you're in the Gary Thompson, insulin spiking causes metabolic derangement scene but in fact we see none of that.
These people live long healthy lives. They're largely free of cancer, diabetes, and heart disease. This has been extensively well-studied since the 1920s. This is what we see in virtually any Aboriginal population that we look at, a hunter gatherer population. It's interesting, something started to change. These folks started eating westernized foods and they started developing cancer, diabetes, and heart disease. The macronutrients didn't change. Amounts of protein, carbohydrate and fat didn't really change, but the quality and the source of these macronutrients did change. They started getting things like refined vegetable oil, which is high in linoleic acid, short chain omega six. They started getting carbohydrates from grain sources, which I'm going to make an argument are pretty problematic for a variety of reasons. What we'd been doing in most of nutritional science in Western world, we've been doing nutritionism to quote Michael Poland where we look at protein or carbohydrate or fat or vitamin B versus vitamin
D. We've been doing a very reductionist story on the one hand. On the other hand, we look at macronutrients and we just keep shuffling a card deck of protein, carb, fat. Should we be high carb, should we be low-carb? What we're finding, we have anthropological examples of the Inuit who eat a very high fat, moderate protein, low-carb diet that are largely free of cancer, diabetes, and heart disease. They start eating a westernized diet, they developed cancer, diabetes, and heart disease. We have the Kitava who eat a high carb, moderate protein, low-fat diet, who are largely free of cancer, diabetes, and heart disease. Eat a westernized diet, start developing cancer, diabetes, and heart disease. For the human animal, we can probably survive on a wide variety of foodstuffs but the argument here that's going to come down as that maybe something about the types of foods that we're eating might be problematic and this is straight from Professor Lindeberg.
This is none of my stuff.
I've got to give him all credit on this,
but he made these two points.
Foods are appropriate for any given species if they were
regularly consumed during most of its prior evolution.
You tend to have some mapping of
the evolutionary biome to
an organism and then plants protect themselves of
bioactive substances directly aimed at animals.
Substances which may have
untoward effects on long-term human health.
I would say health of any organism when you
have a potentially evolutionarily novel substance.
This first paper, agrarian diet
and diseases of affluence,
do evolutionarily novel
dietary leptons cause leptin resistance?
What we're going to do is use some papers and
these ideas from Professor Lindeberg to
maybe build a case that
some evolutionarily novel foods have
some constituents in them which are problematic.
A key thing to keep in mind is that
not all new foods are going to be problematic,
but where we see problems arise,
maybe that's a smart place to look for some problems.
Real quickly, lectins are sugar binding proteins.
Really, really important in cell surface recognition, telling self from non-self.

Some of them are actually therapeutic.

Banana lectin is actually effective in mitigating the effects of HIV-1.

Some are pathogenic, ricen is used in chemical warfare agents, I believe from castor beans, soybean antigen, and some others are pretty nasty on an immunological level.

Leptin is an adipose derived neuro hormone. It is super important in the neural regulation of appetite and also in fat mass.

We've looked at tonic insulin. I think that anything that you say about insulin, you could probably say about leptin with regards to insulin sensitivity, leptin sensitivity, body fatness, energy levels. What we understand now is that leptin resistance, the inability for your body to sense leptin normally seems to proceed insulin resistance and is probably the precipitator of that insulin resistance and all the associated stuff, type two diabetes and all that stuff. When we get in and test this hypothesis, we have a hypothesis that evolutionarily novel leptons,
Leptin resistance tends to manifest in metabolic derangement, type two diabetes, cancer, a whole bunch of stuff.

They looked at large differences in serum leptin levels between non-westernized and westernized populations, The Kitava study.

What they did with this is they looked at 200 Swedish, folks from Sweden, 200 Kitavans, did a number of bio-metric analyses on them. One of them being leptin levels.

What they found was that the leptin levels in the Kitavans were much lower than in Sweden. Macronutrients weren't really that much different. The amount of protein, carbohydrate, fat weren't that different. The amount of energy density wasn't that different. One group wasn't eating significantly more than another.

We have this observation that folks that are eating westernized diets are tending to show higher leptin levels and that tends to be very highly correlative and maybe causative of insulin resistance type two diabetes and that sort of stuff.
Not proof by a mile, but interesting.

It's supporting this idea that these evolutionarily novel foods might have some problems.

The next step down the road, and the interesting thing is I was actually a review editor for this paper testing the hypothesis part two.

Paleo diet confers higher insulin sensitivity, lower C-reactive protein, and lower blood pressure than a cereal based diet in domestic pigs.

What they did with this, they put 24 pigs on a Paleo or a Mediterranean type cereal based diet. What they found was that in the Paleo diet group they had much lower C-reactive protein. C-reactive protein is an indicator of immune activity. If you have a cold, a flu, virus, any type of long-term pathogen, you will have elevated C-reactive protein. It's an indicator that your immune system is fired up. If you have a cold or the flu, we should expect to see elevated C-reactive protein. If you don't have a cold or flu and we see high C-reactive protein, we've got a problem. You've got systemic inflammation and over
activity of your immune function. It's bad news.

In the pigs fed the Paleo diet, they had much lower C-reactive protein than the Mediterranean cereal based diet. The Paleo group had better phase two insulin sensitivity, which means the insulin sensitivity at the muscle level was better, which is very correlative with a good leptin signaling and then no pancreatic leukocyte infiltration. How many of you all know someone with type 1 diabetes? Quite a number of people. Type 1 diabetes is an autoimmune disease and what happens by a variety of mechanisms, our immune system attacks the Beta cells of the pancreas, destroys them and destroys the ability to make insulin. What we're seeing with that pancreatic leukocyte infiltration is actually in the serial based group, is a very high likelihood of developing auto-immunity. I'm going to talk a bunch here in just a minute about the auto-immune process. But we're seeing some pretty remarkable differences between a paleo diet group in which they ate basically lean meats, fruits, vegetables, roots, tubers. They did not eat grains, legumes, and dairy.
The Mediterranean group ate lean meats, dairy, cereals, legumes, stuff like that. Typical quasi-vegetarian type diet.

Now we're doing this in humans. Testing the hypothesis, paleo diet improves glucose tolerance more than a Mediterranean type diet in individuals with ischemic heart disease.

What they did with this, they had 14 folks in a paleo group, 14 folks in a Mediterranean diet group. The interesting thing, these folks were type 2 diabetic heart patients. They were heart patients defined as either having had a previous cardiovascular event or high risk of a cardiovascular event.

They put one group on a paleo diet, educated them about a paleo diet, another group on a Mediterranean diet. Then they were allowed to free. That's one of the limitations of this study, is that it wasn't a metabolic ward. We're actually looking very soon doing a similar study to this under metabolic ward conditions.

But despite that rigor, we still see some interesting results. The paleo diet group is over here and they saw
a 28 percent improvement in oral glucose tolerance.

Basically, the paleo diet group was no longer
type 2 diabetic after an eight week intervention.
The Mediterranean diet group showed
a barely statistically significant improvement
in their oral glucose tolerance,
which this was shocking to me
because you would expect that people being put on,
any dietitian would love that Mediterranean diet.
Low-fat dairy, low-fat protein sources.
Whole grains, whole legumes.
But yet they had a barely statistical significant change
in their oral glucose tolerance and also
in signs and symptoms and metabolic derangement.
It's pretty interesting stuff, but again,
not the final word on any of this.
As we're tracking through here,
we have a conspicuous lack of
cardiovascular disease in the anthropological data.
Just forces us to ask that question, why? Why is that?
Why are we seeing not much cardiovascular disease,
for example, in anthropological record relative to now?
Then we start developing some hypotheses.
The Catawba study looked at
epidemiology from Catawbans relative
to a westernized population.
Then as we start moving into mechanistic testing,
we start comparing different parameters
under animal conditions and then human conditions.

But there's one problem with
where we're at with this story.

Lectins are largely inactivated by cooking.

We aren't getting as large of
a lectin load is what we once thought.

This was something that I was super tied into.

We also haven't been able to find
a strong mechanistic action of lectin on leptin.

But we do see huge clinical benefits from a paleo diet.

We see huge improvements in auto-immunity,
metabolic derangement, insulin,
leptin signaling and what not.

What I've arrived at and
what a lot of other folks have arrived at,
we looked at lectins, no,
this thing is not working at all,
we've looked at leptin are lectins,
they may not be the culprits,
but there is a bunch of
other proteins that probably are the culprits.

The idea was good.

The specific thing that we looked
at hasn't really panned out,
but it's very, very close.

We're still looking at proteins associated
with these evolutionarily novel foods, particularly grains being potentially very, very problematic. We're going to look a little bit at a immune function, and we're going to look at the etiology of auto-immunity as it relates to adaptive immunity, and we're going to see how that comes about from these evolutionarily novel proteins and causing problems with the gut. We're going to look at innate immunity, which is an adaptive just responsive immunity that's involved in initial stage of immune response. It's involved in allergies and stuff like that. We're going to see where some of these new proteins might be problematic. How many of you have heard of gluten? Most folks have heard of gluten. Yeah. This is gliadin. It's a protein found in gluten and what we find is a very dense prolene rich structure. The prolene is this wacky, big amino acid. When you put a lot of prolene in a protein, it creates tons of twists and it makes it very, very difficult to digest. It makes it a gnarly irritating chunk of protein. What we find with just gliadin alone, and there's lots and lots of protein
in wheat specifically,
we see a lot of
different potentially immunologically active constituents
in just this protein alone,
largely wrapped around to
amino acids of glutamine and then also prolene.
Professor Alessio Fasano has done a ton of work on
celiac disease and has proposed celiac disease
as a mechanistic description
of potentially all auto-immune diseases.
Basically starting from a gut permeability level.
What we have is
undigested gluten or gliadin
protein particles and they're
undigested because the tight turns induced
by the prolene causes these proteins to remain intact.
They interact with the CX-R3 receptor
on the enterocyte of the intestinal lumen.
That causes that protein fragment to be pulled
into the enterocyte and it releases a hormone zonulin.
Zonulin for whatever reason,
and this is a big question.
Matt Milan and I were talking about this last night.
Other primates do not develop auto-immune disease.
They don't release zonulin.
It's really interesting things.
Somewhere along the line,
we developed zonulin and this is totally off the cuff.
I was just talking to him about it last night.
I know conclusions about this.
It's really interesting, but we're in
the evolutionary studies section here.
What role does zonulin play in human evolution?
Does zonulin exist in other carnivores?
It doesn't exist in other herbivorous primates?
So somewhere along the line,
we had a significant change in
our gastrointestinal physiology that involves zonulin,
and what zonulin does is it
dissolves the tight junctions between enterocytes.
If you think about intestinal contents,
you probably want to keep that out of your person,
and having the cells stick together very tightly is good.
What gliadin does is via a inflammatory response,
it releases zonulin,
causes a disruption in the tight junctions,
releases various interleukins which cause inflammation,
and then we get a spilling of this stuff,
transglutaminase into the intestinal lumen,
into the interior of the cell.
When that happens, we can
create this thing called a [inaudible 00:45:16],
which is a chimeric form of
the protein related to transglutaminase and gluten.
These things will stick together and then our immune system will attack that complex. It looks like a foreign invader.

Now the reason why this is bad is that we have transglutaminase in virtually all the cells of our body. It's involved in what's called post-translational modification. It modifies proteins in our body. If you create an antibody to transglutaminase, you could potentially have problems in any cell, any organ system in your body. This is a lot of the reason why problems with celiac or gluten related issues and other grain related issues, it's like finding the electrical problem and a car. It's just all over the place and you sound like a crazy person when somebody says, "Could gluten avoidance or grain avoidance fix neurological conditions?"

Yes. Could it fix reproductive issues? Yes. Could it fix this issue? Yes. The reason why is that transglutaminase is ubiquitous. Once you develop antibodies to transglutaminase, you could potentially have problems anywhere in your body. It's a big, big picture.
In celiac disease, this would lead into actual death and damage of the enterocytes in the intestinal lumen, and it's a pretty gnarly condition. I'm one of the lucky people that actually has this. I mentioned to the students earlier on, like the canary in the coal mine, if something's going to kill someone, it usually kills me first. A quick repost state that people have about this topic is that not everyone has celiac and that's true. But what we're finding is that there's a lot of problems with all of these prolenes and the interesting thing is that all grains contain prolamins. When we were in a meeting right before this with the evolutionary studies students, someone made the observation that they have a number of problems and that they had gone on a gluten-free diet, but didn't really see benefit. But a gluten-free diet inevitably means that you start eating large amounts of other grains and legume products. Those things contain large amounts of prolamins. Zein, and corn, hordein,
and barley, they're very, very similar to gluten.

Prolamins, like I mentioned earlier, are unusually rich in prolene. I've mentioned all of the rest of this stuff. On the intestinal lumen side, we don't have the enzymes to break this stuff down. That's why the proline-rich proteins can remain intact.

Interesting thing, [inaudible 00:47:52] showed me this maybe two weeks ago, an interesting study of halofuginone, which is a hydrangea extract that's used in traditional Chinese medicine for a host of inflammatory conditions and also really interesting some chronic parasitic infections.

What it does is it mimics prolene starvation, it blocks an inflammatory pathway that is linked to autoimmune reactivity. If you either starve and organism of prolene, you decrease autoimmune reactivity or if you provide a pharmacological agent like the halofuginone, then you also create a situation which is similar to prolene starvation. It's another interesting supportive mechanism on this development of autoimmune disease being related to proline-rich proteins.
Moving this thing along a little bit.

Annals from the New York Academy of Sciences, tight junctions, intestinal permeability, and autoimmunity.

There's growing evidence that increased intestinal permeability plays a pathogenic role in various autoimmune diseases, including celiac disease and type 1 diabetes. Therefore, we hypothesize that besides genetic and environmental factors, loss of intestinal barrier function is necessary to develop autoimmunity.

What folks are now thinking is that you need some permeable gut, some breakdown in gut lining integrity to initiate an autoimmune response.

In 2004, if you looked on PubMed, there were fewer than 200 citations that would fall out under intestinal permeability or leaky gut. Today there's over 10,000.

In the early 2000s, if you were a clinician or an academic and said intestinal permeability, your career was done. It was the realm of quackery.

Now it's possibly the hottest area of immunological research that we've
got going on and it may tie everything together, ranging from neurological disease, metabolic derangement, and autoimmunity. Depending on the type of protein that irritates the gut and your genetic factors, it could manifest in one person as metabolic derangement, another person in autoimmunity. Etiology of autoimmune diseases, infections, whenever we mount an immune response, there's a potential of having collateral damage and creating antibodies to products in our body. Low vitamin D levels are a huge factor in this. If folks have high vitamin D levels, they tend to have a normal immune response. Folks who have problems with the H1N1 flu and get the cytokine cascade, they tend to have very low vitamin D levels. If you have high normal vitamin D levels, you tend to have mitigated or normalized immune response. If there's one supplement I would recommend it's probably vitamin D3, a lot of it, like 5,000 IUs day. Physical trauma, if you get a crushing injury, your release a lot of protein into your system, you can get an autoimmune response from that also. Vaccinations, when we vaccinate somebody, we're trying to get an immune response
without causing the disease,
and so what they will often stick in
that product is something called an [inaudible 00:51:01].
One of the adjuvants they use is Quil-A HA,
which is a saponin,
and it causes a potent immune response.
There are some other immunological agents
beyond the proteins that can
also cause some gut problems.
Any type of dietary factor that
causes leaky gut can be a problem.
Unfortunately, booze, Tylenol,
there's a lot of things that can cause GI permeability.
I've been jabbering a lot and so I
need a bogey along here.
Tons of autoimmune diseases
that have been linked with permeable gut.
If folks want this particular slide,
I can put it up somewhere later,
but there's tons and tons and tons of autoimmune diseases
that have been linked with a permeable gut.
It's not proof, but it's very, very interesting.
Only about 33 percent of
autoimmune diseases have been
investigated for a leaky gut.
All of them that had been looked at have
presented with gut permeability,
so it's pretty interesting.
If you missed the volume
49 of Hepatology, I'll fill you up on it.
It's increased intestinal permeability and
tight junction alterations in
nonalcoholic fatty liver disease.
Our results provide the first evidence that
nonalcoholic fatty liver disease in humans
is associated with increased gut permeability,
and that this abnormality is
related to the increased prevalence of
small intestinal bacterial overgrowth in these patients.
The increase to permeability appears to be caused by
disruption of the intracellular tight junctions,
like we talked about with zonulin,
in the intestine and it may play an important role in
the pathogenesis of hepatic fat deposition.
People have usually heard
about alcoholic fatty liver disease.
There's also this other stuff,
nonalcoholic fatty liver disease,
in which the normal liver tissue starts getting
displaced by literally fat droplets,
it's a narrowly horrible condition.
Lipopolysaccharide, LPS,
activates an innate immune response in
human adipose tissue in obesity and type 2 diabetes.
Our results suggest that type 2 diabetes is associated with increased endotoxemia.

When we have an infection, there's this stuff on the outside of bacterial coats called lipopolysaccharide and it just absolutely sends our immune system into panic.

Because if you think about evolutionary pressures, we've had this dynamic tension between bacteria and everything leading up to us throughout all of evolution. Our systems are highly, highly reactive to lipopolysaccharide.

Some really interesting stuff here, the roles of insulin and hyperglycemia in sepsis. Sepsis is when you get bloodborne pathogens raging through your body and people can very easily die from this.

But the really interesting thing when you become septic, when you have bacteria in your bloodstream, it is indistinguishable from type 2 diabetes. The way it kills you is exactly the way that a mismanaged type-2 diabetic dies.

Yeah. So insulin resistance characterizes the septic patient and it seems that the balance between insulin and it's counter-regulatory hormones, cortisol, glucagon and growth hormone and
catecholamines is perturbed in
the metabolic response to sepsis.

When you become septic,
you become highly insulin-resistant.

Your liver pumps glucose out
even though you have high blood glucose levels,
because of the signaling between the liver and
the brain and the leptin sensing is gone.

It seems that one of the metabolic
problems that are in sepsis is
an inability to use
free fatty acids as metabolic substrate,
the liver's function as a regulator of glycemia is
also disrupted as a result of hepatic insulin resistance.

This results in increased hepatic glucose output
initially as a result of glycogenolysis,
but later from gluconeogenesis.

When I do my eight-hour talk,
I go in super deep detail with all that stuff.

But you have a situation
in which the metabolic engines are just broken,
the wheels fall off.

Muscles become insulin-resistant,
free fatty acids are released from the adipose tissue,
the liver perceives low blood sugar even though
the low blood sugar is not low,
it just thinks it's low.
The liver and brain think they are starving but they're not. Cortisol is released, which further releases glucose. Liver releases lipids and glucose, inflammatory signaling goes through the roof. The way that you save the septic patient is actually administering a pretty big bolus of insulin, which pushes all of the glucose and the fatty acid substrates into the cells and it decreases inflammatory signaling. Otherwise, that is the primary intervention in sepsis is actually an insulin bolus, and it'll save their life. But this is indistinguishable from what happens. We won't go too deep on this, [LAUGHTER] but this is [inaudible 00:55:44] gland here. But there's a couple of points I want to make here. The end product, VLDL, is what I want you all to look at. DLDDL can also become a liver droplet in the liver. This is a hepatocyte, a liver cell. Obviously it's a hepatocyte. [LAUGHTER] If you pump a bunch of glucose into the cell, you will fill the glycogen, back fill this whole process, eventually palmitoyl-CoA is produced,
triglycerides are released then as VLDLs, very-low-density lipoproteins. If we consume fructose, it upregulates glucokinase and then that whole process actually makes the liver even more spongy or sucks up more glucose. This is why fructose is such a problem. Under metabolic derangement, either from fructose or from sepsis, this whole process is in full tilt. If we go really hard, if we're pumping tons of substrate through this system, then we create a liver droplet. We overfill the capacity for the liver to release fats, so then fats start building up in the liver, and this is the development nonalcoholic fatty liver disease. You guys got all of that in 35 seconds. Normally that's an hour, so my apologies. Some takeaways for this. People are like, oh my God, why did I come to this thing? [LAUGHTER] If you annoy the immune system, everything gets inflamed. Inflammation is the beginning of all of these processes. If you get inflamed, you have a likelihood of either
becoming autoimmune or metabolically broken or both.
Depending on the protein involved in the process
causing it and your genetic factors,
environmental factors, you may head towards
autoimmunity or you may
head towards metabolic derangement.
If you provide a bad substrate to
the liver like fructose or linoleic acid,
short-chain Omega-3s,
it increases the likelihood of all these things going on.
I have inadequate time to go through
all the metabolic machinery
of linoleic acid and fructose.
But that's what I do in my bigger talks.
Sleep, exercise, and vitamin D can either
positively or negatively affect all these,
and it all grosses
in output of the evolutionary template.
Two couple more slides and we're almost done.
Gluten exorphin B5 stimulates prolactin secretion through
the opioid receptors located
outside the blood-brain barrier.
Gluten exorphin B5 is
a food-derived opiate peptide
identified in digests of wheat gluten.
We've recently shown that GE-B5 stimulates prolactin.
Prolactin is critical in pregnancy,
it's also really important in mineral metabolism.
Prolactin secretion through opioid receptors outside the blood-brain barrier.
Since opioid peptides do not exert their effect on prolactin secretion directly
via reduced dopaminergic tone,
our data suggests that GE-B5 can modify brain neurotransmitter release without crossing the blood-brain barrier.
Well, all they says is that wheat extracts can modify hormonal signaling. It's got pharmacological activity. That's really important when we think about the neural regulation of appetite.
A lot of people will say, I feel like I'm coming off of crack trying to get off of these foods and it's like, yes, you are. They're largely an addictive substance. They have a pharmacological activity.
Really quickly, look at all the names. Look at all the names on this one. Look at all the Italians studying gluten.
[LAUGHTER] When somebody says that the Italians eat a lot of pasta, tell them to put that in the pipe and smoke it. Italy is in a crisis of autoimmune disease, an absolute crisis.
They are crushed by celiac disease and a host of other metabolic issues.

This is an amazing paper, interactions between gut microbiota and host metabolism predisposing to obesity and diabetes.

This one paper does the full meal deal of gut permeability to auto-immune disease, to metabolic arrangement.

It is a one-stop shop.

This thing is amazing. If you can track this one paper down, it's a phenomenal paper to read.

It's an outstanding review. Where are we at? We've got the [inaudible 01:00:06] that showed an interesting lack of disease from an anthropological observation story that was an interesting observation.

We saw differences in the serum leptin levels between [inaudible 01:00:18] and the Swedish. That starts leading into some clinical observations. We had animal and human testing that drives the boat towards some proposed mechanistic testing.

Might bioactive molecules play a role in inflammation? I would say that from our mechanistic theory, we're saying, yeah, there might be some issues there,
and I would hang most of it at permeable gut, might be a key player in autoimmunity and metabolic syndrome.

Really quickly.
Where am I at? Holy cow.

A 35-year old woman presented with elevated liver enzymes. Alkaline phosphatase is a liver enzyme that indicates tissue turnover.

If you have cancer or if you're doing like a leading Las Vegas-type drinking binge, you'll see elevated alkaline phosphatase, but this woman wasn't drinking poorly. She was actually eating pretty well, but still had a lot of problems.

In 2005, her alk-phos was 156, 429, and 5407, which is really high.

We started working with her in 6408, and it was 518, and she was a candidate for a liver transplant.

I suggested a grain legume, dairy-free paleo diet. She did it.

One month later, her alkaline phosphatase was 42.

If you do a little digging in the research, alkaline phosphatase is a clear indicator of non-alcoholic fatty liver disease.

It is tightly tied into
gluten and proline problems, causing systemic inflammation.

Everything that comes out of the gut goes directly to the liver, that's why the liver gets so hammered by gut permeability issues.

One other person, 60-year-old woman presented with porphyria cutanea tarda, this is a "genetic disease."

This is a condition in which if you go out in the sun, you get a burn that. It doesn't just burn the skin, it burns all the way down to the periosteum of the bone.

She had had this for 20 years.

I did a little Googling.

I googled two things, porphyria, metabolic derangement, didn't find anything, porphyria, gluten, found a ton of stuff.

I told her to go off a bridge.

She said, "I like bread".

I said, "I like bread too, but you should go off of bread."

[LAUGHTER] She did it.

She was asymptomatic after one month on a paleo diet, went to the Middle East, did all this stuff.

If you find linkage between a disease and auto-immunity, a disease and transglutaminase,
you can bet your bottom dollar
that an intervention like this is going to work.

Funny thing is even though she
can run around and play with her grand kids,
half the time she's in a recidivist still,
because she's addicted to bread.
She'll go back and she has to wear
big-brimmed hat and long-sleeved
shirts to be out in the sun.
It's crazy. One final person,
a 28-year-old fighter pilot had narcolepsy.
Really bad scenario.

[LAUGHTER] Carrier landing and you fall asleep.
Contacted me, what did I do?

Narcolepsy, metabolic arrangement,
narcolepsy, auto-immunity,
narcolepsy as auto-immune linkage one month later,
asymptomatic with a paleo diet.

The commonality is all these conditions
share antibodies to transglutaminase.
It's easily discoverable in the literature.

Most modern degenerative diseases are an obvious lack
between genotype and epigenetic inputs,
but without a Darwinian orientation,
it's like doing physics without quantum mechanics.
Almost done here.

Cows eat grass.
Ricardo Salvador was an in-line to take over the Leopold Center for Sustainable Agriculture at Iowa State University.

As anybody in the academic scene knows, when you're going through a tenure track professorship or taking over a directorship, it's like running the gauntlet.

Professor Salvador was at the very end of this process and was getting a pre-acceptance speech. In that speech, he made the observation, cows evolved to eat grass. The appropriate food to feed a cow is grass.

The following day, he was fired.

[LAUGHTER] This made huge news impact. Wendy Wintersteen, who's the dean of the school of agriculture, was asked while being interviewed about this, "Do you believe that cows evolved to eat grass?"

She said, "I have no opinion on that topic."

[LAUGHTER] This is amazing that this evolutionary studies program exists, but evolution is such a hot button topic even in academic settings that people get fired over the topic.

This is the guiding principle of biology. How the hell do we do business without
the basic epistemological structure that underlies all of biology?

We don't, we fail, which is a lot of what we're doing on food production, and health, and a variety of other issues.

One final point, if you look at Lasik surgery, since I started this off talking about economics and medical cost, Lasik surgery in about 1999, 2000 was over $2,200 per eye. Now, in non-inflation adjusted dollars, it's about $600-$800 an eye. In inflation adjusted dollars, that would be equivalent in these dollars to about $200-$400 per per eye. Lasik has improved. Lasik has gotten cheaper, and it's because it is open to the market and the technology that underpins Lasik optics and surgery are accurate. They do the right thing. If we were to actually apply evolutionary medicine to health maintenance and wellness, our medical cost would be virtually zero. We have a healthcare crisis because we're driving the boat off the cliff, driving off the waterfall.
That's it. We're doing the wrong thing.

It is not surprising that it costs us a ton of money,
because we're doing fundamentally the wrong thing.

There's lots of market-based examples where if
you allow the market element
and the technological element to
innovate the way it should and you don't stifle
it with not being able to say that cows eat grass,
if you don't stifle the evolutionary theory,
then we wouldn't pay hardly anything for
medical cost because it wouldn't be much to pay for.

We would generally be healthy.

We would occasionally get an infection.

Every once in a while, somebody would
get run over by a bus
and our emergency medical system would deal with that,
but cancer, diabetes, Parkinson's,
Alzheimer's, reproductive issues would largely disappear.

This isn't a matter of opinion.

All these things are based
around systemic inflammatory conditions,
and this is what we've been talking about through
this whole talk. That's what I got.

Thank you-all. Thank you to
Professor Staple and Glass so much.

Evolutionary studies program,

[APPLAUSE]
and Dr. [inaudible 01:06:51], [APPLAUSE] thank you so much.

Also happy birthday,

Charles Darwin's 203rd birthday like two weeks ago.

Thank you-all for coming.

[APPLAUSE]

Thanks so much. I'd like to remind everyone we're having a reception in the terrace just after we finish here.

I think we have time for just a couple of questions.

Please wait for the microphone because this is being recorded.

[NOISE]

I just wanted to ask you if you think that the liver deals differently with high fructose corn syrup than it does with natural sugars.

The question is, does the liver deal with high fructose corn syrup differently than natural sugars?

Absolutely. When you put in fiber, when you have other co-factors that we get from fruit and vegetables, then it reacts very differently.

We're not overloading the system to the degree that we get out of high fructose corn syrup.

Here's an aside.

Whenever you see a low glycemic load sweetener, like agave nectar, it means that it's high in fructose.

Agave should only be used to make tequila,
Hi. I'm a little disappointed with the lack of evidence between the lectin-leptin connection. Tell me again why I'm not eating beans. Because of other similar proline-rich proteins. Instead of it being lectin specifically, we have other proline-rich proteins similar to what we have in grains that are the problem. Basically, we were looking at A, but it ended up being B. If you just swap out prolines or globulins for lectins, then we've got the same story. So those are effecting? Yes. Absolutely. Without a doubt.

Thank you.

Yeah. [NOISE]

We can see evidence in guys like Art De Vany. In the slides you showed today that the more lean muscle mass you carry with you into advanced age, the healthier you're going to be. With that in mind, there's so much conflicting evidence about what exercise is going to be the most successful for you. In your opinion as a former power lifter, someone who has been involved with CrossFit,
what would you recommend as
the most successful exercise modality state
would you really on the everyday basis through your life?
I did a podcast on this.
If you like lifting some weights through
some hypertrophy appropriate type
weightlifting a couple of times a week,
I think mobility is huge.
Like doing yoga or capoeira or something like that.
So you doing a lot of nonlinear stuff.
But ultimately, do something
you love and do it all the time.
Then if you need a little bit of PM,
some preventative maintenance lift weights twice a week,
you do a full body gig.
I'll say if folks aren't familiar with Art De Vany,
his technology the hierarchical sets,
and the elastic sets and stuff like that,
they are the most productive way
I think you could do strength training on
a return on investment like
minimum investment, maximum return.
If you want to be an elite level
Olympic lifter or want to have
super high power lifting numbers, it's not going to work.
But you can be pretty strong,
carry a fair amount of muscle mass and for
a tiny input of time, yeah.
I'm curious about how the prolamins act on
a molecular level with
the phospholipids in the intestines
that make the wall permeable.
Well, they're indigestible.
So when they're interacting with the wall,
there's the potential with it interacting like
the CXR3 receptor site.
So what it does is it leaves them whole and
available to cause a variety of problems.
Mat Lalonde, his AHS talk
is going to be specifically on prolamins,
globulins and all these
immunologically reactive proteins.
So be ready, man.
Just wear two pairs of pants or something,
it's going to be big, [LAUGHTER] yeah.
Hi. You mentioned wheat, rye, corn.
Are there any other substances that
fit that description that
caused that problem [OVERLAPPING]?
All grains contain prolamins.
So corn is a grain?
Yeah.
Anything else?
Rice, oats, barley, millet, sorghum.
So we shouldn't eat rice either?
I'm not saying what you should or shouldn't do.
What I'm observing is when people don't eat this stuff, they get a lot healthier,
so [LAUGHTER] yeah [APPLAUSE].
You spoke about high ginger extract.
Yeah.
How it can help with chronic parasitic conditions.
Do you think that can help Lyme disease?
The parasitic conditions that they mentioned were gut oriented,
so it's actually organisms living in the gut.
If you Google Robb Wolf Lyme disease, we've had several people who have had Lyme disease and saw remarkable improvements eating this way.
Improvements in the immune function and what not.
Interesting aside, I did spiral kit research and so that was some of the stuff in my wheelhouses actually Lyme disease.
But we've seen some really interesting benefits with it, yeah.
Go ahead Robb.
We just have time for one more question.
I'm going to pass the microphone down.
Hi. I was just wondering,
I know a celiac disease if you cheat and you eat gluten
even once even through cross-contamination, it kills the villi again and have to start all over again. Is that how the paleo diet is? Can you cheat and that you still get the benefits [LAUGHTER] from it I guess? I like to equate this almost on like a relationship type level. [LAUGHTER] [APPLAUSE] For some people, their significant other would consider looking at someone else as cheating. Or other people, a little bit of flirting is cheating, for other people whatever is cheating. [LAUGHTER] So I don't fully know. I'll tell you this, here's kind of an indirect way to answer that. I do a lot of work with special operations community like Naval Special Warfare. These guys are super detailed, dot the i's cross the t's. They want to have super high performance and they will go full craziness on this. No gluten, no grains, no legumes or dairy eating. They have amazing performance. They get deployed, they eat in MRE and then they've got the trots for like five days.
Because the gut actually heals and then you get this potent immune response there.

What I tell those folks is that when they get into a pre-deployment scenario, they should actually put a piece of bread, a beer, some gluten containing item in the mix so that they have low-level inflammation consistently instead of that, I'm going to not make it to the latrine kind of gig if I eat the other stuff.

So to a degree, yes.

But what that's indicative of is that you are healthy and then you're eating something that's causing a reaction.

If you think about it this way, if you react to poison oak or poison ivy, if you get re-exposed it's going to give you a blister, you're going to have a problem.

This is very similar to what's happening in your gastrointestinal tract.

So what I recommend to people is that they try this and then they play around a little bit.

Like we had a question about rice, I eat rice maybe once or twice a week.

Sometimes I'll go a month without having it and then other times we'll go out and get sushi and I bring a gluten-free Tamari and I'll have rice a couple of times.
I don't really notice much problem
if I have it punctuated.

If I have it three or four days in a row,
I start getting acid reflux and GERD.

Same deal with corn.

What you can do is figure out what level of
philandering you can get away with without getting bang.

[LAUGHTER] So in that way then it's hopefully
not a religious doctrine,
it's something to help you live your life
better and then you can find the wiggle room on your own.

But what I recommend is people really give it a shot,
get healthy, see where they're
at with that and then reintroduce stuff.

Even within that if you had dark chocolate and ice cream,
I think you're going to be much less reactive
versus having a bunch of grains.
If you're going to kick your heels up like some tequila,
dark chocolate and ice cream,
that seems like a good night to me,

[LAUGHTER] I don't know.

I just have a couple of quick closing comments.

First off, as director of
evolutionary studies I'm often asked,
"How can this evolution stuff help us
understand ourselves and how can it make life better?"

It's obvious. I think what Robb Wolf is
telling us here is that
the answer to that is very obvious.

So I really appreciate you
bringing that to the forefront.

A couple of other things,
we are going to have

a Paleolithic feast which will be over at the terrace.
So we look forward to seeing you there.

There will be books for sale and they will
be potentially signed, I believe.

We are going to have Darwinian charades,
so something for you to think about.

[OVERLAPPING] [LAUGHTER] I guess

a final comment and then a final question.

One thing students will often
conflate with the Paleolithic diet is the raw food diet,
this is not the raw food diet.

Human beings and our ancestors have been cooking
for thousands and thousands of generations.
So I think that that's a point that should be clear.

A student prior asked Robb, "What do I eat?"
Which I thought was a pretty straightforward question
and he gave a great answer.

I guess if you wouldn't mind to
wrap up to sort of
answer that question for this audience.

Sure, and what I do with this folks that say,
"I don't know how I would do this."

So as a raise of hands,
how many of you all have ever eaten
scrambled eggs and fruit for breakfast, ever?
Okay. How many of you have ever had
grilled chicken or grilled fish on a salad for lunch?
How many of you have had some pork tenderloin or
pork spare ribs with
some grilled veggies and a glass of wine for dinner?
You've probably had thousands of paleo meals,
you just haven't strung them all together.
That template, let's say you're
a hard charging athlete, you play lacrosse,
I would see a bunch more yam,
sweet potatoes, bananas, apples,
oranges in that mix because you need
more carbohydrate for glycogen recovery.
Most people are like, "I don't need that."
I do some Brazilian Jiu-Jitsu and stuff
but my activity level is not that high.
So I do a little bit of carbs,
but not a ton of carbs.
You use carbs based on
how you run your activity level and stuff like that.
But that literally is
a day of paleo eating and you have done
thousands potentially of days of
paleo meals you just haven't strung them all together, so it's really not that hard to do.

Thank you-all. Thank you very much.

[APPLAUSE]