[MUSIC PLAYING]

COLIN E. CHAMP: Do I get five extra minutes at the end of this?

AUDIENCE: [INAUDIBLE] [LAUGHS]

COLIN E. CHAMP: Well, regardless of 35 minutes, which is probably five more minutes than I was taught diet and nutrition during my entire med school experience. So we're off to a good start. Yeah, she said I'm a radiation oncologist. I also research diet, exercise, nutrition, cancer prevention. I see cancer prevention consults. And I see survivorship patients, a lot of breast cancer patients and try to help them segue to a healthier lifestyle.

Everything I talk about today is my synthesis of the data on diet. And I gave it a critical eye over the years. It's very controversial. So I'm sure some emotions in the room will pop up and flare up when I make some comments. My talk is an anti-cancer diet. Can it be healthy and enjoyable?

Well, I'm going to talk about the negatives-- the current issues with a lot of food and lifestyle studies. So the studies that my mom calls me about that were on KDKA in a panic about what she should eat. The issues with currently recommended foods.

A lot of these current recommendations aren't backed by substantial data. Part of it's due to some of these hopes and dreams about what foods we should eat. A lot of it's from issues with the studies. And when we published a study looking at what cancer patients are told, it's all over the map-- low fat, high fat, low calorie, high calorie.

We're going to talk about the issues with ignoring cellular mechanisms. That's going to be, by far, the most boring part of this talk. But I'll try to just weave those in, because we have to have some kind of tangible mechanism that describes why we're telling people to eat what kind of food. The positives-- I want everyone to leave here with some tangible mechanisms with which food and health interact-- methods to improve compliance and enjoyment.

And I want to review supporting studies to provide tangible takeaways. So the point of this is to-- much like the conversation we just had on exercise. Not just tell people they should go exercise. And they say, great, I'm going to go do, like you said, five days a week for 30 minutes. And they go home and don't do anything.

I think that happens a lot with our dietary recommendations. We telling these idealized recommendations. And then they get in the car and say, that guy's nuts and go home and continue to do what they've done for 50 years. My conflicts-- again, financial, I write books. So I get a small amount of compensation for that. It's on the same topic. But it in no way impacts what I'm talking about. And then my dietary conflict.

So realistically, any talk you go to on diet, exercise, lifestyle-- whoever's giving that talk should tell you about their conflicts, because those are going to interact, right. So if certain foods were off limits for me based on my religious views or moral-- ethical views, I should be telling you about that, because it's going to interfere with what I'm telling you.

And I don't have any, except, that I eat a real food diet. I eat whole foods. My family's Southern Italian. They came over to the beautiful McKees Rocks. You guys have been there. And so they've always favored these long drawn-out meals-- cooking food. It's always been a big part of my life. And so certainly everything I tell you is biased by that.

But in terms of what type of foods I eat, I've done every diet in the world-- low fat, high fat, low calorie, high calorie, ketogenic, paleo, vegetarian-- everything. So I have no beef with any of them from the start. So everything I tell you is what the data shows me that's what's best for me to tell patients and what's going to give them the ability to be successful in their dietary changes.

I never tell people to count calories. And I believe the food pyramid was the largest public health mistake in US history. And I think everyone in this room-- we're still cleaning up the mess from that told to so many people, because it just derailed so many people's healthy lifestyles. So that is, I guess, one of my big biases.

Every time I give a talk on nutrition, exercise, any kind of lifestyle activities, I was talk about this man-- this is Sir Richard Doll. And he's famous for being the first-- well, it's controversial. Some say the Germans were but being the first person to link smoking with lung cancer death. And he published on the right here-- you see it's 1954.

He studied, I think, it was 20,000 to 30,000 people at hospitals across England. And he found that people that were smoking had a 40 times increased risk of dying from lung cancer. So 40 times-- that's a massive number. Taking a step back in actuality, if you smoke, you really only have an overall risk of 16%. OK, I'm not saying anyone should go and smoke. And that's still a very high risk for a very lethal disease.

But there's two points I'm trying to make here. One, that 40 times increased risk is massive. It's the largest increased risk we've seen from occupational exposure. We've never come anywhere near matching that in any of the studies that come on KDKA. So when they tell you that coffee's going to cause some other kind of cancer, it's usually like a 0.2%. OK, not a 40%.

And we have to remember, overall, it's still a 16%. So when we talk to patients and say, your risk is 40 times higher, we still need to give them that absolute number, because it may not actually be that high. It was 3000% of an increase in dying from lung cancer for heavy smokers. So again, keep that number in mind-- 3,000 times higher.

Richard Doll was made Sir Richard Doll. So he was knighted. That's how important this data was. Shortly after 1975, with one of his colleagues, Armstrong, they published a similar study when it went on the world-- compiled data trying to link-- so on the heels of tobacco, they wanted to link other occupational exposures to cancer.

So they looked at a ton of different food types-- coffee, dairy-- all these different things, and all these different countries all over the world. And there was a bunch of things they found. But three the ones that stood out most was that fish consumption was associated with stomach cancer. Coffee was associated with kidney cancer. And dietary fat was associated with breast cancer.

Now, the first one we found over the years, had nothing to do with fish. It was the way they were processing the fish. Some of the preservatives they were putting in them. In some of the Asian countries, they're increasing the risk of gastric cancer. And that has since been disproven that it's from fish and themselves. But at the time, people believed it.

Kidney cancer and coffee-- that was an easy one. It was a social issue. In some of these third-world countries-- some of these poor countries, the people that had access to coffee were wealthy people that were basically following a Western lifestyle. And we know that a Western lifestyle is much unhealthy in many different ways. And it had nothing to do with the coffee. And thank, god, coffee actually nowadays looks like it lowered your risk of cancer.

So to show how ridiculous some of these studies are, there's a study out, I think, last month that showed it was a linear no threshold. So based on that, the more coffee you drink, the better. It never stops. So if you drink 100 cups a day, you'll never get cancer. So Doll followed up his tobacco study with another food study. This man, Walter Willettt, followed up Doll's fat and breast cancer study with a series of studies trying to link the two.

So what runs the nutrition center? I forget the exact name over at Harvard. He's a big-name player. I forget how many publications he has-- it's like in the thousands. So he wanted to take Doll's hypothesis that breast cancer and dietary fat were linked. But he wanted to look at more moderate and Western data.

So he had the Nurses' Health Study. He had 90,000 women, ages 34 to 59-- these were all nurses. They had no history with cancer. And he followed them for five years. And then in 1987 published a study that was trying to prove that dietary fat increased your risk of breast cancer. It was published in The New England Journal of Medicine. And it was negative. Or he said it was negative.

If you actually look back through the data, fat intake actually trended with a lower risk of breast cancer. It was not statistically significant. So he was honest in saying that there was no relationship. But as you went from unsaturated to saturated to absolute amount of fat, breast cancer rates lowered. So it was exactly the opposite of what Willett was trying to find.

In 1996, he pulled an analysis of six additional studies looking at the same topic for a total of 337,000 women. And he found the same thing that was unrelated to breast cancer risk. In these papers he said, perhaps, it's just not related. Perhaps these women were eating too much fat. So we need a cohort of women eating very little fat, and women eating high fat so that we can compare. Or perhaps the follow-up wasn't long enough.

He republished again in 1992. At this point, he had eight years of follow-up. This was also around the time that fiber was considered the end all be all when it had to do with health. So he looked at fiber intake as well. Again, he found that fat and fiber was unrelated to breast cancer risk. Again, he said maybe we just need a little less fat and a little more follow-up.

In 1999, he published a study with 14-year follow-up. In this time around, it showed the same thing-- increase fat trended towards a lower risk of breast cancer. Willett took it a step further and said, well, when you reduce one food, you increase another. So let's look what happens when women increase fat by 5% by swapping out more fat for less carbohydrates. And he found that their breast cancer risk went down by 4%. And that was statistically significant.

The weird part about this all in all of his conclusions, he kept saying there was no relationship between fat and breast cancer. And maybe that's true. But when you actually read these papers, you see that there's clearly a relationship. Or at least there's statistically a relationship, especially in the last study. When you swap out carbs for fat, it increases your risk of breast cancer.

He never said in his conclusion. But it was buried in the study. That illustrates part of the bias at the time. I'm not even blaming Willett. He was following up on Doll. This guy was knighted. So it's understandable why he would say, well, if his data showed this, then it should cause it.

Later on in 2012, Willett says there was never any strong evidence for this idea. But it was repeated so often that it became dogma in the 1980s and 1990s. So in my eyes, Willett's vindicated for his bias in these studies, because he's since given multiple talks stating this-- talking about their issues. And it really overshadows the whole issue with a lot of these dietary studies.

They believed in their heart that fat in the diet was causing breast cancer. And they just wanted to prove it so badly in their studies that they were overlooking their own data disproving it. And Willett's been pretty vocal about that. We see it happen in dietary studies again and again. There's no better example about what happens in these population studies when we just look at associations in the fiasco with hormone replacement therapy.

So in 1985, hormone replacement therapy was being prescribed for post-menopausal women. There's a lot of issues that accompany the drop in hormones during menopause-- hot flashes, fatigue, decreased activity levels, weight gain, weakening in the bones, et cetera. Some women are being prescribed hormone replacement therapy. It was making them feel better. But doctors didn't know how it was affecting them.

So the same group at Harvard, Willett and some of his other colleagues decided to look at this. They went to that same group of women. They had 90,000 nurses. They actually had even more to look at some other things. They went through all these women. They adjusted for all these different factors. So tobacco usage-- how much they smoked, whether they had diabetes, high blood pressure, cholesterol.

If their parents had a myocardial infarction-- if they were on oral contraceptives, and if they were obese. And they got two groups of women that looked identical in all those factors. And the women that were on hormone replacement therapy had a 50% decreased risk of coronary artery disease if they've ever used hormone replacement therapy and made a 70% decreased risk if they were actively using hormone replacement therapy.

Their conclusion was we pushed our statistics to the limit. It has to be that these hormone replacement therapies that these women are being prescribed is reducing the risk of heart disease. At that point, it was published. Hormone replacement therapy was widely prescribed-- 1985 for about the next 20 years. Only in 2002 did they actually published a randomized study.

And this a key point here. The difference between an observation study is in an observation study, I just watch a group of people do a bunch of things and then say, hey, when this guy did this or when this girl did this, it increased their risk of certain things. It's hypothesis stimulating, but it proves nothing. A randomized study, on the other hand, you choose who gets what. So you're grouping the people as best you can to make sure that there are the two very similar groups of, in this case, women. The only difference is hormone replacement therapy.

What they found was that women that were getting the hormone replacement therapy actually had an increase in their heart disease by a lot. They had a 25% increase in breast cancer risk, increase in stroke, increase in blood clots in the lung, pulmonary embolism, and an increase in total cardiovascular disease. The only benefit of this all-- and this makes sense if you give estrogen-- was that they had a decreased risk of a hip fracture.

So for two decades we prescribed women a medication that we thought helped in all these ways. And it actually hurt in multiple ways, because it was never tested. When they went back and looked-- why did this happen? Why were these women so much healthier? Why did they have so much lower risk of heart disease? And this is what I call the Lululemon effect. But it's called the healthy user bias.

So for women that were going to their physicians and asking for hormone replacement therapy were the women that were most bothered by being fatigued-- by having hot flashes. These were women that were going to Lululemon lemon on their way to do yoga and then do Pilates. Then they would come in for my breast cancer treatment and then leave and go to Pilates.

I literally have women that come to my appointments from the gym and then go back to the gym. These are the women that were going to their doctors and saying, please prescribe me hormone replacement therapy. You could be the best statistician in the world. And there's no way you can account for that. It's called healthy user bias. And this perseverates throughout the research and the dietary research world to this day.

If you tell a group of people to do one thing, and then follow them for 20 years, the people that do that thing are probably going to do other things that we tell them to do. So they're going to exercise. They're going to wear a seat belt. They're not going to have a gun in their house. Or if they do, they're going to keep it locked up. They're going to do all these positive things. And so to try to pull one of out and say this is why they're so healthy is very difficult, if not impossible to do. It's why we need randomized trials.

So I personally, when I go through lifestyle recommendations to give to patients-- to give to family members-- to follow myself, I think of a couple of things. First off, I think, Mark Twain, if he actually said this, it is questionable. But there's three kinds of lies-- lies, damn lies, and statistics. So keep in mind we can have the best dad in the world, and the best Harvard-trained statisticians. And that there's always going to be issues when you try to pick through and pick things out.

So do these recommendations have backing by population studies? So Willett studies, for instance, I'm not saying they're worthless. They're hypothesis stimulating. So we need to have some data showing that this is what happens. Number 2 is really important-- is there are mechanistic support? In the cancer world, when we look at dietary studies, so many of them lack any mechanistic support for what they're proposing that it's hard to take them serious.

Are there animal studies? This is the one step below us. Are there studies in mice? Monkeys are less often. But it support these mechanisms that change or these diets. And oftentimes when it comes to dietary studies, that's the best we have. And then finally, are there randomized human studies? These are by far the creme de la creme.

When we look back at Doll and Armstrong's warning or Doll and Armstrong's data, they gave a strong warning at the end. And it's from 1975. It's on the internet. It's a great article. They caution that their findings may reflect some other variable correlated with economic development.

Something about people that were eating more fat made them do a bunch of other bad things. This is the reverse healthy user bias. These people were doing a bunch of bad things. They were smoking. They were drinking a lot of coffee, which at the time, they thought caused kidney cancer. They weren't exercising. They were. Doing a lot of unhealthy things.

And their second point was the quality of cancer incidence that is significantly affected by economic factors, particularly as controlling for any of the food consumption variables can reduce this correlation. People that were eating more fat were generally eating more food, in general and were generally more obese. These were the Westerners in the East. And that's all their data was showing.

That's why it was negative for Willett. But Doll and Armstrong were very, very straightforward in those issues back in 1975. Everyone just ignored it, because they thought this is finally the chance to start linking our lifestyle with different kind of cancers.

So back to the last comment about weight interfering with this. So being overweight is one thing we know that consistently increases risk of breast cancer. It's been shown in population studies. It's been shown randomized studies. It has mechanistic support. Adipose tissue secretes inflammatory factors. These are the fertilizer for cancer cell growth. It secretes hormones if you have an estrogen-positive hormonal cancer.

If you have an IGF-1-positive hormonal cancer, your fat tissue is going to secrete these hormones that can cause it to grow. This is a study from JAMA. This is a population study. This is women when they become menopausal on the left there. These are women that lost 10 pounds after menopause-- 5 to 10 pounds in the blue. No weight is in the middle. Orange-- these women gain 2 to 5 pounds. Gray-- 5 to 10. And then lighter gray above 10 pounds.

So if you lose 10 pounds after menopause, and you have a history of breast cancer, your risk of recurrence goes down by 30%. If you gain 10 pounds after menopause, your risk of recurrence goes up by 30% in a stepwise fashion. And again, this is a population study. But it's been shown repeatedly it is bad to be overweight if you have a history of breast cancer.

So that's the first thing, When we're going to tell patients what should they eat? What should they do? Want to do things to ensure their weight will be low. And that's regardless from any dietary things I'm going to say. We need a diet that works for an individual to keep their weight low. So there are tons and tons and tons of low carb versus low fat studies out there. There are tons of randomized studies. These are all in noncancer patients. But they do give us some insight.

In this study, you all may have heard it. This came out two weeks ago. And this was in JAMA. They called it a low-carb verse low-fat diet. Realistically it was not either. It was kind of low carbish. What was the floor at the Hollywood? Was there a Hollywood diet? Can't think of it. What is it?

AUDIENCE: South Beach.

COLIN E. CHAMP: South Beach. Yeah, it was like a South Beach diet, which was like, what my mom was doing in the 90s. So it's awesome that they're randomizing people to that in 2018. And then there was a low-fat diet, which I don't know if it was that low fat. These men and women were still eating about 30% fat.

Well, it showed that all people in both arms lost 12 to 13 pounds in one year. And it was a success in that everyone just lost weight, whether they were low fat-- whether they were low carb. Realistically, it had nothing to do with that. This is the first study of its kind that really didn't care about low-carb or fat. They didn't count calories at all. People were purposely told not to count calories.

They focused on real food. And they made people change the way they viewed food. So there was no calorie counting. They were told to eat nutrient dense foods. This is something that we have not told people to do. So you get the food pyramid. The base is 11 servings of bread, pasta, rice-- these very nutrient sparse foods, which lead to overeating, and a lot of other issues.

They told everyone to limit processed foods-- sugar, bread, and pasta. So in terms of simple carbs and really overall carbs, everyone had dropped their carbs in this study. And everyone dropped the same carbs that make you overeat, whether they were low fat or low carb. People were told to cook. People were told to eat with their family. People were told they shouldn't snack.

OK, this is a very different view. This is like, I'm proud of this study, because it's like my Southern Italian view that food is not some mathematical problem. Food is actually a cultural experience. And people are told to eat whole foods. People were told to eat quality foods. People were not told to cut meat or red meat. They were told to eat grass-fed red meat, because grass-fed red meat has a lot more anti-inflammatory components in it.

This is a first study of its kind where they were less dogmatic about calories and numbers and all these different things and more dogmatic about just eating real quality food. And so in the diet wars, which are, again, this is a minefield, one thing we need to keep in mind is that when we're telling patients what diet they should engage in, they are generally somewhat failed a year.

But we compared low-carb calorie unrestricted and low-fat calorie restricted. That's been the standard, right? Low fat, count your calories, exercise more. When we compare that to low-carb, eat as much as you want, a low-fat diet has never been victorious. A low-carb diet has been victorious about 26 times. And they've tied 27 times. OK?

So I wouldn't say it's a current recommendation. I don't think it's a recommendation. But our old recommendation has never been shown in a randomized study to be successful. So we really need to bury that recommendation because it doesn't work. And I'm sure a lot of people in this room have seen patients struggle with this where they're counting calories. They're low fat. They're barely eating any fat at all. And it's just not working.

If we can even just here telling them to eat as much as they want, this gives us some insight. It's not the end-all-be-all. And this is likely going to have to be individualized. But it's an interesting graph, nonetheless. And the reason a lot of this happens-- there's two different theories right now in the obesity world. There's the calorie-in-calorie-out theory. And then there's the insulin theory of obesity.

And this is basically that we've eaten certain foods, like simple carbohydrates, like 11 servings of grain, pasta, and wheat per day. And it cause our pancreas to secrete insulin so much that insulin pulls sugar into ourselves. It makes ourselves fatter. Eventually we become less insulin-sensitive. The cells are still hungry. They're pulling all our food in. But our body feels like it's actually starving, because our other cells aren't getting any food.

Controversial but from a cancer point of view, it's important because of studies like this. This is a prospective study from women with early stage breast cancer. This is from Pamela Goodman and her group. And this is published in Journal of Clinical Oncology.

We know that after a cancer diagnosis, you're fasting insulin level. So your amount of this chemical that helps pool sugar out of your blood and into your cells-- it's an anabolic chemical. It tells your cells to grow. Well, it also tells cancer cells to grow. And so they put women in four different quartiles. And they looked at their overall survival at five years. So if you had a low serum insulin, your survival at five years was about 93%.

As your serum insulin rose, your survival drops to about 80%. The women in the highest cohort of circulating serum insulin-- at five years, their survival was about 68%, which is a horrendous number for early-stage breast cancer patients. These women should be the vast majority. 95% or above with early-stage breast cancer should be alive at five years.

Again, it's a prospective study. It has all the issues I've talked about before. But it's intriguing. The most interesting way I've visualize this. And, yet, I'm going to talk about a worm study. But if anyone here is familiar with Cynthia Kenyon. So she's one of the first people to show that you can make a genetic change and increase the lifespan of worms.

She has these worms. And she cut off this gene here. It's called DAF-2. And what DAF-2 does is-- this is when it gets a little boring, so I apologize. But so DAF-2 it's like a bouncer. It sits outside of our nucleus. That's like the mainframe of our cells. That's where all important information is. And it stops this guy-- this DAF-16 from going in.

What she found out was if you block DAF-2, DAF-16 goes into the cell-- binds to the DNA. And it turns on a bunch of genes that do a bunch of things. So genes that when you sense damage-- genes that help fix that damage-- antioxidants in longevity genes. So that's why these worms are living so long, because this DAF-16 is bonding to their DNA and helping to promote proteins that help you live longer.

So when she took normal worms and gave them glucose-- even a small amount of glucose-- 2% glucose-- it would activate that DAF-2. So it would tell that DAF-2 that like it's nutritional party time. There's a lot of nutrients coming in. This is not stressful. And that DAF-2 would be blocked outside of the nucleus.

Next, she took worms that she mutated that the ones that were living twice as long. She gave them glucose, and they no longer lived twice as long, because of that glucose again shut off the mutation. And it let DAF-2 block this guy from going in there and transcribing some anti-inflammatory genes.

What she and others have found is that this DAF-2 parallels, in humans, the insulin and IGF receptor. And there's dozens of studies showing that people that have mutations, the centenarians in Italy, Greece-- all these Blue Zones-- they have huge rates of mutations in their IGF receptor.

So they're basically her worms for whatever reason, Sardinia, a lot of these islands, they have this mutation. So regardless of how much insulin or IGF they have floating around in their blood, it's not binding to it. And it's not telling it to do several things. And what it tells the cells to do is it tells the cells to grow. IGF is a growth hormone. Babies want that, right? It tells your bones to elongate. It tells you to grow taller.

What it also does is it activates several of these pathways that we actually try to block with cancer therapies-- so mTOR-- AKT. It promotes cell growth and survival. If you're a growing child, that's a good thing. If your cells are damaged, that's a good thing. If you're getting older, and you need to repair your bones and muscles, it's a good thing. If you have too much of it, it causes cancer.

It also blocks a lot of these stress-- and I mean stress in a good way-- activating genes that help to promote anti-inflammatory hormones in the antioxidant defense system. Again, this is a population study. But in that same group of women in the Women's Health Study, they actually tested their breast cancer specimens for IGF-1.

And those women that had IGF-1 receptors on their breast cancer cells, a lot of them do. Not all of them. Those that ate a high carbohydrate diet had a almost 600% higher risk of recurrence than those women who actually decreased their carbs. If you didn't have the IGF-1 receptor on your cells, it didn't matter based on their data.

But you do not want to increase the IGF pathway. And you don't want to increase the insulin pathway. And this becomes difficult, because a lot of foods do it. Vitamins, nutrients, et cetera, do it. But there's certain ways outside of diet to shut off those pathways. And this is a very busy slide here. But this is the IGF-1 receptor and insulin receptor. These turn on all these processes.

Something else that parallels what Kenyon was seeing in her worms is this thing right here called AMP kinase. All right, everyone's heard ATP? ATP's like the energy currency of our cells. When our ATP gets low, AMP kinase senses it. Pushes the oh, crap button and tells the cell to stop doing whatever it's doing. It turns off mTOR. It says stop replicating.

We don't have a lot of things going on right now. We don't have nutrients to provide that replication. We need to break down instead of building up. So things like, exercise-- intense exercise-- up regulates the AMP kinase. Fasting for prolonged periods of time up regulates AMP kinase. So I tell patients don't eat all day. Eat small, distinct-- or eating distinct meals.

Carbohydrate restriction does the same thing-- varies based on different people in terms of the level. Ketosis-- there's this big thing about ketogenic diets now. Celebrities are doing them, et cetera. It's like a biologic hack that turns on AMP kinase without having to fast or starve yourself. And again that AMP kinase thinks it's a fasting period. Food's going to be sparse. It turns off all those pro-cancer pathways.

The other thing it does is it ramps up our mitochondria, because as that happens, we start burning fat at that point, right? Our storage form-- we have about an hour of glycogen in our muscles, OK. That's our glucose source. Once that's gone, our body starts burning fat. It mobilizes fats. And it turns them into ketones. Those ketones go across your blood-brain barrier and tell your brain that it needs energy.

So anything that's going to ramp up at the mitochondria-- and this is where things get a little backwards. It's going to produce free radicals. OK, these are less are free radicals much like Kenyon studies-- much like cutting off glucose-- free radicals that are produced within our cells actually tell our cells that they need to increase their stress response. Free radicals can cause cancer. So it's stressing ourselves in a healthy way. Does that make sense to everyone?

So once those free radicals increase, it increases antioxidant production. And this is likely why if you give people antioxidants before they exercise, they don't get these benefits. It cuts off all these benefits. A lot of that data on antioxidants has been very negative. Antioxidants have increased rates of cancer. It's increased rates of lung cancer.

There's a melanoma model that just came out of mice. When they gave them antioxidants, metastases increased. It's likely because of it, you do not want to shut this off. We want to stress our system in a healthy way. And there's ways to do it through diet, exercise, et cetera. And this has been shown in multiple species studies to improve [INAUDIBLE] function.

So the stressful balance of food-- So we have sushi on the right here with wasabi. We have red wine from an area in France called Cahor. We have an onion. We have the guy from *Full Metal Jacket* screaming with Indian spices and Brussels sprouts. Does anyone know what's similar about all those?

So they all-- except for him-- they all have chemicals that stress our cells to increase antioxidant production. Red wine has proanthocyanidins. It actually increases antioxidant production, which is why they think that it actually decreases cardiovascular disease risk. Brussels sprouts have sulfur. Onions have sulfur. Wasabi has a sulfur derivative. And it's not oil-based, which is why when you bite into wasabi, it immediately blows up, and it burns your nose.

These are defense mechanisms for plants. These are fatal to insects. And they're irritating to animals. But to us our body senses them as a poison. Much like the sergeant, it doesn't kill us. It trains our troops-- our cellular troops-- to come back stronger. So these are all foods that push cancer patients towards green leafy vegetables-- pungent vegetables.

Spices-- so we know that spices turmeric-- I mean, everyone's saying turmeric now. It actually increases our BNT cell modulation. It decreases inflammation again by stressing ourselves. It is noninflammation inflammation. It's basically exercising ourselves. Blueberries-- raspberries-- I tell patients to eat the smaller fibrous fruit sources, because they have these chemicals that do that. They have less sugar. They have a lot of fiber in them.

So if someone's struggling with weight, I tell them let's not do bananas. Let's do this instead. And then in terms of green leafy vegetables, I tell patients to eat these as much as they want, especially cruciferous vegetables. Sulfur increases this other path. We're going to get into it. It's called NRF-2. And it's again, it's a stress response pathway. It increases anti-oxygen support.

Lastly, it actually increases carcinogen detoxification. There's a couple studies from China where they gave heavy smokers cruciferous vegetable extracts. And then when the chemicals [INAUDIBLE] in some of these cancerous chemicals-- instead of your body metabolizing it, it actually binds to it. And you secrete it.

So it's one way to get rid of all these harmful chemicals that we're all getting bombarded with on a daily basis. And then, last but not least, what all these things mean to me in a JAMA study-- these different chemicals and foods. Instead of telling people to eat certain foods or not eat certain foods, we need to focus on quality.

And this is my favorite study in the world. This is from Francesca Sophies from Florence. For 10 weeks he gave a cohort of really lucky participants a half pound of Pecorino cheese. So Pecorino is a sheep. It's a sheep's milk cheese. Sheep roam around. They eat grass. It's extremely high in conjugated linoleic acid.

So that grass ferments in the sheep's stomachs. The bacteria in there actually eat the grass. They give off byproducts. And that's what the sheep eat. So the sheep are actually on like a ketogenic diet. But that grass is fermented into conjugated linoleic acid. We have the same bacteria in colon. That's why green leafy vegetables are good, because we ferment them into the same anti-inflammatory chemicals.

He gave people 10 weeks of a half pound of this cheese per week, which like, when I was in med school, they would have thought he was crazy. And then he gave people 10 weeks of just a normal over-the-counter cheese. What he found was that people eating Pecorino, the serum inflammatory markers, IL6, IL8, and tumor necrosis factor all decreased significantly after 10 weeks.

Having people eat cheese, which is really a shun food. But the point here is it's the type of cheese. If you get a normal cheese, none of these things change. So if you have patients that want to eat cheese, instead of say, no, you can't eat cheese, they're going to go home and go eat cheese and maybe not see you back for follow-up. You can tell them, well, maybe try to eat this cheese. And they're actually selling this stuff at Costco, because I got in a fight with my mom about it.

And then this is another study on meat. Meat's been this powdered keg, red meat, et cetera. There's a couple of things we know. In any meat, it has a lot of vitamins and nutrients, et cetera. So do we really want to tell cancer patients not to eat very nutrient-dense foods? It all depends on their belief system. What I try to tell them is eat the right ones. So don't burn it. We know that burnt meat has carcinogens.

Really burnt any food has carcinogens, especially cooking meat on the grill. The fat drips onto the grill. It gives off acrolein. It's the same chemicals from cigarette smoke. I tell people when they do eat meat, try to eat it with green leafy vegetables. Increase these antioxidant support systems. And what kind of meat you eat actually matters.

This is a study where they randomized people to Wagyu beef, which is a marbleized beef where the cows are fed grains to increase their fat. Or they gave them wild kangaroo meat. And the dotted line is the traditional grass-fed beef. When you eat meat, oftentimes, inflammatory markers are going to rise, because it's a foreign body in your body.

TAG decreased in the wild meat. There was a small increase in IL6-- tumor necrosis factor in CRP. But in the hybridized beef, it shot up through the roof. And it persisted at two hours. So again, you can't say don't eat meat when meat means like, 500 different things. And these are actually conversations that patients really appreciate.

So they know you care when you're sitting down and saying, all right, what do you eat? What kind of meat? And you're not going to get rid of it. Maybe you can get it from here. Maybe you can get it from the CSA or the Farmer's Market. And again, quality over quantity. I know I harped on low-fat diets.

This is a study from JAMA where they took 63 people and put them in a metabolic ward. And they put them on a low glycemic Mediterranean diet. They put them in a low-fat diet. And they put them on a very low carbohydrate diet. They calorie-restricted them. They all lost a lot of weight. I think it was about 30 pounds.

On the left here is resting. Energy expenditure on the right is total energy expenditure. When you lose weight, your body senses it. And it slows itself down. Your metabolism drops, right? This is why weight loss is so hard. So what these two graphs show us is-- so here's a low-fat diet. Here's a low glycemic index Mediterranean diet. Here's a very low carbohydrate diet. Same thing here, here, and here.

People's metabolism is all over the map. So this woman's metabolism basically say the same, even though she lost 30 pounds. Whereas this woman's metabolism dropped. She's burning 500 less calories per day. So she's feeling really, really crummy after this diet where she's like, this is cool. I lost 30 pounds, and I still feel good.

And then you have these people over here where their actual energy expenditure went up. So what this means first off is we need to be individualized in our approaches. The other thing what this showed was the very low carbohydrate diets-- again, and my suspicion is this is all based on insulin. Those people that lost weight-- their metabolism dropped the least and on average was almost a null drop.

So if we want people to actually eat less and not feel really crummy afterwards, we need to think about what they're telling them. The Mediterranean diet was kind of in the middle. So for a lot of people that probably works out fine. But again, the low-fat diet, it just fails again and again. And this likely explains why so many of my patients are anxiety-ridden over their failure at losing weight.

But it's also good news, because we know that certain types of food can foster a better metabolism after weight loss. That same study showed that again, the low-carb and the low-GI diet was pretty good. They both increased insulin sensitivity. So what this means is you get more bang for your buck, OK.

So when these people go to the mall and get a Cinnabon, these people need to secrete way more insulin to pull that sugar out of their blood. These dieters had much higher in sun sensitivity, which means there's less insulin floating around. So if you're my patient, you have breast cancer. And I don't want you to have a lot of insulin floating around. I'm going to cite you a randomized study showing you which diet will lower your insulin the most.

And again, we have data to promote this. Again, we have a conversation of quality over quantity. And this is probably the most fun part of my job. We talk about everything. We talk about red wine. So people that drink a lot of red wine, I tell them where the highest amounts of anthocyanins are in Southern Italy and Southwest France.

We talk about Mediterranean diet. We talk about this is Buffalo mozzarella, which has a higher amount of conjugate linoleic acid than regular mozzarella cheese that you can get at Giant Eagle. And this is my favorite, Pecorino. And these are conversations that patients like. This is a website. I told you I was in a show that's called Eat the Butter. And it's obviously a play on the issues with butter.

But this is from a nonprofit that's actually from here in Pittsburgh. And you can click buttons to change food items here. And it's basically just a protein/fat with each meal. A green leafy veggie makes up the majority of it. And here you have sweet potatoes. Or there's some other. But just to give people a visualization about what foods they should eat. It is generally on the lower carb side, which again, I think for my patients is a little more helpful.

So this is my last slide. I'm wrapping it up. So tangible advice that I give to people-- again, to make an anti-cancer diet actually doable is does it not promote overeating? So if you're telling people to do something that's going to make them overeat, it doesn't matter what else you tell them. They're going to fail.

Does it minimally stimulate insulin and IGF? Again, from the mechanistic point of view, this will decrease your risk of cancer. It also probably decreases your risk of overeating and being obese. Does it provide a plethora of vitamins and nutrients? Does it incorporate social and cultural aspects of food?

If we don't do that fourth one, people are going to view food as a mathematical equation. And they're going to buy packaged foods and processed foods. We need to look at it culturally. And does it stress ourselves to be better troops? And I tell everyone food is not a math equation.