Ivor Cummins	00:00:37	Hey, everyone, Ivor Cummins here aka the Fat Emperor and we're bringing you another free podcast for your enjoyment. We only asked one thing, if you could go to ihda.ie, the website, and scroll down to the bottom of the homepage and share using the social media buttons. So this podcast is kept free by being funded by Irish Heart Disease Awareness. So we really need you to pause the video, pop on to ihda.ie and help us get the message out there on the calcification scan for middle risk people.
	00:01:13	Today we're back with an old favorite, Gabor Erdosi. And we're going to talk about leaky gut and some of this microbiome stuff that's getting everyone very excited. So I'm really looking forward to hearing all about that from Gabor. Hey, Gabor, how you doing?
Gabor Erdosi	00:01:28	Hey, Ivor, I'm doing fine. Thank you. I hope you are doing well, too after a lot of traveling. But finally we are here and discussing something purportedly important.
Ivor	00:01:42	I like that - purportedly important. Well, yeah, actually, I've sent a video by Dr. Steven Gundry the other day and he was promoting a new nutraceutical he had or multiple nutraceutical ingredients and a miracle kind of cure for leaky gut. And it was a 14 minute advertising video. I watched some of it. But yeah, pretty much pointing to leaky gut being the be all and end all, essentially. So I think the leaky gut is a really important thing, particularly if plant foods can cause the boundaries to break down, allowing proteins or endotoxins, I'm sure it's a major problem for some people. But you are looking at papers recently and kind of getting into what it's really about, when it may be an issue, when it may not be. So maybe you can kick off with your own kind of high level summary.
Gabor	00:02:32	In fact, I believe that there's a lot of confusion about what leaky gut is. Actually, I don't even think that when problems starts, it's a problem of a classical leaky gut. I think that leaky gut, the classical… let's define what a classical leaky gut is. You have a intestinal barrier, kind of a layer of intestinal cells covering the wall of your intestines is called epithelial cells. And leaky gut is defined that some things in the lumen, so within the pipe of your intestinal system, some things which shouldn't invade your system, just leak through the intestinal barrier. Meaning that just among or between the cells, they gain access to the systemic circulation. Scientifically it's called paracellular. Paracellular, meaning that between the cells. In fact, I believe

and very strongly believe it's kind of a scientific conviction already that this is not what happens early in the process of having problems. Intestinal inflammation due to bad food, environmental contaminants, and a lot of other things can cause this. So let's make it clear that when we talk about leaky gut, and when most people talk about leaky gut, it's not really a classical problem of your gut, really letting in things that are totally not intended to enter the systemic circulation. So I think it's part of the confusion.

What initially happens is that people develop a kind of dysbiosis, the name of changing microbiota, microbial strain composition in the intestines. You know that the intestines are full of microbiota, little lower in density in the upper small intestines and higher in density in the lower intestines, in the bowels. But actually, what really happens is that some things can really alter the composition of the microbiota and then it causes a little bit of inflammation, and this altered microbiota start releasing a lot of stuff. For example, it's called lipopolysaccharide. It's component of the bacterial cell wall in the gram-negative bacteria cell wall.

00:05:50 Just to present it through an example, the refined grains and simple sugars for example, can alter the small intestinal microbiota in a way that these lipopolysaccharide producing strains are in much higher numbers than in a normal microbiota. So, this lipopolysaccharide, the production is increased. Another problem is basically initially is not a problem, that this lipopolysaccharide can gain access to the human system. So, it can go through the intestinal barrier and it is done so by being taken up into so called chylomicrons. The chylomicrons are one kind of lipoproteins, and these lipoproteins, this means that we have a small ball or sphere which is water soluble on the outside, so it's polar, and a polar in the inside, meaning that all the lipids which are not water soluble are sequestered into this chylomicron. So this is actually how the release of digestive lipids enter the system and not the systemic circulation, not the blood initially but into the lymph. So the lymph circulation - the intestinal epithelial cells take up lipid when you eat fat, then it mainly causes off of triglycerides. And these triglycerides are broken down to free fatty acids and monoacylglycerol which means that only one fatty acid is esterified to the glycerol backbone, and these partially broken down lipids are taken up by the epithelial cells, and then they form this chylomicron, the lipoprotein and basically making it water soluble. And then this chylomicron is secreted on the other side of the intestinal wall into the lymphatic circulation.

So, long chain fatty acids are not delivered directly to the liver, but they are taken up into the lymphatic system. And the lymphatic system is also known for its role in immune cell distribution and its role in the immune function.

00:08:42 And it's no coincidence. I believe that this has evolved precisely due to the reason that these lipopolysaccharide and other bacterial fragments taken up together with long chain fatty acids can be neutralized by the immune system. So, when you absorb lipids and secrete it on the other side of the intestinal wall into chylomicrons, what happens is that they first go through several filters, immune filters. There is already a huge immune system in the intestinal wall. The biggest part of your immune system resides within the intestinal wall.

00:09:31 And then you have the intestine-associated immune organs or immune parts. For example, one is the mesentery, or people usually just call it belly fat or visceral fat or whatever. It actually hosts lymph nodes which are part of the immune system and the second filtering layer of the lymphatics coming from the intestines. And then these enter the systemic circulation, eventually reaches the liver and the liver has another layer of filter. When this blood enters the liver, then there is another layer of filtration, a lot of so called catfir? [Inaudible 00:10:28] cells, which are basically macrophages, liver specific macrophages, and you have this multiple layer of filtration. And one can only wonder why it has evolved this way. There are so many filters. And of course, when you understand that you have all these microbes, trillions of microbes within your digestive system, and inherently some bars or even some microbes can make it through the intestinal barrier, you need a lot of filters to get rid of them.

00:11:03 That’s important for understanding how our system works. I always like to start at understanding the anatomy and basic physiology so that you can better get a grasp of disease, how the physiology altered.

Ivor 00:11:24 Absolutely, Gabor. That's a great kind of run through there. So maybe if I just parse it out then, you mentioned up front about the lipopolysaccharide, these bacterial elements, and by eating simple sugars excessively, you can very much promote the biome or the bacteria in your gut, which would increase LPS or lipopolysaccharide. And they are the things you ideally don't want going across your gut barrier into your bloodstream, but they do get across. So one way to minimize a leaky gut problem would be by fewer simple sugars, I guess.

	00:12:01	Then you moved on to I suppose the lipoproteins, the chylomicrons, that take the energy from your food that you eat, they bring it across the gut lining into the lymph system, not the bloodstream, and there you can actually pass these monoglycerides to be as this fuel, etc. But also inside, I suppose, if within your stomach and your gut, your intestine, that's non self, that's the outside world to you. But inside behind your gut lining, you've got this organ which some people think of as belly fat but it's an organ that wraps around the intestine and it's a fatty organ called the mesentery.
Gabor	00:12:47	Yeah, Ivor, I call it mesentery, but my English pronunciation is of course not a guidance how to…
Ivor	00:12:54	In this case it is Gabor, because I believe it is mesentery, so we'll fix that. But you've got this organ wrapped around the intestine. And that's actually a clearing house of sort so that when bacteria do get through your gut and lipopolysaccharide, you've got an immune type organ, a fatty organ, the mesentery, that can be the first shield to dealing and managing with these kinds of toxins, whereas of course the glycerides and the fatty energy can pass through and be used through the lymph system. And then you mentioned when you get to the liver, you've got a second barrier. You've got cells that basically have macrophage to deal with bacteria at the liver level. It's a further safety system I guess. And it makes sense to me Gabor, because as we evolved, you know, the outside world, the nonself, is very threatening, not just with tigers and whatnot, but of course, all the microbes and viruses so to have multiple layers of immune activity makes absolute sense to be a very robust system, right?
Gabor	00:14:06	Yeah, I think it has evolved to deal with high loads from time to time. And it seems that these times when the body experiences high loads, happens when you eat simple sugars and fats together, which actually rarely happens in nature. But you are still able to deal with a certain threshold of this mixing. So some sugars, starches and lipids. But it seems that the very basic problem of modern metabolic diseases, at least those that are initiated by food, it's a problem of exceeding that threshold of mixing simple sugars and lipids. And lipids are actually necessary to be part of the problem, because lipids are necessary for the enhanced uptake of lipopolysaccharides. So as we just briefly mentioned during the introduction, these lipopolysaccharides are taken up together with long chain fatty acids and mainly with long, long chain fatty acids.

	00:15:32	So this kind of also explains why extremely low fat diets can work. Because even if you maintain a high lipopolysaccharide producing gut flora in small intestinal microbiota, you don't have a big problem because the translocation of lipopolysaccharides, the access gaining capability of these bacteria or debris is still low due to the lack of long chain fatty acids. When you are on a ketogenic diet, you have a high lipid uptake and potentially these lipopolysaccharides could be taken up in a huge number, but you lack the LPS producer microbiota, due to the fact that you are not eating almost any carbs, not to mention simple sugars. So you have a microbiota that doesn't promote LPS production. This can explain why you have particular problems in the middle, which is often called the swamp land of diets.
Ivor	00:16:47	Excellent. That's really cool Gabor, because this has come up, the Kempner Rice Diet, and these very high carb, ultra low fat diets that can achieve insulin sensitivity and reasonable health. Now I would call them to question for long term nutrient value, which is quite a separate thing. But a short term very high carb, low ultra low fat diet can get someone more insulin sensitive and they may feel quite better. Now, the mechanism, we talked some time ago about simple sugars, especially mixed with fats is a terrible combination to release GIP in the upper intestine, right? And that's not a good thing and also to have lower GLP 1 down the intestine. But that was very much a hormonal.
Gabor	00:17:37	There is even probably a link between the inflammatory responses, how these are stimulated by these intestinal hormones. Whenever you have a low GIP 1 response, and the high GIP response, you have a inflammatory response basically. So these are closely interconnected. We left some studies there but… yeah.
Ivor	00:18:07	Oh yeah, no, for sure. I don't mean to butt in, but absolutely. So the GIP/GLP 1 balance, which high simple sugars and potentiated by fats with the sugars, the GIP goes high, that was a hormonal view of the problematic mix of sugars, carbs and fats together. And now I think it's very elegant. That was a whole other mechanism whereby simple sugars and fats together may be detrimental. Though connected to the hormonal one, I absolutely agree, but this different view that the few eat very high carb, very, very low fat, you're going to have the simple sugars coming in, but you're not going to have the fat transport through the gut lining which would tend to bring in lipopolysaccharide. So maybe it's not so bad. And then if you eat a really high fat keto diet with very low carb, yet,

		you've got the lipopolysaccharides that may come across with the fat transport because you got a lot of fat coming in, but you haven't got the simple sugars to promote the biome that will generate a lot of lipopolysaccharides so you may be okay. But again with this whole thing around, the transport of fats and LPS, it's the same thing that high simple sugars and high fat together, the donut, the classic modern 50/50 carbon fat diet, is the worst no matter what way you look at it. It's quite elegant, I think.
Gabor	00:19:46	Yeah. And actually when you start thinking about it, it makes a lot of sense why you have these huge elevation in GIP and subsequently insulin. Because both hormones, half the system at least are acutely anti inflammatory. So, when you trigger a high inflammatory response, for the resolution, you need high anti inflammatory milieus. Basically that is done by a high insulin response in this very case.
	00:20:27	So, it explains that while you have this additional effect of simple sugars and and long chain fatty acids on GIP subsequently on insulin, recent studies show that GIP can be responsible for 60, 65% of the overall insulin response. So, that's basically what decides the big majority of your insulin response. And insulin, insulin is just an anti inflammatory hormone acutely. It's not when you have chronic hyperinsulinemia. But, as long as you have these returning insulin peaks and you can handle the load, it's clearly anti inflammatory hormone.
Ivor	00:21:21	Yeah. And you know just recently I was looking at papers and I'd seen many many years ago that insulin appropriately spiking can actually be good for endothelial cell health. And in terms of when you have infections, insulin rises to partake in the process of fixing the infection and resolving inflammation. So insulin isn't all bad once it's pulsatile spiking and acute locally. And then when you chronically drive the system into hyperinsulinemia, well then the kind of everything falls off the bush/bus/ [Inaudible 00:21:56] and the whole thing falls apart, which is probably what we're seeing with most modern chronic disease.
Gabor	00:22:03	Yeah. Well then we are back of course to the chicken and egg problem of hyperinsulinemia and insulin resistance - which comes first. I think a recent study in the Journal of Clinical Investigation gave us some clue how all this may happen. And we are back again to GIP, the upper intestinal hormone, which is exaggerated when you eat simple carbs with long chain fats. It

was just a mouse study, but what it showed was that frequent high GIP secretion can blunt leptin signaling in the brain.

00:22:56 What does that mean? Leptin is responsible for suppressing appetite and increasing energy expenditure. So, when you kind of overeat or you start depositing fat in your adipose tissue, leptin levels go up and then signal to the brain that you have enough reserves, "Please stop this heavy eating pattern of Christmas," you start burning more fat through activation of [Inaudible 00:23:32], so browning of white adipose tissue, activation of brown adipose tissue and the reduction in food intake. And it seems that when you over stimulate GIP in the upper small intestines, this very high GIP load in the brain, they actually showed the mechanism but it's not very important right now. It can blunt this effect of leptin. And then you have high circulating leptin, but your brain doesn't sense this level of leptin, but it's still a sense of low leptin levels, which is a signal to continue eating as much as you can.

00:24:20 So, basically, I think that eating the wrong things can plausibly trigger this scientifically called hyperphagia, which is kind of overeating. It can all start with this overeating signal. And then of course, when you have depositing fat, together with increasing inflammation, and increasing insulin resistance, hyperinsulinemia is the response to cover the lipid outflow from adipose tissues and somehow compensate for the high leptin and inflammatory signalings. So it initiates a cascade and then you are really into problems.

00:25:12 Recently, several studies showed that actually the real problem is not hyperinsulinemia per se but insulin resistance. A lot of people say that the problem is hyperinsulinemia, but mechanistically, the underlying problem of many, many disease pathways is insulin resistance. So starting in the endothelial wall, when this knocks and nitric oxide balance is tilted and in many other tissues, you see that the blood pressure regulation or the vessel constriction. So, insulin is normally has a vessel dilating effect, so your blood vessels are dilated and your blood pressure should go down. What you see in chronic insulin resistance is high blood pressure, which is kind of paradoxical because if you have a high circulating hormone with a vessel dilating effect, which is telling to the blood vessels that, "Okay relax and let this blood pressure drop," and you still have high blood pressure, that's a little bit of paradoxical. But the explanation, well, I think I just tweeted it earlier today that there are some researchers in Korea found an explanation that it's blunted insulin signaling, so kind of insulin resistance in the insulin signaling pathway, there is a break. Despite the

prevailing hyperinsulinemia, this blood vessel relaxing effect is lost.

Ivor 00:27:16 Exactly. And I know there were some debates about this in the past and you know, some people think that, “Well insulin resistance isn't necessarily a problem and it's really hyperinsulinemia.” I always loved what Joe Kraft said decades ago. He said, “Hyperinsulinemia and insulin resistance, they are not combatants, they are one and the same.” And for him what that meant was not that they were the exact same, but the combination of insulin resistance with hyperinsulinemia is the problem, right? And if you have insulin resistance locally, say on a very low carb diet with low insulin, that's only a response to a dietary regime. In other words, glucose sparing. But what you're talking about is pathological insulin resistance, generally with accompanying high insulin, trying to get the job done, trying to keep the fat in the fat cells for someone who's diabetic, you know, trying to basically act as insulin act in a correct way. But it's in the milieu of insulin resistance, because the control systems now have become unhinged. I like to see both of them together, but I agree with you. Yeah, insulin resistance is the fundamental dysfunction and it's concurrent with hyperinsulinemia when you're in trouble.

Gabor 00:28:42 Yeah. I mean, it's rather straightforward when you have a look at normal physiological roles of insulin. So it's anti inflammatory, it should relax your real blood vessel wall, the smooth muscle cells so that reducing blood pressure, dilating the blood vessels. You see just the opposite in insulin resistance. So that's why it's the problem of insulin resistance because there is a resistance. Even the high insulin cannot signal through its normal physiological role.

Ivor 00:29:24 Yes. I like the way that's parsed out. And also insulin for sure, you know, in general, that's a positive thing and it makes sense. It's one of the oldest hormones and all of its functions will be positive unless the control system is broken, in which case it becomes part of the problem. There was one experiment under some evidence that high insulin alone when excessive, can go from being a good thing with vessel dilation and endothelial health to being a bad thing. I’m thinking of the famous one in the 50s with the dogs when they injected one leg and they separated it from the body, and they developed atherosclerosis in a very high insulin concentration leg with exogenous insulin. But again, that was over a long period and the very high insulin super normal could have generated insulin resistance and the accompanying issues in that particular limb. What do you think?

Gabor	00:30:23	Of course, after a while it's a cascade, it's a self perpetuating cascade. So high insulin will result in more insulin resistance that goes on and on of course. I think that's normal thinking. The problem has always been kind of pinpointing the the initiator effect that starts either hyperinsulinemia or starts insulin resistance. And I think one good candidate in this is inflammation. You see developing hyperinsulinemia very early after an infection, for example. You start seeing this glucose sparing, but this glucose sparing caused not by fuel competition, which is the case when you are on a very low carb diet for example, then you have fuel competition. Ketones and fatty acids compete for oxidation in the cells and just outcompete glucose oxidation and then subsequently glucose uptake. That's what you see as insulin resistance, if you measure insulin resistance as glucose uptake into cells. But why should you measure insulin resistance as glucose uptake into the cells? This is just kind of a convention. There is nothing which should make it the good standard of measuring Insulin resistance. Why don't we measure insulin resistance as taking up fatty acids into fat cells? Because that is at least as important, a physiological role of insulin to stimulate lipid uptake into fat cells. And I think actually that's much closer to the root problem.
	00:32:25	This brings us back to chylomicrons secretion. It was recently shown that chylomicrons secretion, again, this is lipid, digested lipid release from the small intestines, into the system can be influenced by simple sugars, particularly glucose. Glucose can stimulate chylomicron secretion. Another factor that can stimulate chylomicron secretion is GIP two, which is co-secreted with the GIP 1. This hormone GIP 2 is known for its role in the maintenance of intestinal barrier. And then what turned out was that glucose and GIP 2 stimulate chylomicron secretion by very distinct pathways. And what happens is that glucose results in accelerated accumulation of lipids of triglycerides within the chylomicron lipoproteins and this results huge sized chylomicrons. And what GIP 2 does is it increases lymphatic flow so the delivery speed of chylomicrons away [Inaudible 00:33:58] from the intestines.
	00:34:02	The difference between the two is extremely important. Because what happens is that when you eat simple sugars together with long chain fats, you will secrete the same number of chylomicrons but with a lot more lipids in each of them. This is kind of non physiological, we are not really prepared to take this hit long term. And then there is a double whammy of

		getting all these lipopolysaccharides within this huge chylomicrons. This is at the very root of the problems I believe.
Ivor	00:34:43	Yeah, and that circles right back again to the classic problem, that for most people, particularly modern people, it's the mixture of simple sugars and again, it might be with steel rolled oats or very fiber intact processed carbohydrates, are truly more complex slow digesting lower GIP higher GLP 1. But most complex carbohydrates these days that are marketed are not so complex, right? A lot of them are relatively split up very quickly into simple sugars. So we have this standard diet that has a heavy load of relatively simple sugars mixed with of course the fats.
	00:35:27	Now, I wonder if, there are some people who seem to be sensitive to fats, and I'm not sure, there may be people that even with lowish carb but with certain fatty acids may still have a slightly high insulin or an inflammatory response, and I know toll receptor 4 gets involved there too, but if someone has a response to fats that doesn't appear to be ideal in blood markers, is it possible thing to try to go really, really low carb when you're eating those fats even more than normal?
Gabor	00:36:05	It's difficult to tell. I mean, when you are into this kind of disentangling the puzzle or putting the pieces together, I think the personalization is still far down the road. So it's very difficult to tell what our outliers are. So what the problem is with outliers, those who do not react normally kind of as the majority to dietary interventions. It can be a big difference in microbiota composition. There is some kind of an overgrowth which is not typical. It can be an alteration in how their intestines handle lipids or can be a problem of an enzyme of chylomicron assembling, you know, the enzymes used as in the adipose tissue is [Inaudible 00:37:11] and all the others. There are so many steps involved that a significant, just one mutation for example in one enzyme can cause significant differences. But I think most frequently, the biggest difference between individual people is in their immune function. So, how your immune system developed when you were an infant or a small child and what you were exposed to? These are hugely individual and almost never taken into consideration that the immune function is one of the highest variable in this process.
Ivor	00:38:03	Yeah, individualization. That's pretty tricky. I mean, working out even the core of what's going on here generally is hard enough, I guess, though we seem to be getting very close to the essence. I was thinking as well of the ApoE4 people who have extensive

heart disease and calcification, and there seems to be a sensitivity to cheeses, maybe excessive fats. And I wondered, are they people where microbiome has over decades of processed food that drove the cardiac disease, maybe ended up with a biome and a scenario and genetic disposition that does actually make them now sensitive to fats. Though I am curious too, and the work of Dr. Gundry is all around cheese on animal fats. And I wonder how much is the casein A2 or A1, the cheeses' proteins which may trigger the immune system and it may actually be a big part of that observation more than basic animal fats themselves.

Gabor 00:39:07 I'm not an expert on milk proteins so I don't really know much about... I know some about the differences but it's highly plausible that what you are exposed to intrauterine, so within the womb or after as an infant or a small child, you can get used to. I mean, you can develop immune tolerance towards that and what you are not exposed to. Or alternatively it can trigger problems very early on. Now it's becoming clear that between sexes, I mean, male and female, the biggest differences in immune function because females has evolved to deal with the huge immune stress of having a baby. So it's a not non self, different kind of tolerance and a very different kind of immune system is necessary for that. And that's why they are more tolerant to these kind of diseases as well. At least as long as they are pre menopausal. After menopause, you see all these problems accelerating and many women just catch up with men with regard to metabolic diseases and it's in their immune function. And then it's the same with aging. What you see is the immune system is a major determinant of aging, how you age, how fast, in what way, and these kind of things. So, once you start looking into immunity, then you cannot really cope with the comments on on Twitter and elsewhere that, well, inflammation is just there to repair something or deal with the problem. I think we still don't fully understand what inflammation is and how it works. So actually, I started using immune activation because people seem less triggered when I use immune activation compared to inflammation. Because there are so many ways of different types of immune activation. When we talk about inflammation, one should be aware that when you have a Helminth infection or a bacterial infection or a viral infection, or you have a so called sterile inflammation, endogenous or internal injury of some kind, your immune system is activated, your immune system is activated after every single meal. You see the cytokines go up and down together with the meal and these cytokines assist, for example, the pancreas in releasing the necessary insulin and so on and so

		forth. So the age of immunology has just started in the 90s, I think, really started in the 90s. And that's why I had to do a complete deep dive because I graduated in the mid 90s, and all this innate immunity and innate immune memory and these kind of things were completely undiscovered back then or were just discovered, which takes decades to make it into textbooks and finally into the education system.
Ivor	00:43:03	Yeah, there's so much in the immune system. I mean, it's basically, I suppose, because it evolved with everything else over such a long period, it's kind of inveigled itself into every aspect of metabolism. So it's not simply an attack dog when you get an infection. It's, as you say, it's involved with even digestion of a meal and many, many…
Gabor	00:43:24	And then what you see is that those people at the forefront of these metabolic diseases, for example, in cardiovascular disease, just basically stopped discussing different lipoprotein levels and these kinds of things and the focus has been constantly shifting to the different kinds of immune activations, because it turns out that by the immune activation, you are able to explain the difference is in lipoprotein levels or cholesterol levels or whatever. So, if you step back and take a different perspective on these diseases, kind of an immune and metabolism perspective, what you see is that, basically a lot more things can be explained. You don't have to pinpoint single symptoms or single markers or surrogates or whatever, or proxies, but you can start thinking in a system, in a true system, how does the system interacts with the environment and how this is disrupted in disease, and how the body tries to heal itself kind of with the inflammation and then how it fails to do so if the trigger persists? And I think it's absolutely fascinating. And those people who are stuck at whatever LDL cholesterol or LDL particle or these kind of things are missing out huge in a huge way.
Ivor	00:45:06	I would agree, and again, [Inaudible 00:45:08] who spend here is arguing against the simplistic cholesterol particle theory because, you know, the lipoproteins engage in all the digestion, metabolism engage in the immune system response, they are intimately linked to insulin dynamics on our hormonal dynamics and even stress. So, basically, they are the cart, not the horse. Yes, they move up and down and correlate with disease states because they're engaged in the processes. But I agree with you, you've got to look at the actual core processes of what's driving the problem and the root causes. A big part of which is

understanding the immune system to really get to an understanding of how to resolve it.

00:45:54 One thing that is really confused people I think over the decades is because lipid lowering had a beneficial effect, which tended to track with the degree of response of the person to the pharmaceutical. You know, lower LDL, higher response to the drug, maybe more benefits. It was used to seal the deal that the lipoprotein number was somehow central. But that yet another obfuscation sadly. But I think we're not going to see much change and view there because if there are effective, multi multi billion dollar products that helped with LDL numbers and help with cardiac outcomes, and there are no drugs products or path forward in terms of resolving the immune system and the root causes, at least no profitable ones or actionable ones with simple pills, then this situation is going to stay the way it is for a long time yet. So I guess it's just for us to keep working on the root causes.

00:47:05 There is one thing Gabor, I had a chat with Dr. Malcolm Kendrick in a podcast a couple of weeks ago. And you might not agree with everything he said, but I think he raises some really fascinating points. And one of them does relate to the immune system for sure. The rheumatoid arthritis and lupus and autoimmune conditions can cause massively accelerated atherosclerosis, completely independent of lipoproteins. And even mentioned again, one I'm not sure you're so much into, but he mentioned the sickle cell anemia, that he has seen cases of a 14-year-old boy, whose younger brother died at four or five years old of a stroke, and the 14-year-old boy himself needed to get a leg amputated because he had so much calcified atherosclerosis throughout his whole body and indeed his leg. They had to get a leg amputated. And that boy, his cholesterol was perfectly normal, but the sickle cells through their shape and other immune factors destroyed the endothelium or damaged it enough that there was atherosclerosis everywhere in the body.

00:48:19 He was just making the point that there's much closer things to root cause, vastly more important illustrated by cases like that where really the number of particles is in the heap in a place. [Inaudible 00:48:31] It's just so far remote from the real mechanisms. What do you think?

Gabor 00:48:37 Yeah, I think what we really have to work on is finding the unifying mechanisms. I think Dr. Kendrick has done a great job on this in pointing the blood clotting, the traumatic effects as a

unifying mechanism of atherosclerosis. And of course, it's also important to find the different contributing factors because of course, in this discussion for example, we found the combination of simple sugars and long chain fatty acids, kind of a structure free ultra processed food, if you can just give it one name a structure free, I think is extremely important here, which is a typical driver of metabolic disease and we discussed the pathways the mechanisms, but there are other initiating factors. So you see sometimes single compounds, for example, lead, lead poisoning, or arsenic poisoning, or different environmental pollutants, BPA, BPS, and all these others, which obviously do not go through the same pathway, but somewhere downstream join into this and then a little bit later on, a little bit downstream stimulate the same detrimental processes.

00:50:28 Yeah, we have to find the unifying mechanisms and all the initiating mechanisms as well which converge upon these unifying mechanisms. Dr. Kendrick has done a great job, I think pinpointing blood clotting as a unifying mechanism, direct mechanism of causing atherosclerosis. I think, in general, what seems to be a unifying mechanism, and I have a longer Twitter thread on this, is the chronic suppression of autophagia, which means that the average people, modern people are never in maintenance, repair and maintenance mode. Their system is in chronic anabolic mode, in a chronic immune activation and not in the repair immune type of immune activation but the defense type of immune activation. Just that they don't leave enough time for repairing. Despite these triggers and then we are back to for example, eating simple sugars with long chain fats, and some people might say that, "Okay, but my grandparents always had fatty meat with mashed potatoes and they only developed these problems in their 60s or 70s or even 80s, and why this has accelerated now?" This is the unifying mechanism. Because other differences in lifestyle or the load of this kind of structure free foods is much higher and then other lifestyle factors converge on the same pathways, it just keeps the average folk in a chronic anabolic state, never cleaning up the system from the inside, and that's what chronic suppression of autophagia.

Ivor 00:53:27 Yeah. And that is a major problem nowadays. I mean, those grandparents, the closest they got to processed food really was mashed potato. But nowadays, we've got foods everywhere that most people don't even think of as bad processed food. They, in some cases, think of it as healthy whole grains and whatnot.

Gabor	00:53:47	Energy density is a proxy for lack of structure in foods. Because when you eat boiled potatoes for example, the energy density is still low. And this i represented in these intestinal responses we have discussed so many times. And then of course you eat good load of meat, you add some pickles or whatever, some vegetables or salad or whatever. People used to eat or start with a green salad and eat the meat with the potatoes next. And this is also important. So I think these major steps in this initiating pathway, you can eat a lot of things as long as there is no simple sugar dumping into your small intestine. So if it's a long digesting this simple sugars, these simple sugars are released slowly and you can take off almost as quickly as it appears and there is no food basically for the microbes left. There is no overgrowth of these [Inaudible 00:55:04] producers.
	00:55:05	It's a very interesting study I saw that when you eat these simple sugars time and time again, what the body does is it increases glucose absorption. This is absolutely paradoxical, at least at first sight it seems paradoxical, if this accelerated glucose absorption, which will result in the extremely high GIP and following crazy high insulin response. If those are detrimental, why the body still increases glucose absorption instead of trying to get rid of it so that it remains into the intestines and goes further down and maybe you've developed some gas and get rid of it? I think the problem is exactly that there is something which is… there is even worse than a very high insulin and GIP responses. And that is the bacterial overgrowth simple sugars hanging around for a long time in the intestines can cause. So that's even worse because that's highly inflammatory and there is a threshold with which the body cannot cope with. And beyond that threshold, you are really doomed because you develop very severe intestinal inflammation. Then you really develop leaky gut, meaning that the paracellular leakage of bacteria and debris. And paracellular meaning again, that between your intestinal cells, and if it goes between your intestinal cells, then it goes straight to the liver, there are no filters like your intestinal immune system, your mesentery immune system, it hits the liver. Even if the liver has its own filter, it's way beyond its threshold that it can deal with. So that is liver inflammation and cirrhosis, and liver cancer and whatever.
Ivor	57:04	And you know what, there's a couple things there. I'm going to grab those excellent stuff Gabor. One is, yes, so the body preferentially absorbs more glucose when a lot of simple sugars come in. And that makes sense by your very description not to

		leave an overload of simple sugars for bad biome overgrowth in the intestine.
Gabor	00:57:22	Yeah, that's my assumption, at least or speculation, because we don't really know. I mean, evolutionary, it makes sense that you do seemingly bad thing to your system if there is something even worse, which can happen otherwise.
Ivor	00:57:40	Yeah. And I agree, it's a speculation or a hypothesis, that's fine. But it would make sense evolutionary wise because evolutionary, there's a seasonal aspect. And if you get a lot of simple sugars, well, bringing them all in and converting them to fats could be good for the winter. And also, you're doing indirectly what you describe, you're getting in the mount of an area where they could be a problem. And it reminds me of the SGLT 2 inhibitors that tend to stop the glucose transport and send more of the sugars at the bottom end. But then I think we've seen with those urinary tract infections increasing and yeast infections down in the nether regions. So it might be a little sign that what you describe is quite appropriate. You know? There are knock on effects to trying to dump the glucose and of course anyone listening knows just stop eating the glucose if you've got an issue. Don't be trying to dump it out your ass so to speak.
Gabor	00:58:39	Yeah, I find this eating for winter hypothesis a little bit controversial, because it doesn't really happen in nature. I spent quite some time looking into what happens to hibernating animals, which is another fascinating area of research. I think we can get such a great understanding from what happens to hibernating animals because they become obese, they become insulin resistant, they become hyperlept anemic, [Inaudible 00:59:12] so their leptin is also crazy high. And then they start this hibernation and get back to normal. You don't even see the effects when somebody is yoyo dieting. So people gain weight and lose weight and gain weight again. And then there are a lot of adverse effects of that, but these are completely missing from these hibernating animals.
	00:59:40	Yeah, just to focusing on the initiation of obesity for these hibernating animals. If you ever look at a grizzly bear for example, just check out my Twitter feed again, search with my handle and hibernation or hibernating or whatever and you will find these stories. It doesn't really happen the same way to these bears. They don't eat very simple sugars and fat together. I mean they eat salmon, they can eat tons of salmon, they eat berries, but try to eat salmon and berries; you will never get

obese. Never. You can eat as much berries and salmon as you want. It's basically impossible. So what happens is, I believe that there is a switch, probably a circadian switch when they start becoming shorter or whatever. I haven't even looked into this art, but there is a switch which makes these animals leptin resistant. And it starts with this which they become leptin resistant so that they can eat more and they can gain all the fat. And when they are leptin resistant just before winter, they have very high leptin levels, they are insulin resistant. They have very high insulin levels. But one difference compared to the pathological insulin resistance in humans is that they also have very high adiponectin levels, which is an insulin sensitizing hormone release by adipose tissue. Chronically insulin resistant humans have just the opposite - high leptin and low adiponectin levels. And these bears have very high adiponectin levels.

01:01:34 There is a yearly graphs I posted on Twitter and adiponectin concentrations closely follow that of leptin. So it is maybe a way they can cope with this obesity and insulin insensitivity, which is basically just glucose bearing in their case, it's not pathological at all.

Ivor 01:01:55 Right.

Gabor 01:01:55 I think it's absolutely fascinating.

Ivor 01:01:58 Absolutely. And you know, it might be they are somehow analogous to a very low carb or keto person having a glucose sparing insulin resistance, but they really do have high insulin and high leptin in contrast, but then the high adiponectin shows that it's a benign state, which indeed it is. [Inaudible 01:02:18] Dr. Garod Orly in one of my early podcasts actually pointed that out that these guys have all many of the bad signs yet they never develop hypertension, or the pathological sequelae of the problem. They have a very special scenario.

01:02:35 Now, I will ask one thing that occurs to me Fabor. So they are eating salmon, a lot of polyunsaturated fats, which you could say are obesogenic, but maybe not necessarily pathogenic, but obesogenic and they're eating a lot of berries, which are not too simple sugars, there are fibers as well, etc. Now, if you fed that bear kinds of donuts, like true simple sugars, refined starches and modern fats, I'm guessing that bear would get quite a different kind of obesity.

Gabor 01:03:13 Yeah, most likely, yes, I believe. Perhaps all these types of foods also play a role in how this leptin insensitivity is triggered. I can

		only speculate. I haven't looked into this. I should. I have quite a few papers on my table, it's lack of time. And I always find something even more interesting to read.
Ivor	01:03:43	Oh, I know the feeling Gabor. I know I was being provocative there. I was just thinking to myself as you were speaking.
Gabor	01:03:50	We can also take a look from an intestinal perspective. For example, the high omega 3s in salmon and cod water fish, what they feed on omega 3s are known to be reinforcing the intestinal barrier function while omega 6s can do the opposite. So, if they have this high omega 3 load, this may be an insulin sensitizing, or adiponectin promoting and perhaps the gut is also involved by having a very strong intestinal barrier function.
Ivor	01:04:35	Yes, absolutely. And you know, when I mentioned obesogenic in terms of PUFAs or polyunsaturates, I did really mean omega 6. In fairness to the salmon would have an insulin sensitizing omega 3 effect, better gut barrier, as you say, so it's a different kind of thing. I think the circadian those switches [Inaudible 01:04:56] I'm sure are more powerful in bears because of their very nature of probably have an overriding function too. Absolutely. I'm just curious about if you fed them a modern kind of high sugar, high fat diet, would things quite work so well?
	01:05:11	You know, it also brings us back to what we started and what we're talking about today, the permeability, the leaky gut. So what can cause the leaky gut? Well, we've said the simple sugars, overload sugar and fats together where the fat potentiates the responses.
Gabor	01:05:30	I think… well, we have to make it clear what intestinal permeability is. And some pollutants and some compounds can really make this paracellular flow. So between the cells, things getting through this type of permeability can be increased by certain compounds. But the typical problem is not that… what I found a major problem in microbiota research and intestinal permeability research and generally the metabolic and the immune activation perspective in the digestive system is that these microbiota studies use fecal shotgun sequencing. Meaning that they take a stool sample and scratch the surface of the stool sample and then they try to determine what kind of bacteria are there in that sample. But the very basic problem in my opinion, at least is that the interaction between food, microbes and host metabolism and the host immune system occur not at the surface of your stool, not even in your distal large intestines, but it occurs on mucosal surfaces. Meaning that

		on the surface of your bowels, the intestines. And when you have a look at the surface distribution of different parts of your digestive system, it turns out that human small intestines, at least an order of magnitude larger in surface than the large intestines. So, even though there are a lot more bacteria in your large intestines, in your bowels, by numbers, the surface area where the actual interaction happens is much larger in the small intestines. And we have so few studies looking at the small intestinal microbiota, the influence of diet on the small intestinal microbiota, the small intestine on immune system, this small intestinal leakiness or permeability and these kind of things. There are just a handful of studies. And what we know, the vast majority of our knowledge comes from associations from stool samples, which is I think, is just due to convenience and not due to good science.
Ivor	01:08:23	Exactly, Gabor. They're looking for the magic pill, as always. It's the easy entry and then find correlations, find relationships, jump to hypotheses, and of course, run after some profitable targets.
Gabor	01:08:39	Yeah. And all these discussions about short chain fatty acids purportedly are only in your large intestines, in your bowels, so you need to eat a lot of fibers to keep these microbes happy. There are very basic understandings how the host influences the microbiota composition for example, and how host metabolism, how disturbances in host metabolism can cause disturbances in the maintenance of proper gut flora microbiota composition. Because if you are not able to burn, to run an oxidative metabolism in your intestines, then you leak oxygen into the lumen of the pipes of the intestines and then you are not able to maintain a strictly or at least partly strictly anaerobic. So, lacking oxygen environment for the necessary microbes. And if these microbes get oxygen and a lot of oxygen close to your intestinal wall, then these will be not the microbes you want to be close to your intestinal.
	01:10:02	That’s a very basic problem, which is often often neglected. And then the host, I mean, we have evolved over hundreds of millions of years since there are microbes in animal digestive systems. We have evolved together and the host has certain tools, how to influence the composition of the microbiota. There is a mucus, composition of mucus, thickness of mucus, there is all the antimicrobial compounds secreted by immune cells. There is direct immune cell interactions, reaching through the intestinal wall. We have a lot of tools and that is the basic metabolism just sequester oxygen, so that they don't get

oxygen and then in itself is highly protective and promotes a certain type of microbiota composition.

01:11:05 We have a lot of tools and nobody seems to talk about this. Everybody seems to talk about eating fibers. Again, short chain fatty acids in your stool, which is completely unimportant. Who the hell cares about the fatty acid short chain fatty acid content of your stool? What's really important is the short chain fatty acid content of your blood, not even what happens in your intestines. Again, there are associations. But we are just at the beginning of disentangling how the actual interaction happens and how different microbial strains, not high level associations of [Inaudible 01:11:54] what happens and the ratio, how its dealt with, and this kind of barely important things, but how certain kinds of strange work together with other strains so that the [Inaudible 01:12:06] are handled properly. And the 4-chain fatty acids are synthesized properly, and so on and so forth.

01:12:15 There are so many details which we just started putting together. And people still seem to focus on not important and easy to measure things.

Gabor 01:12:27 Yeah. Gabor, I love that, the arrow of cause of course as always goes multiple directions. I always say to people about the microbiome, if you want to eat a healthy microbiome, or say if you want to have one, well, they need all the right types of foods and maintain your own health. And now you've described not just that, that the foods contribute to good, if you will, microbiome, but the host, the self, the health of the body, and the physiology contributes also to the actual microbiome makeup. So it's a very complex system. But we've seen over the decades again and again, they go for the simplistic but not quite true answer based on kind of correlations. We've seen it with cholesterol, we've seen it with damn near everything at this point. So if we go and we just run through the leaky gut again before we roll to a halt. The simple sugars and other problem foods can drive a poor microbiome and leaky gut and paracellular transport, lipopolysaccharide and problems. The health of the physiology can also directly affect all of those vectors and the microbiome and the leakiness, as we've described.

01:13:47 There's also, I will go through one more, the problematic plant proteins and lectins and gliadin, and you know, wheat germ Agra gluten, but particularly lectins that Dr. Gundry and many others promote so strongly. What about the plant world proteins that have a direct deleterious effect on the good health

and tend towards leaky gut? And indeed when said proteins do come through paracellular or otherwise, they then contribute to an immune reaction, which can be very deleterious, develop autoimmune, or God knows, all the other things associated with over immune activation by your words. So what about the plant world problematic compounds, which are, different ones are probably problematic for different people and some people are extremely robust to any of them.

Gabor 01:14:46 Yeah. I haven't looked into this in high detail. I could say that I don't really know. What I suspect is that these problems dwarf if you don't feed the system with simple sugars. And if you maintain a steady low blood glucose levels, so no hive spikes, no steady hypoglycemia, and then you eat otherwise whole foods, then it's a minor problem. I strongly believe it It must be a minor problem, because then you can maintain a stronger intestinal barrier function. And even if there are small issues here and there, I mean, some aggressive molecules penetrating, you can very quickly repair those. The intestinal surface you know surface, you know surface is a very highly proliferating tissue so it renews in two to seven days approximately depending on subtypes also.

01:16:03 It can be quickly repaired if you leave the time for it, and if it's otherwise healthy, and the host metabolism is healthy. Your biggest problem is eating whole foods, whole plant foods. But not eating simple sugars, I don't actually think that it's a major problem. But it contains a lot of assumptions and again, I haven't looked into this in detail.

Ivor 01:16:32 Yeah, no, fair enough, Gabor. Absolutely. And it's great to have an alternative view as well. There's a lot of people running with the plant as poison kind of whole theme. And I think in fairness wheat, because we know about celiac disease, and maybe one in 100 humans will essentially die if they eat these compounds. There are certainly compounds out there that are problematic for many people, but it may be very exaggerated the extent to which that's the case, I think you're saying. And it could very well be that if people were eating a whole foods diet with no simple sugars and getting a lot of nutrient density from meat, fish, eggs, to make the physiology really healthy and having stable blood glucose, blood insulin, and a very competent immune system, it may very well be that some of these so called problematic plant proteins are not so bad at all and we could manage them or deal with them, especially if we're not eating six times a day and overloading the system and having no autophagy to your earlier point, we may very well be

		surprisingly robust. I think if in like process control and engineering, if you lower the water level, the rocks begin to appear. So there's many rocks there, but if you keep the water level, your physiological health quite high, these potential rocks of plant proteins may simply never surface.
	01:18:02	I like that idea. And I think that if people have particular issues that are profound or autoimmune in nature, it seems to be quite a good idea to do an ultimate elimination diet. You know, you've heard about the carnivore movement. But again, after doing the ultimate elimination down to meat and water where you're basically have eradicated everything, you I suppose can reintroduce carefully, more benign vegetables, broccoli, cauliflower, etc. and maybe just carefully then bring back in all the foods and see where your pressure point perhaps was.
Gabor	01:18:40	I don't think you have to. I mean, what I would do as a perfect diet for myself, I'm also an autoimmune patient myself, I would do a meat. When I say meat, it's kind of animals, fish, eggs, organ meats, whatever I like. And I believe that those are healthy foods. And I would eat berries Ad libitum as much as I want. Always the meat first and then the berries. And I think that's a low, low stress kind of diet. So you can eat certain plants in high amounts without any problems. I'm sure because I can myself eat some plants and I have to be more aware of others doing some disturbances, some GI disturbances. And I literally can feel it in my lower back. And I kind of cheat. I can say cheating. Other people of course wouldn't say cheating when we're eating the stuff that trigger me.
	01:19:57	Yeah, it's straightforward by now after many years of experimentation that some things I can eat, some plants in whatever amounts without any problems, and others even in small amounts, will trigger a response.
Ivor	01:20:13	Well, just because the question is bagging now for anyone listening, so berries, yes, and maybe just name a few other plants would you find benign, and then a few plant world things where, as you mentioned you would not do so well. Just to give a rough idea.
Gabor	01:20:29	You mean just listed the plants I'm sensitive?
Ivor	01:20:33	Well, yeah, I mean, a few of the ones you might be sensitive to, I know you had a starches type problem.

Gabor	01:20:39	Yeah, especially grains. I can eat some potatoes as kind of a dessert. It doesn't seem to trigger a lot of response anymore. Even though it contains starch. Probably the starch I eat is lower, and it may be that everything is about the rate of appearance in the small intestines. As we discussed previously, lack of structure, this is the major problem. And if you eat some potatoes and don't think of a huge plate of potatoes, but I eat some whole animal foods and then add a few tablespoons of potatoes for example, to a meal is kind of a dessert or with the last bites of the meat or the animal food. Then I don't seem to be triggered. I can even eat a little bit of white rice without much problems. Again, I eat that as kind of a dessert, with the last bites of the meat. I can eat as much berries or relatively low sugar fruits if I want to without issues.
	01:22:04	I think there can be another issue if you eat a lot of fruits, it can still wreak havoc on your teeth, regardless of lack of intestinal problems. That's another consideration.
Ivor	01:22:23	Yeah. And actually what you mentioned there, what you can eat, it's still a relatively small group of things. For instance, all the other vegetables and various legumes and all.
Gabor	01:22:36	Yeah. Once in a while I can eat some beans for example, not huge amounts, but I'm really sensitive to beans. I'm also sensitive… probably I'm in general FODMAP sensitive. So if I eat say cauliflower, not more than a few bites, or broccoli and these kinds of things, it just makes me bloated
Ivor	01:22:57	Ah! And I thought of those as relatively benign in terms of lectins and other supposed problems, but there you go. So you're careful with your plant sources and you place the meats, fish, eggs and the fatty and protein up front in the meal and then allow the carb at the back end for slow absorption following the fat and protein down through the intestines.
Gabor	01:23:23	[Inaudible 01:23:23] with the glucose spike as well. So you will experience almost the same flat glucose line as without those carbs. So there is no punishment in that regard. Yeah, I cannot feel these carbs in my lower back. That's the most important things, personally at least.
Ivor	01:23:49	That's your bellwether. That's great, yes, so meat, fish, eggs and all these nutrient dense ancestral foods, and then some of the plants and maybe some potatoes or white rice on the backend of the meal. And then other meals, ideally on a one big meal a day, maybe one more meals should be enough, couple of meals

		a day, that's what I find. And maybe the other meal will be, you know, relatively very low carb indeed compared to the main meal. That's a safe diet, especially for someone who has any immune or diabetic type physiology, as you say ,it's going to keep your glucose insulin low, it's going to keep your immune reaction low and probably to close it, it's going to probably keep your leaky gut type phenomena relatively low as well.
Gabor	01:24:39	Yeah. I have another measure. My ketones, for example, but I can imagine that I'm in ketosis, dietary ketosis every morning. Because even if I eat, let's say, hundred grams of carbs, that's mostly in whole foods. So slowly releasing, got added to the end of the meal. And then I usually eat in a restricted feeding window, like six hours for example. I usually two meals a day, eat one at 8, 9 in the morning and around 3 in the afternoon. So that's more or less, you know, six hour window. And then after 3 in the afternoon I don't eat anything. By the morning, I can easily imagine that those slowly absorbing carbs disappear and and I'm in dietary ketosis. And I'm not very much interested in measuring ketones but certainly sooner or later I will come to that.
Ivor	01:25:42	Great stuff, Gabor. Sound similar to me though. I often go for a breakfast and an evening meal at 5 or 6, or a lunch and an evening meal of five or six or some days, a couple of days a week, I just do the evening meal at 5 or 6. And like you, little bit of rice. It's almost, in terms of phenomenon or philosophically, it's almost a dessert. I see it that carb and fat mix at the end of the meal with a bit of potato or a bit of rice on the back end of the meal is a very pleasurable way to finish your meal while staying low carb. And because of the restricted window, you're kind of cycling in and out of ketosis to. You know, a little bit of autophagy going on there and no overfeeding really. So, it sounds like a good way to go.
	01:26:27	So on that note, we'll probably wrap it up. Any last thoughts because we're going to be meeting up again for sure, so we don't have to go over everything here.
Gabor	01:26:34	Yeah, just that, what you should really avoid is high glycemic variability and of course, high continuous hyperglycemia. It was shown, I think it was a study published in Science, that hyperglycemia, elevated blood sugar is the ultimate factor that can [Inaudible 01:27:01] your gut barrier function. So if you're really looking for increased intestinal permeability or a leaky gut, then just play around with your blood glucose at the very high levels all day long and you will get it for sure.

Ivor	01:27:20	You will get your just desserts.
Gabor	01:27:23	Yeah.
Ivor	01:27:24	Okay. It's a great stuff, Gabor. We catch up next time. And just to everyone out there, I hope you enjoy the podcast. Gabor, as usual is a font of knowledge, deep research and information. We are finding that search engines nowadays and other kind of web based algorithms don't particularly favor alternative health or alternative hypothesis. So just ask you again, if you could go to fatemperor.com, subscribe down the bottom of the homepage, that'd be really great to stay up to date with new releases. And also ihda.ie. That's Irish Heart Disease Awareness, ihda.ie that makes this podcast possible. If you could go on there and share with family and friends for awareness of heart disease prevention, that'll really help us too. So thanks a lot.
Gabor	01:28:15	Yeah, no problem. One more thing as Steve Jobs used to say, it reminded me of Google, I just tried out a alternative search engine. It's called DuckDuckGo. It works exactly the same way as Google Search used to work. So if you just want to get back your old Google without this kind of filtering alternative health sites and screwing with the results, then there are alternative options and it works just as well as Google Search used to work.
Ivor	01:28:55	Yeah. Sadly, that may be the way to go now because I mean, it's not censorship but search results should be thrown out as they come. It's a utility. It shouldn't be biasing. It sounds like there's other alternatives. Yeah, so maybe start using. And also, as I mentioned, by subscribing to email list, you're really staying connected to the people you want to hear from and you can bypass some of this bias.
	01:29:23	Okay, great stuff. Thanks, Gabor. We catch you next time.
Gabor	01:29:26	Yeah, thanks a lot for inviting again.
Ivor	01:29:29	Not at all. Great stuff. Bye now.
Gabor	01:29:32	Bye, bye.