Gabor:	00:29	And then if I understand the kind of the historical background, the healthy physiology, then I may be able to address how it goes wrong. But without having a very good picture of the mentioned two factors, I think it's impossible to get a get a good understanding of what goes wrong. And then you can come up with crazy things like bombarding your blood vessels with particles which have been there for millennia. And then suddenly these are harmful.
Ivor:	01:03	I agree. And I the mechanistic science and the evolutionary science and origins, and the healthy state of the body, healthy physiology, what that works like and all of these things are crucial when you're looking at a new problem to see, you know what it's mechanisms are. But you're right, there's a simplistic view taken that millions of years old, exotic, crucial, say lipoproteins are now suddenly the main cause for a problem that only started really in the 19th and 20th century. So there's an absurdity there.
	01:41	We'll come back again, I want to come back to the personal fat threshold. But just because we're on the topic now of cardiovascular disease, and to continue our point, I recently and based from research, like to look at the primary problematic things to lead to atherosclerosis and the inflammatory issues in your arterial wall, and I tried to keep in mind all of the technology and all my research, like you do. But there is the number of particles being higher and will promote more of the problem. That's the suggestion. And I think that's okay. Because if you have a fundamental problem with your system, and you pile more and more lipoprotein particles, it could exacerbate it. I'd no real problem with that.
	02:26	So that's one thing but then I have five layers after that and there's probably 10 big ones, but I've got five. One is the place of oxidized LDL, what place that takes part off. So, to what extent the particles oxidized and desialylated I think it's called in your plasma, trigger the arterial wall inflammation rather than native on damaged, and if oxidized had a big part in this, you would now be looking not at the particles but what oxidizes them for root cause. You know, smoking and a million other things. The second then is the glycocalyx, which is the barrier layer on the inside wall of the artery and those fantastic science around that, that it can manage the contact between LDL lipoproteins on the wall, then you have the endothelial cells, and they can transfer LDL across themselves through an evolutionary design

system – to your point, that was all made for the right reasons we have to trust – and you can leak to your leaky point. There's evidence for leaking of LDL between the endothelial cells, and that is greatly accelerated by things that insult the endothelial cells and damage the barrier, right? So that science is all there.

03:45 And the third one is the tendency for the LDL to be trapped in the wall on proteoglycans, and that can be accelerated massively by diabetes and many other causes. And the final one is HDL efflux, and that if you do have lipoproteins excessively becoming engaged in the wall. We've got HDL, an absolute miracle working macro molecule that does a million things and stops oxidation. But it also takes out cholesterol as part of its job. And if it is healthy and can keep up, you won't ever get a problematic buildup one would say.

04:22 So, all of these and there's many more. I've listed now five key things as well as the number of particles. But people don't talk about the five crucial mechanistic things, and what your health level is and each of those. But the HDL ties us back to the lymph, because something new to me a few months ago was you began to read papers, as we discussed HDL functionality, and you began to connect HDL to the lymphatic system. I always thought of it as coming out with cholesterol from macrophage and simply getting into circulation and going back to the liver. So maybe talk a little on that.

Gabor: 05:00 If you allow me to step back a little bit.

Ivor: 05:04 Yeah.

Gabor: 05:05 And you said that you have these five or 10 things which you can list. But is there one thing which can explain all the others? Is there a single one which can which is able to explain all the others? And then I will come back to this HDL stuff very soon, but I have a one paper for today in front of me, and it's released. So, everybody will love it because… this is just a list of symptoms.

05:37 When I when I asked questions, usually the two questions I ask are why? This is one, and the other one is, is that a normal physiological state which can explain all the observations? And then the problem is eventually the chronic activation of this physiological state, which was intended to be acute, I mean temporary. So, if we have a temporary state, which can explain all the observations and the problem is not this temporary state But the real problem is rather that this temporary state is being maintained on and on and on. So, medical people call it, it

becomes chronic, then we face a very different problem, because we have only one problem really. So, I will tell you and the paper is called something like "Effects of Infection and Inflammation on Lipid and Lipoprotein Metabolism."

06:51 So, what does infection inflammation do to your lipoprotein metabolism. It's a list. "Infection and inflammation induce the acute phase response." This is an immune response mainly described by the innate, so called "innate immune system." And then secondary phases like the liver response and all the other tissues response and the inflammation builds up. It happens every time you get a virus or bacterial infection or whatever. So what happens with your lipoproteins, with your insulin - when this happens?

07:30 Plasma triglyceride levels increase from increased VLDL secretion. As a result of increased adipose tissue lipolysis. increased hepatic DNL - de novo lipogenesis which means liver fatty acid synthesis, so you have DNL and fats now increasing in your liver - all the lipid circulation goes up and then suppression of fatty acid oxidation.

07:58 With more severe infection via the clearance also decreases, and then secondary to decreased lipoprotein lipase and apolipoprotein E in VLDL's. And then what else happens? You develop insulin resistance and hyperinsulinemia and also lipoproteins become enriched in ceramide, Glucosyl, Ceramide and Sphingomyelin. These are so called "bad lipid" things which are connected to inflammation. But why do these go up? Because they need to support your immune system. This is perfectly normal. This is a normal physiologic state when you respond to an infection for example.

08:46 And then these lipids are taken up from your lipoproteins, and then these lipoproteins becomes small and dense - because your immune system empties then because it's needed for quick proliferation, quick dividing of the immune cells and very weak and high level of metabolism of the immune cells, and then all the other stuff. HDL becomes a pro-inflammatory molecule – small and dense and pro inflammatory molecule. Oh my God, the HDL, the good cholesterol becomes inflammatory! So these guys just describe what happens in an **acute** state. But at the end of the abstract, they have a small little comment which is also very insightful***. "However, if prolonged, these changes in the structure and function of lipoproteins will contribute to atherogenesis."***

09:44 So there you go. You have one single mechanism, which basically explains everything you observed. You have insulin resistance and hyperinsulinemia, you have small dense particles of LDL, small, dense and inflammatory HDL particles. Basically, everything is there and it's one normal physiological state, which is intended to be temporary – acute - and not chronic. But what happens I believe what happens is that this state doesn't resolve. So you have this inflammation going on and on and on and then your system is overwhelmed.

10:25 And then coming back to the lipoprotein dynamics, LDL and HDL - you now have these higher LDL particle numbers with small dense LDL, and these are more prone to oxidation. Because your immune system spits out the reactive oxidant species (ROS) to get rid of the inflammation. They get rid of the intruders. They have their tools for fighting i.e. releasing the reactive oxidant species. So this state is also associated with higher oxidative stress, which can explain (it). Then you have the Omega-6s, which are more prone to oxidation. All the PUFA's, all the polyunsaturated fatty acids are more vulnerable. So, these are more easily oxidatized and then it exacerbates, all the other stuff… OK, coming back to the LDL & HDL. So, you have these increased LDL, flux into the arteries.

11:33 And then I think actually there is another point which I have to make is that it's perfectly normal that you have these macrophages, the molecules within your artery of all because these are basically everywhere. Some kind of immune cells are everywhere, and the role of these cells is to take up all the debris again. There is some debris like oxidized LDL which the Liver LDL receptors won't recognize. So if your liver cannot take it up, what takes it up? It's your macrophages in your circulatory system. It's perfectly normal. So, when you are healthy, you have *some* oxidized LDL coming in, this is taken up by macrophages, they become form cells, they release the cholesterol into the lymphatics, or it is transferred into the into the Apo-A1 molecule and the new HDL is formed - and then all the cholesterol and some other stuff is transferred into this new HDL particle, and then it goes into the lymphatics and goes into your vein, under your clavicles and then it joins the whole blood circulatory system.

12:57 So this is how it works. But what happens when you have an inflammation? You have an increased influx. So more and more oxidized LDL is coming which is not taken up by the liver because the receptors don't recognize it. And then you have increased number of macrophages and they try to try to gobble up all the stuff, and as usual, push it to towards the HDL return

system. And the problem is that when you have an inflammation, your lymphatics is slow down, because it's also a root for the immune system, and the immune system needs time to work on the problem. So the lymphatic circulation, the flow of the lymph slows down - and then your HDL flow slows down together with it.

13:50 I think it's a simple balance of influx and efflux to the arterial wall. You have an increased influx delivery and the decrease remover so then it starts building up. And I think it's okay, you can observe it every time you get a virus infection. You are in bed for four days for example, and if you if you take a sample from a from a blood vessel, from an artery, before and after, you will see these lipid-rich plaques building up in your arteries and these infections are closely associated with risk. These represent high risk of cardio events. So myocardial infarction and then these kind of things. After an infection, you are, I don't know double or triple the chance of developing a myocardial infarction. Why? Then that's the reason.

14:46 And the problem is when this kind of inflammatory response becomes unresolved and it goes on and goes on, and overwhelms the system which has evolved to deal with certain level of deposition and removal - but not that continuous level of deposition and removal. That's my current view of it.

Ivor: 15:08 I think that's very well summarized, and I resonate to say the least. And now it is basically an infectious agent triggering an immune response and associated inflammation, and largely mechanistically can explain all of the aspects of cholesterol, lipoprotein involvement with atherosclerosis. And also, you've got the associational data that when you have the infections, yeah, there's huge hazard ratios for events, for instability of plaque. Basically, it kind of explains largely everything with no major discord or conflicting fact. So from my world of root cause, I would say - that could be presented to management as a comprehensive and solid root cause presentation and no one could really “call foul” with any contradictory fact that would completely knock it down.You know, as Popper Karl Popper said, the great Karl Popper, you know, a single negative piece of data against your theory has huge power much more than the thousand positive pieces. But for this one, everything lines up.

16:20 I also have begun quite some time back and with discussions with you and others, begun to view the circulatory system, the vasculature as an organ that part of its role is to mop up problematic things in the blood system. And it makes sense when you think about it, because the liver is sent problematic

components to deal with. Fine, the Liver is the great clearing house. But you've got this enormous surface area of the vasculature that's in contact with the blood and through evolution would be perfectly placed to carry out functions to stabilize or help put blood.

17:02 Just take one example there. Like you say, you have oxidized lipoproteins in the blood. The Liver does not recognize them so well and they will become toxic and they are going to cause damage etc. So the blood vessels have an endothelium which has a LOX-1 receptor. And I've got some great papers on this. And the LOX-1 is linked to every single mechanistic step of atherosclerosis. But what the LOX-1 receptor does in the artery is it takes oxidized LDL – not native – it takes oxidized LDL out of the circulation and brings it into the arterial wall where it goes through the process of being dealt with by macrophage etc. And if it's acute, (this problem) - low level and your HDL is functional, because you don't have a major problem, say you only have an infection or you only have a problem for a few days, what goes in will be easily taken out and there will be no significant implication. But if you have a chronic problem driving oxidized LDL – and in the laboratory, this has been demonstrated that oxidized LDL in the plasma - damages endothelial cells and causes a huge amount of destruction. But it if you only have a moderate amount of oxLDL, like an evolutionary human, you will never develop the extreme atherosclerosis that leads to events. Your HDL is working, you're not pushing in a huge amount of this, and it won't happen. It's like you say it's the chronic insult involving the immune system and the damage to lipoproteins. That's the big game.

18:41 And, another point is that it's not just infections, because you've picked a great “case example” of an infection that can explain nearly everything in the process. But there's also chronic inflammatory conditions like lupus - there's a hugely increased rate of atherosclerosis with that. There's also smoking, which causes immune reaction and damages all the lipoproteins. There's also sugar and SAD diets and high carb, you know, bad diets, we know cause a storm of damage throughout the …

Gabor: 19:20 And Insulin resistance and hyperinsulinemia which can also be explained by a chronic inflammatory reaction.

Ivor: 19:28 Exactly. So often people say, “Oh, don't blame the inflammation, because that's a natural response.” But the point is that the inflammation is a natural response - acutely. But if you for push it for a sustained period, myriad causes of atherosclerosis that we know about, all align with this view of

		the lipoproteins involvement. And to come back to what we said at the start, so I would always acknowledge - if you have way more lipoproteins to be damaged and to become entrained in this system, this dysfunctional system - by all means they are part of the process. And if you reduce the number, by all means, if you take the number out artificially, you may mitigate or reduce the rate of the process. And that's all fine. But it doesn't take from the fact that the real root causes by an engineering standard are not really the evolutionary molecules; they are everything else we talked about - and more.
Gabor:	20:33	Yeah, that's a crazy idea, that the particles are inherently "toxic". I think that generally having more particles shouldn't be a problem. And I think you showed it several times that people with familiar hypercholesterolemia or whatever it's called, they can have higher, much higher levels of circulating LDL particles and LDL cholesterol, but it's not necessarily a "death sentence", I mean, if this inflammatory response is not chronically behind, then the system can still deal with that higher number because the balance can be maintained in the arterial wall. And then you can again explain that if you have a functional HDL and non-inflammatory HDL molecule, and it is transported into the lymphatics and lymphatic flow is normal, then it's just a perfect merry-go-round and it works. It's not a "death sentence" at all.
Ivor:	21:40	Yeah. And just on the HDL functionality, and I often bring this up because it does annoy me somewhat that we see a lot of press about how HDL is not so important, and it's not causal, and it's only a correlator - and lowering it or increasing it doesn't help. But that's because in fairness, the drugs to increase HDL achieved a paper increase, you know, in the numbers - but of course, they didn't help in any way with this system we're talking about. They didn't improve the functionality. If anything, they might have damaged the functionality as they forced HDL up.
Gabor:	22:18	Anyways, it's just a proxy. I mean, what you should, based on this hypothesis, what you should be able to measure is the actual efflux, the loading of the HDL, on the other side of the arterial wall into the lymphatics and the normal function of HDL going and returning to the blood circulation through the lymphatics. And everybody's measuring it in the blood. This is the first problem. By the time it reaches the blood, a lot of things happen to this HDL molecule. It's not just counting the function, it's not only by counting them, but we have this many and then we know how they function. That's how it works.

Ivor:	23:02	Yeah. And you know, the way I would look at it from the data and published science, if the HDL number is higher, it correlates with better outcomes. And this has been known for many decades. And if the HDL is higher, generally the LDL being high doesn't seem to matter or cause an issue. But all of this would make sense - not because HDL is causal through its number - but because a higher HDL generally correlates, generally correlates with a more functional HDL and better efflux, that's all.
Gabor:	23:39	With lower inflammation.
Ivor:	23:41	Yes, and with lower inflammation because a higher HDL correlates with lower insulin resistance, lower hyperinsulinemia, and it also correlates with many other good things like exercise - it goes up with exercise.
	23:53	The one exception is excessive alcohol can make it go up in a dysfunctional way. But there's always going to be exceptions. But the key thing is that the higher HDL tends to correlate with better HDL efflux and functionality. And that's why the higher simple number can tend to look like a good thing. But that study we talked about a couple of months ago we'll have to mention and I'll put it in the video and attach it of course, where they actually measured in the laboratory exactly what you said, the HDL lipoprotein efflux capability and capacity for individuals and gave that a number.
	24:33	Now, that's science. And it's the only example I've seen, and what they saw on a large cohort of people where they had them tracked over eight or nine years, and they had the data on who had events and who had no events. They got their blood, and for every person in a petri dish or in a test tube, they actually found out how good the HDL was at effluxing cholesterol from macrophage - the “functionality”. And they gave a number for the HDL efflux capacity for every person. And guess what? The HDL efflux capacity for a person indicated a 70% lower chance of a future heart event and a 90% lower chance of a future stroke.
	25:17	So if you measured the HDL for these people, and they did - it showed the usual “high HDL reading in the blood means 30% lower future chance”. But the efflux capacity properly measured showed 70% reduction and even up to 90% lower stroke rates. So this is the power of measuring the right thing. So, when anyone hears on the radio or on the television people questioning whether HDL is important, keep that in mind that

they're all talking around completely irrelevant points. And they're not talking about the actual science at all.

Gabor: 25:54 Understanding healthy physiology is absolutely mandatory. To be able to come up with the viable hypothesis, that's the basis. And yeah, I'm now there with regard to the immune system because I graduated mid-90's. And what you studied during the 90s was basically you have this immunity, mainly adaptive immunity, you have these as T cells, B cells and they remember all the patterns coming from the infectious agents - and how they need to react the next time. And then the innate immune system - which is the first level of defense, and which is the first level of surveillance system in the body – understanding of it was in its infancy.

26:52 So, basically we knew about, we have these macrophages and they can gobble up things. That was almost it. And the bigger evolution of the innate immune system started exactly in the mid-90's. I think it's still not fully implemented in biological education. So for example, if you are at the medical school right now, I'm not sure you get the level of knowledge, what we already have in the scientific journals. I think, usually it takes 20, 30 years to get through the system, so that it gets mainstream knowledge and that becomes a mainstream knowledge and from that, it will make it into textbooks and then people will better understand how the system works.

27:50 I've actually been back to the drawing table for a couple of months now. I've been studying innate immunity and immunometabolism. This is a crucial thing and this enables you to understand basically hyperinsulinemia and insulin resistance, how it develops. There is a there is a normal physiological state again where your immune system **initiates** hyperinsulinemia and insulin resistance so that they can get the stuff which hyperinsulinemia and insulin resistance results in, all the excess nutrients, all the extra lipids, what the immune system needs for activation.

28:39 So, this is a perfectly normal state again, becoming insulin resistant and even often hyperglycemic, developing high blood glucose during an infection. It's normal. So what is diabetes then?

Ivor: 28:56 Yeah, and that's exactly it. And I often say myself, that hyperinsulinemia / insulin resistance has a huge part in the causal pathways, but also all of the bad things that cause issues - overwhelmingly, most of them cause hyperinsulinemia and insulin resistance to occur as a reaction. So then those

measures of insulin function act as a gauge. So if you have low, very low insulin and very high insulin sensitivity or low insulin resistance, you may still have some issue going on and you might find it with some other blood markers. But you know you're way ahead of all the people around you in the modern world - just by having low insulin and great insulin sensitivity. It's one of the biggest metrics you can target. Whatever way you do it, you might have to give up certain foods that are causing an inflammatory response. You'd have to give up smoking, right? You'd have to eat a healthy nutrient dense diet. You may have to get sun exposure, a reasonable amount. There are many things you'll have to do to become very healthy and get longevity. But the insulin resistance / hyperinsulin gauge will give you a pretty good indication that what you're doing is likely to be pretty good, and bode well for the future.

30:15 I think of it as if you have an airplane and if you're flying a high tech airplane, you'll have many gauges. There are many blood tests, but you don't depend on one. Like, the particle number is one gauge that you need to watch. But there have been countless crashes were pilots have fixated on one gauge that's worrying them - and they've lost their spatial reasoning and they've lost the big picture. And in that aircraft analogy, I would also add that the "insulin gauge" is one of the most important gauges. Again, you don't just stare at it and ignore everything. But it is a really important one. And it's probably like the "attitude indicator" in the middle of the panel.

30:54 So, a bit a bit off there on analogies, but I agree totally. Now, I know we've both got hard stops coming up, but I really would like to come back and talk again on some of these topics because they're so rich in value. So, we're certainly going to do that. But if we could finish perhaps and wrap up with if a person has a very significant issue, likely autoimmune components, because that's very common, or obesity, diabetes, all of the modern chronic diseases - what would be the first steps in your diet and lifestyle in sequence that you would take to start dealing with a very significant issue? Right? So 1-2-3-4 kind of thing…

Gabor: 31:45 It comes back to my kind of unifying theory, of all maladies and diseases of civilization. I tend to believe that… the differences we see between people. I mean, if there is only one mechanism underlying or a bunch of very similar mechanisms underlying all these disease, modern diseases, then how come that we see so different manifestations of this? So, some people develop diabetes, some people develop obesity without diabetes, other people develop autoimmune diseases or allergies, or whatever.

And if we come back to the immune system, and chronic immune activation - and we do know that it's mostly not genetic. So there is a genetic component, there is an epigenetic component, there is a prenatal conditioning, intrauterine, so within the womb - and early infancy conditioning. That is some predisposition, of course. But this booming of all these metabolic diseases, and we can mention also cancer because it's linked to an impaired immune system, and so is extremely close. Then you start thinking about what are the environment of disrupting factors, if it's not genetic? And then there is an old study, not that old - a couple of years old - when I saw that there is this single cell organism called the Baker's Yeast, or Brewer's Yeast, whatever you like to call it. So the yeast is a single cell organism. And the biggest stressor on a yeast is guess what? **Feeding.**

33:50 So whenever it starts feeding, all the stress molecules within the single cell go up. All the stress factors go up and when it stops feeding? Stress decreases again. And there are of course other factors similarly converging upon the same pathways. And this is chronic stress, circadian disruption. So lack of sleep, poor quality sleep, wrong timing of sleeping and these kinds of things. But this is chronic stress basically because it activates the stress response. And then you have the smoking. We discussed smoking.

34:34 There are some others, like some people are really focused on getting blue light in the evening and they believe that it's the start of all problems. These are all contributors. And then these environmental chemicals, the exposure to air pollution, to phthalates, BPA, whatever, there are thousands of these and these add up. But the single biggest one, I still believe what you can easily manage is feeding. And feeding is what you eat, when you eat, your food order, when you don't eat. I mean some intermittent or even maintained fasting.

35:20 With these you can eliminate a huge chunk of stress from your body. Just adding the nutrients, the foods that you really need and causing the least possible amount of stress. With feeding - it always causes stress. Don't forget that whenever you feed your, all the interleukin 1β (IL-1β) and these inflammatory markers go up so it can easily be measured.

35:47 And then there are some smart tricks and I learned from you this Pareto Principle and making 25% of the effort, you can get to 75, 80% of the results. So if you are smart, you implement only what's really important. Leave out a few foodstuffs which are really inflammatory - endocrine or immune disrupting, like

		modern grains, most common grains which result, you remember my talk (in Prague), an imbalanced stimulation of the gut - and don't eat very frequently. Forget about even the world of snacking. Snacking is a modern word. Our ancestors had no clue about what snacking was.
	36:44	When you eat different kinds of foods you can include carbs - even some quick carbs maybe, depending on your current metabolic health. But then eat your salad first, proteins and fats second, and only quick-absorbing carbs last. Never eat something which is quick absorbing carbs and added fats without any structure and proteins. This is dangerous.
	37:17	Guess what, these items are mainly so called ultra-processed industrial foods. Most of the foods our ancestors recognized as food are okay still.
Ivor:	37:32	Yeah.
Gabor:	37:33	Not surprising!
Ivor:	37:35	It is not surprising in the end, but like we said earlier, it's very confusing if you're looking at an exploded jigsaw - but it's not that surprising when it's put together and then it almost becomes obvious, if you will. So a real-foods diet, depending on your insulin sensitivity or your level of tolerance, you know, simple carbs, be very careful with. You eat nutrient-dense fats and proteins, and ideally put the carbohydrate especially when easily digested - towards the end of the meal. And that's actually quite an interesting trick. Some people prefer to remove the quickly digested carbs for sure, but you can keep it at the end of the meal to help with metabolism and get all your nutrients. It's a whole-foods, lower-carb (certainly low high-glycemic carb, kind of diet - and eat maybe twice a day, and occasionally once a day is one of the ways I view this.
Gabor:	38:35	Yeah, absolutely. Coming back to the gut stimulation which is one of my favorite topics, you know, I dealt in great detail before with the imbalanced hormonal stimulation these ultra-processed foods can cause. And just when preparing for my talk last May, I recognized but of course, there was not enough time to include this one, that this imbalanced simulation is also one of the main reasons for seeing the impaired intestinal barrier function.

	39:18	So when you have lower upper-intestinal stimulation, and higher lower-intestinal stimulation, actually what you see that these lower-intestinal hormones, together with the surrounding immune cells create a very strong intestinal barrier function. So you don't have this leaky gut. This is exactly the same pathways, just different hormones I mean. Then I focused on insulin-releasing hormones, upper intestinal and lower intestinal, but the same so called "L-cells", releasing GLP-1 and releasing GLP-2 - which is kind of the guardian of the intestinal barrier function. And guess what, it is stimulated by the same mechanism, the very same foods, so it's released when the cell is correctly stimulated. But if the cell is not stimulated because you eat donuts (because you absorb everything in your upper small intestines) - then don't be surprised that you develop leaky gut.
	40:16	And then we can also come back to the… I mentioned the fat absorption - that long chain fatty acids are not absorbed to the portal circulation. So they don't go directly to your liver, but instead, they are taken up by the lymphatics in the so-called "chylomicrons". These are lipoprotein molecules, the largest of all we because they are loaded with triglycerides, the fat that you absorbed. And why? I mean, again our question comes, "Why are these not absorbed to the portal circulation and go directly to the liver like other fats?"
Ivor:	41:00	Yeah! Well you know, there's an excellent few topics, and we did go through gap GIP, GLP-1 (not GLP-2) in Budapest last year.
Gabor:	41:09	That's a new addition (GLP-2)!
Ivor:	41:11	Excellent! And I've seen and I've been very busy but I've seen the "Lower Insulin" Facebook group which you run, and I've seen the papers coming and I've made a note. I'm going after you for those! So next time we meet, we'll go through the whole intestinal hormonal balance, how the foods affect it and all of this new science which explains and unifies - not just the disruption of your intestinal, the hormones that in one pathway lead to obesity and chronic disease - but now unifying that with the other, basically the other immune type pathways (GLP-2) and tie it all together for people. So no processed food, no refined carbs and sugars, no vegetable oils. And we might also next time talk a little bit around the plant-world foods that may be a big problem for some people - while not for others.
	42:02	Excellent. So hey, thanks a lot, Gabor and always a pleasure. There's no question about that.
Gabor:	42:09	Thanks for inviting again.

Ivor:	42:10	No problem. We'll catch up next week as well. Have a great day!
Gabor:	42:14	Yeah! Have a nice day. Bye.