# Discover Fasting Mimicking Diets For Cancer Therapy Transcript

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## Origins of the Fasting Mimicking Diet

Dr. Longo. It's such a pleasure and honor to have you on this segment of integrate of the. The Breakthroughs Cancer Summit. I mean, you you are a leader in this field in regards to the fasting mimicking diet.

I wanted you just to kind of lay the ground first, you know, why did you even get into this direction of fasting, mimicking diet? I mean, why why there everything else you could study?

Yeah. So we were many years ago. 20 years ago, we were experts in, in protecting abnormal cells against all kinds of toxins. And because we were working on aging.

And so, looking at chemotherapy, it seemed to me that was probably one of the most toxic intervention that a human being will ever get in his or her life.

And so that was the beginning. What really fasting can we protect the cancer patients versus mice? And then Bayesian and and not the cancer cells against chemotherapy.

there was the idea. Then we started at the first trial, a clinical trial at Norris Cancer Center at USC. And it was a disaster because, patients did not want to do water, only fasting.

And the oncologists were very worried about water only, fasting and, yeah. So then we went back to the National Cancer Institute, the National Institute on Aging, and, and they funded, they co-funded the development of fasting, mimicking diet.

So basically making diet actually started with, with cancer patients. And that's exciting. And so, and we, we we talked a little bit in the past in regards to, you know, how it protects the healthy cells during chemo.

And then opens up the chemo at the cancer cells for, for chemo during fast American diet. But you've gone so much, so much further beyond that in the research.

And there are a lot of clinical trials that are out there right now in regards to how fasting, mimicking diet can then be combined with some of these specific therapies that are out there.

And do you mind sharing a little bit where we're at now with the understanding of fasting, mimicking diet and cancer? Yes. So I think that, now we in many, many laboratories, done studies in mice and lots of cancers and the fasting making that keeps working on, almost every cancer that has been tested so far in combination with the standard of care.

Right? So, so immunotherapy, chemotherapy kinase inhibitor radiotherapy, hormone therapy and so, so that's already been very successful in the mice. And then we can talk about, clinical trials, next.

But the, I think a lot of these studies or most of the studies show slowing down of the to more progression, major slowdown, much better than chemotherapy, much better in mice, much better than immunotherapy, even.

Right. So so if you combine fasting, making diet and immunotherapy it works very well. And in mice the immunotherapy doesn't work well at all. And so but but then what about regressing causing tumor regression.

And for that we move to what we call RNA seq so that we look at our, a cancer cell rewires, and then we, target these escape pathways.

## How FMD Protects Healthy Cells During Cancer Therapy

So, for example, in, in, in a paper that, we published a few years ago, Julia, in my lab, looked at triple negative breast cancer and and applied the fasting mimicking diet to triple negative breast cancer in mice.

And then the the the cancer clearly rewires and turns on par itrillioneads. Turns out it turns on tau. It turns on Cdk4 six. And so now she target it with drugs that are already widely used in cancer therapy and and the cancer.

Now, instead of growing more slowly, a reverses course all the way to to zero. so so now we've done it over and over and over with different models. And so we're very excited about the possibility of in a patient identifying quickly, let's say within a couple of days the escape pathways and target them with FMD which is a wild card plus standard of care drugs that are relatively nontoxic, or nontoxic.

So, so help me understand so you have the, like, triple negative. It's hard then to have a target type of chemo or target kind of therapy. You know, because you don't have, you know, you don't have the Her2, so you don't have their septum.

you don't have, you know, any positive. So, so the FMD then kind of rewires as you do that PhD, it rewires escape mechanisms and all the cancer cells and that it opens up targets that normally was not there prior to FMD.

Yes. Not only opens up, but makes it very easy to to know what they are right quickly. So within a few hours we can tell, what our cancer as rewired. Right.

And that seems to be very effective in representing all the cancer cells because we don't see then much of a survival once we get all of them or the major ones.

So the cancer goes down and the stays down, right? So, yeah. So now we're still at the beginning of this, but it's working very, very well. And so now we're hoping that we can isolate cancer cells from the patient's, circulating cancer cells.

And then we can, target, quickly learn how they're surviving. And then so theoretically, within a day, we can adjust the, the aim and, and use FMD, plus keep changing the drugs until the cancer disappears.

Now, it's a little bit of science fiction right now. And I understand that, but, yeah, it's working well, so I don't want to. Yeah. Make it sound like it's conclusive for humans.

It's very, very promising. And, now we we need to do the trials. Right. But, for example, the needy group at the National Cancer Institute in Italy, combined FMD with, chemotherapy, for triple negative breast cancer.

And then after four years, the overall survival was about two and a half times higher in the FMD. Plus chemo versus chemo alone. Right. So so I think now this is four year overall survival.

we're starting to see and I think they're going to publish additional paper supporting with more patients supporting the initial results. So so it's good news already for the last sophisticated interventional chemo plus FMD.

So now we're this is why we're optimistic about the more sophisticated intervention. What if you get rid of the chemo. What if we now, use this multiple, relatively low toxicity drugs and with the FMD.

yeah. So we're optimistic, but we got to do the trials. So when you say less toxic, I mean, of course, you have the field of repurposed drugs. You have the field of of natural, you know, interventions like vitamin C, curcumin, an artisan aid, you know, repurpose drugs, you know, talk about metformin, you know, tours of estate and all these kind of things.

So is is that what you're talking about in regards to less toxic drugs and, and, and learning which escape mechanism to, to target.

## Escape Pathways and Rewiring in Cancer Cells

You know, once you, you know, once a cancer cell rewire or and you kind of measure with the circulating tumor cells and see where the genetic instabilities are so to say.

Yeah. So I think there are two levels. One is the relatively low toxicity, let's say compared to radiotherapy or chemotherapy. Meaning now we if we target we have some of these new generation PI3 kinase inhibitors right there relatively.

I mean whereas the people say but maybe more toxic. But some of these new generation kinase inhibitor may be less toxic. Right. So that's number one because now we have a lot of options, right?

A lot of options. And so we can target almost any escape pathways that we see. And that's what we're doing. It's working very well in the mouse studies.

then we we published a few years ago FMD plus vitamin C, and now we publish FMD plus vitamin C plus chemo. And that stopped even very aggressive cancers.

but now we're saying, okay, can we do FMD plus vitamin C? And as you pointed out, we remove the chemo. And now we introduce other completely nontoxic drugs.

Let's say, as you pointed out, metformin. Rapamycin. We have to see. Right. But but some of these of course are now informing. Rapamycin are both they're both in trials or about to get into, clinical testing to slow down aging. Right.

So, that's how the, our nontoxic they're viewed. Now, I don't know if my colleagues, Nir Barzilai and others are correct about that, but certainly the view is that you could get lifelong treatment with some of these drugs, in fact, to slow down aging.

Right. So this one makes it very exciting. I think, for the cancer field, could we actually use 3 or 4 anti-aging drugs to then target the cancer cells specifically while helping the normal cells?

You know? Yeah. Because, I mean, you have the issue of, you know, dealing with excessive senescent cells, you know, excessive immuno senescence and all all these type of things where you're bringing in rapamycin, bringing in metformin, and then also, you know, autophagy with, with FMD.

So it almost seems like by addressing the the aging process in the body, you're also then addressing cancer at the same time. Yeah, I think there's much more at the cancer prevention level.

Yeah. So what you said is absolutely true for cancer prevention in this case. I think this is much more powerful. because these basically you could probably and at some point we may do that like a mathematical model that this shows that it's just a matter of time right before, we can differentially target normal cells in cancer cells just because we already know that often with especially during fasting conditions, they go in the exact opposite direction, right?

So so we push them to make a decision. What are you are you are normal cells. Are you going to go to standby mode or are you a cancer cell and just going to ignore by definition right of the cancers?

Are you just going to ignore integral signals? And that's what makes it very easy then to target the escape pathways. So yeah. So I think this is it's a truly it's a technology that just, quickly identifies, escapes and targets them and, and try it progressively will less and less toxic intervention.

And and you mentioned that you know as quickly to find these escape escape mechanisms. But it sounded almost like, you know, when you bring these, safe drugs on board that sometimes escape mechanisms and can, can change, and you have to shift again, this is that is that what I'm hearing, or do you have to kind of adjust a little bit through that process, or is it just kind of a one way track.

And, and we're going to hit the target. Well, it looks like a lot of the cancers we look at, after we get to escape

number 2 or 3, they're done. Right.

So over and over and over in many cancer types. So, but but even if they escape the blocking of the escape pathways, we could go back and say, okay, how did you do that?

Right. So it and then it becomes progressively more obvious because there's probably only one left. so now there's one way that you may be able to get away from three drugs plus the FMD, plus the wild cat.

Right. And then, so the expectation and what we're seeing now, something really lights up and, and now we can go after that. So, so explain to me a little bit and for the audience, well, what does an escape mechanism look like?

And what would be an example of an escape mechanism, an example of and how to address that mechanism. Yeah. So an England example may be for example or signaling.

Right. So Tor normally during fasting goes down, but in lots of cancer cells it goes up. So the cancer is trying to do protein synthesis is using the Tor pathway to make more proteins, more amino acids.

But it can't do that. Right. So so well again up to a certain point and that's how it survives. Right. But if you target that, now it's in trouble. Right. But that's usually not enough. Right.

So if you do fasting plus Tor so for example, to reverse triple negative breast cancer, Julia had to do three drugs act inhibitor Pi three kinase inhibitor and or and CDK four six or Tor inhibitor plus FMD and now I started to work in another case with er positive, breast cancer.

So the hormone therapy now the fasting mimicking diet reduce insulin IGF one and leptin right.

## Combining FMD with Targeted Drugs and Immunotherapy

Plus Cdk4 six inhibitor plus E are signaling estrogen receptor signaling. And that is a quintuple. So five different block blockades. Right. And and then and only then you see that the two more coming down and and the mice are fine for a long time.

Right. So no toxicity to the mice. And they slowly they regress. And until you see a cancer free survival. So now we've done it in people, both triple negative.

And also you are positive and you are positive. We are able to do it for years. And we're starting to see women do very well and and surviving a lot longer than, than expected.

But, you know, there are there are no formal, formal, clinical trial any are positive. And so, so now we're encouraging, of course, lots of, cancer centers to, to take it up and test it out. You and, and these kind of checkpoint inhibitors, you know, that you're bringing in.

Is that like, Keytruda? Opdivo. You know, that that's very safe or. Well, what kind of what what do you use? What what are some of the common ones that that you look at?

Yeah. Now, so, so far we haven't talked about immunity immunotherapy. But we've also combined the FMD with immunotherapy. And so yes PD1, Pd-l1, inhibitors and and those are and those are fairly effective.

in slowing down the cancer. We're not really seeing, you know, with that combination cancer free survival. That's why I, I like more because I think in a sense, I mean, people and of course immunotherapy is great, but, you're still sort of hoping that the immune system does.

Does that all for you? Right. That is the hope. Right. Whereas with the escape pathways targeting this, that I hope is, is truly, learning I you survive and targeted and and yeah.

So I'm more optimistic, believe it or not, about the this combination therapy, with the non immunotherapy intervention, although we'll see we are also doing the combination immunotherapy and that's working very well.

But thus far we struggle to see any cancer free survival in any mouse model with immunotherapy plus plus it's yeah. So so I don't think it's going to be that easy to get a lot of, cancers, in in check with that combination.

But maybe if we do more. So for example, at USC, we're starting now a fast even begin die plus vitamin C plus Ctla four plus PD1 or one inhibitor. yeah.

So then now with the quadruple intervention, this is in patients, right? yeah. Will we see, remission. And now the Vernier lab, showed a couple of years ago they did a 100 patient trial, and then the patient trial did show it.

The patients that got fasting mimicking that, there was clearly much more immune infiltration into the tumors, all kinds of tumors, all kinds of, combination.

And, but then they had five cases which they felt it was very it was so unusual that they had five, stage four, it was breast, colorectal, and pancreatic and yeah.

So breast, colorectal and pancreatic cancer in lung and one lung cancer. So stage four and all of them by combining immunotherapy plus FMD or chemotherapy and indeed all of them went into remission right.

Long term remission. And they felt it was so unusual that to see that they published the paper, describing these five cases. So okay. Of course, it's just an anecdote.

It's five cases, but enough for a group of oncologists to say, that's really strange, right? Maybe we could see 1 or 2 cases, maybe by chance, stage four, you know, ending up, in, in remission.

But long term remission for five cases within the same trial. All stage for all in trouble. yeah. So so I think that also shows that the potential now is just a question of, how do we get a lot more patients and how do we prove it in, in, randomized clinical trials?

Well, I mean, not all those those four cases are abnormalities. I mean, all those four, because, I mean, regular standard of care is very it's not very successful in regards to addressing these type of cancers.

So, so I mean, obviously that that that's a huge observation. And you're absolutely right. I mean, small small caseload doesn't really prove anything, but it really gives you no pause to kind of see the potential of what can be.

Yes, yes. What if right. What if I stage four lung cancer patient now can be can go into remission by combining immunotherapy with FMD as they did in this one case.

And so what what does in these clinical trials. What does the FMD look like? I mean because I know you have the five day, you know and prolong pact. You know that that you know, I use for patients and and other people use for patients.

You know what in a in a trial, what does that look like. And the trial looks at a lot it has a lot less calories, about 600 calories, per day and last five days.

And it's got a lot of options there. Why is that? Meaning that you have a soup, but then you have two backup soups and then a broad. And so we we did it because, often cancer patients develop food aversion and they start disliking any, any one or several of the ingredients that they, that they have to consume together with whatever therapy they're receiving.

So, yeah. So now we've learned and, and we, we're, we're making it easier or much easier for patients because there's a lot of flexibility in the things they can do during those five days.

and so now we're getting, a pretty high compliance in the clinical trials. We just finished another trial with 120 patients at USC. We're analyzing the data now, but in most of the trials, with one exception, the compliance was was, high.

That's incredible. So the and and it's, you know, the five days a month or how what what do we what are we looking at in regards to, you know, to, to achieve that state, to really, you know, to open up these escape mechanisms so that you can then address.

I, I think it depends on the therapy. Right. So if it was chemotherapy, let's say three weeks, that was the trial, a USC with breast cancer and prostate, that was done together with the bolus chemotherapy three days before, one after and a big warming year.

But we chemotherapy because, as we had suspected for a long, long time, never combined chemo. We're feeding or radiation. We're feeding what doesn't meet.

There's a paper. It was a mouse paper. But, you know, a scary one, I think, looking at intermittent fasting. So one day and one day off and in mice and then give doxorubicin, I think it was in showing increased, cardiac cardiotoxicity.

## Clinical Trial Results and Patient Protocols

Right. Which is exactly what we expect because now you have a high dose of the chemotherapy in the moment where the mouse recedes. And we suspect the same problem you will see in patients.

So I'm that's a warning to people that think fasting doesn't matter how you do it, it's all going to be, working. And this goal is going to be effective.

It makes a big difference how you do it. And I think in this case it should be very respected. All of the FDA type trials, right? So many trials in a standardized way.

And then colleges in the physicians out there say, okay, this was done in the trial. It worked. It was safe. I'm just going to follow exactly that rather than, oh, it's fasting.

Let me just figure out the way that I want to do it. Yeah, yeah, because that's an important point, because a lot of a lot of patients out there, they do the intermittent fasting and, you know, they do ketogenic intermittent fasting with it.

And so you're saying that around the chemo that it can actually be more detrimental than for for the heart muscle, you know, to do that? if you don't follow kind of these strict guidelines in regard to how to do it with the fasting mimicking diets, there's a big difference between the two.

Yeah. Big difference. The opposite. Right. So if you do three days of fasting or fasting mimicking diet, then you give the chemo, then you wait 24 to 48 hours and then you repeat the protein high protection against doxorubicin.

Really remarkable to the point where sometimes we see all the live mice all dead. So all life with the fasting making that all that with the regular that.

Now if you do intermittent fasting one day you the mice eat when they don't or 16 hours or whatever. now you see the opposite because now you have high levels of chemotherapy when mice are re fat.

So now IGF one, insulin, glucose, they are going up very rapidly. They're promoting cellular proliferation and cellular activation, including probably the cardiomyocytes.

And now that combination is toxic or highly toxic. You so so you have people then obviously that they're concerned about tech sexy. and so if they do kind of a shorter version where 72 hours is, is that not advisable?

You know, during the process, you, you it's the five five day set. That's really what you got to do. Well, I mean, there are exclusion criteria, right.

So and it also depends on this stage. Right. So somebody stage one and they've lost a lot of weight probably not a good idea to do the fasting making diet.

Right. and body somebody. Stage four. Even though they lost a lot of weight, the oncologist and the doctors have to make a decision. Right. you know, what are the options?

Are there viable option? And then, yeah, there's going to be a compassionate use type of situation. Right. So it could still work very well. Even somebody lost a lot of weight.

and, and make a big difference. And then they have, they have all the time in the world to regain their weight. but, yeah, in general, I think that if somebody has lost a lot of weight and they, they, they have something else that we know, but 98% chance of overall survival.

I wouldn't, I wouldn't, think even consider the fasting, meaning diet. but, if somebody has 25%, chance of surviving five years, then, I think that the risk, it could be higher, even considering the weight.

And now a lot of patients, of course, don't lose weight. hormone therapy, they gain weight. The important hormone, in in prostate and in breast cancer.

But but for those gastrointestinal excess, other type of cancers for which there is a lot of weight loss, then I think it's a matter of, identify personalizing. Right.

So some patients can do it and should do it. And some patients probably are better off, and not do it. Now, I'll keep in mind that we don't see any lean body mass loss even after many cycles.

So in the trial, for example, we we breast cancer women in Italy, we did not see any lean body mass loss, only fat loss even after seven eight cycles of the fasting.

Meaning that. So I think for the majority of people, potentially great majority of people, it's not a issue because once they're feed, they'll regain back whatever muscle mass they've lost.

But in some cases them that may not be the case. And and so the doesn't need to be that the five day or the 72 hour still have a benefit. No. Not only has to be five days has to be five days.

Following that, what is being clinically tested. and so say four days before the chemo, then 24 hours after the chemo, treatment or same thing. Now for the for the immunotherapy trials.

Right. So 3 to 4 days before immunotherapy and then the immunotherapy and then it continues. But again each protocol I mean a lot of this has been published already either by by us or by vanity or by other other groups clinically.

So I think the oncologist should just look at the papers and see for that particular treatment, what was used. So for example, for the hormone therapy, which is a continuous therapy, this the FMD was done for five days, once a month, and just big in no particular time.

If patients were, let's say, fulvestrant and palbociclib or other Cdk4 six inhibitors. I mean, it's, it's it's huge. I mean, what what what you guys are presenting. Yeah.

To be able to turn these kind of cancers around. The triple negative. Yeah. And potentially pancreatic, you know, stage four lung, etc.. I mean that that's just tremendous to show you know what you can do and how you can manipulate the immune system.

And, you know, cancer wiring just using diet. Yes. Yes. And I think the diet is just, making the standard of care work a lot better right there. So I, I always say I'm never say I'm almost never cured anything with fasting.

Right. And, so cancer wise right now for all diseases work, but for cancer, it just slows it down. But when you combine it with the right standard of care.

So I think that's also nice because I think it's also respectful in all the all the decades of work that a lot of people have done, you know, with immunotherapy, chemotherapy and etc., etc..

Right. So, so those that that's the combination that works very well. And so that combination of course takes, you know, the 30 years of work in immunotherapy and now makes it better, maybe make turn something this slow down into something that now regresses.

and talk to me a little bit. I mean, we, we talked to, you know, briefly about, you know, because some people don't want to do chemo, they don't want to do immuno.

And, here you're talking about like, something simple as vitamin C, so, so what have you seen. You have this kind of therapy ptosis type of protocols where you, you know, you do vitamin C in artists and a, you know, to trigger oxidation of the iron that's within the cancer cells.

And so how does that combine then with the FMD and what happens. Yeah. So so if you give so they look into the lab and all this has shown and Linus Pauling had been talking about this for, you know, since 50 years ago.

## Vitamin C, Ferritin, and Nontoxic Combinations

And but what happens is and we tested these in lots of different cancer, lung cancer, colorectal cancer, pancreatic cancer. What happens if you give a lot of vitamin C?

The, the, there is increased toxicity and there is oxidative stress within the cancer cell. And there is that. Right. But then when we look at it molecularly, it turns out that the cancer cell is smart and it turns on the ferritin pathway.

Right. So it produces a lot of scavengers of iron. Right. And so now you get some toxicity, but you get a little toxicity because it is a ton of disparity.

And he is one and Faraday. But when you pass them when you do the first imaging that this there is a differential effect on ferritin. Meaning in the normal cells it increases variety.

And in the cancer cells it decreases. Right. So now you see just a major, major increase, sometimes tenfold, in the toxicity specific to cancer cells.

When you put the first imaging data together with vitamin C. So now the excitement doesn't even come from this because, you know, we're so far we haven't cured very much with vitamin C plus D.

But the excitement cancer escape pathways now also target it. And that's the next generation. I think we are one year away from describing this. And so.

So how do they escape from vitamin C and FMD and how can you can you target it with all their nontoxic drugs? Right. yeah. So that, I think we're very close to that, to that, showing that and yeah.

So that I think that's going to be, very exciting. because, because this introduces this possibility of, like an antibiotic for cancer. Right. So can you imagine going to the doctor and the doctor say, yeah, I take this cocktail for a month, let's see if this goes away.

Right. Yeah. So now, at least theoretically understood, it's not a reality. But theoretically, I think it's important to start seeing this possibility that, you know, you have the cocktail is completely nontoxic.

Take it for a month. Let's see if it goes away. If not, let's see what all the cancer survives and then change around. And I give you something and I see.

I have I mean is incredible. I mean because I mean these these are the kind of things that, you know, we've been kind of barking down the same or barking up the same tree for.

Yeah, yeah. Since the 1970s, almost in a way. Yeah. And in the medical field and there's been some kind of in additions there. But this just seems like all the research that has been done, like you're saying, people have been researching 30 plus years, is now really then we're able to use that and just take that to the next level where we're really seeing the effect.

Yeah. And I think that technology, right. We just did not have like this RNA seq, single cell RNA seq. It was science fiction, you know, even ten years ago.

Right. so now to be very rapidly being able to take a single circulating cell and, and, and, and tell us, educate us on what's the escape. What how is this cancer in this patient surviving, this, combination therapy?

yeah. So this technology is making a big, big difference. So is is the 30 years of research, but also the new technology that allows us to, and this is without even bringing in the AI.

Right? Then I can, I think, make it even easier. I think eventually, to to get to it, but that even without the I just a computational straightforward computational strategy, as we have shown a couple of years ago in, in, in our paper, that's enough to, to be very effective now.

Yeah. Of course understanding that it, it could take very little it could take a long time to get there for lots of cancers. So we'll see. We'll see what we try to go as fast as possible and see what happens. You and you mentioned earlier also you talked about that, that there's more with the fasting mimicking diet and, you know, some of these therapies that there are more kind of immune cells that were found within the the tumor environment, that tumor microenvironment.

So, so you almost it's like an immune system activation in that you bring in by bringing in the SMD along with some of these therapies. Yeah. So what we think is that for example with with most therapies, chemotherapy, radiation or even vitamin C, you now have the cancer cell, as you pointed out, making out of free radicals like iron.

You know, I drugs so radical superoxide hydrogen peroxide. And so we think that that's making the cancer cell much more visible to the immune system. Right.

So by creating all these antigens that we're maybe not as visible or not as a variety of antigens, right. So we haven't demonstrated this, but that all the data that we have is consistent with that.

And so now you have a lot of DNA damage, a lot of new possibility opportunities for the immune system to make a decision. That's not a normal Sal. I will attack it and I will attack all of the ones that have the same mutations as, presented, by antigen presenting cells as this one that I just attacked.

So it's almost like it's creating a, like an a vaccine effect in a way, you know, where you have more antigens that are being expressed and then the immune system can then trigger, you know, creation of antibodies or whatever immune system component that's needed to to address that.

Yeah. So now, for example, Moderna is is, generating vaccines, RNA based vaccines to, expand the level of these, antigens within the cancer cells and so make it much more visible to the immune system.

And, and so maybe the fasting making diet is, is doing some of that on its own by just, you know, within cell or cell autonomous generation of a lot of mutations and a lot of, a potential antigens.

So with this, then, I mean, so since we able then to kind of capture some of these mRNA and really kind of analyze

what that is and, you know, they really kind of fast track to them how to create vaccines based upon our on mRNA.

Yeah. So it almost seems like, you know, then we can create a more targeted vaccine. You know, quickly for that specific individual. And then using fasting mimicking diet to, to create more of these, these kind of die off material to, to be able to see more what would need to be done.

Yeah. So Moderna is already doing that. Right. So Moderna is already personalizing it.

## Future Directions for Personalized Cancer Treatment

So finding, what the mutations are in a particular cancer patients and then generating the vaccines specifically for that cancer patient. And so now the question is, what happens if you combine it with, with the fasting mimicking diet and, yeah.

So that's something that I hope we can convince, Moderna to, to test that. That's exciting. So where do you see kind of this, this research be, you know, that, like, in a year from now?

I mean, you're seeing the directions. I mean, where do you feel will will be in a in the year, two years from now in in regards to cancer therapies? I hope that within a year or so we can begin the first trial, proof of principle.

Right. So say 100 patient trial. Can we, this escape, pathway targeting, does it work, consistently in humans? And so we'll see. Right. So that's that's the first one.

And then, and then could we, in the patient for whom it doesn't work, is it easy enough to identify the escape pathways and target them? and or at least I mean, is, you know, the the trials are not don't go as fast as you like, but but at least if we have isolated cancer cells from patients that don't respond, and then we see a clear signature of escape, even though in the same trial we wouldn't be allowed to target that.

But we will have to wait for another trial. But I think if some patient responded and all those patient escape, but we clearly see a, how they're escaping, I think that would be a very, a very, exciting, success, at least initial success.

And how do you feel the, you know, the, the oncology community is responding to, to this type of science that that's coming forth? I mean, do you feel that they are open to it, or do you have to kind of push a little bit harder to for them to to see the the possibilities of this?

Yeah, I think that the science has always been the same way. And, and I think the immunotherapy, Allison, on Joy and others, found out many years ago when everybody said, immune immunotherapy.

What is that going to do. Right. So yeah. So you have to go through, 20 years of nobody taking it seriously and then, all the way to the clinic, you know, and, and, I was reminded in a meeting that, that the first immunotherapy trial, was viewed as a failure.

And, it wasn't until they went and looked at a subgroup that they realized is not a failure. it's working, but it's not working for everybody. Right. And, so that's how hard it is, to, I think, to convince the community so all the way to that this big trial, everybody had doubts that, it would not work.

And then, of course, started with immunotherapy with a subgroup and, and then slowly and moved to lots of uses. I think that we did not. What is the first thing making that in the same category is it last is it more.

Right. So in mice it certainly works better. I mean that's, that's interesting. Right. In mice, a lot of the mouse model that we do triple negative breast cancer, lung cancer, melanoma, it the FMD works better then and then either Ctla four Ox 40 and PDL one and PD one.

Right. So we try them. All right. So yeah of course. Does it mean anything in people. It works but in lots of cancers. But so I'm just saying that the fundamental theoretical level, at least in many my mouse model that we tried the, the

FMD, the first thing that works better, than the immunotherapy again, no, no claims about humans, but that's, interesting.

start if you think about that. Yeah. I mean, it's a it's a phenomenal observation. And as really intriguing them to see, as you're saying, when we bring into the clinical trials in humans and then obviously we, there are other factors we have to address.

And as we then kind of refine, you know, how how the, the therapies are being administered based upon what we're seeing. I mean, that that's exciting times ahead.

Yes. Yes, absolutely. Yeah. So I think, also so we follow, we have foundation clinics and we follow thousands of cancer patients. And I think it's also important, so that if I was a cancer patient, I would want, think that there's always an option, right?

There is always a possibility that. Hey, and this is why we really love this nontoxic great intervention, because we're thinking if we can get it within a year or two to the point where where we can say, okay, try right.

Compassionate use to all to the oncologist. If the ecologist goes for it, try. So that's what I really like it. You know, no matter what, you know, as we've seen for the stage for patients that you can say, okay, you know, not easy not not likely maybe.

But it could work. Right. And so I, I really like that that part. And I think if I were a cancer patient, I would want those options. however, you know, late they may come, but I would want that.

What if it is stage four? If the oncologist says doesn't look good at all, you know, that that could be, a, an option. Yeah. Yeah, yeah. Love it. Well, Dr. Longo, I so appreciate, you know, that you're driving this forward to kind of make safer options available.

You know, safer options that that are more powerful than the tools that we currently have. Thank you. Thank you so much for putting this on the map. Thank you, thank you.