What Dolphins Taught Us About Aging Transcript

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Introduction and Webinar Overview

Thank you. And can you see in the screen right? And can you see me? Yes. Okay. Excellent. Well, it's great. Thank you. Great to see everybody. And we'll go ahead and get going.

So the goal here as we have these HCP healthcare provider webinar series is that not to provide the same talk every time I come on. So I'll try. We need a little bit of redundancy on the front end just to catch anyone up who wasn't on the prior.

webinar, but I'll do my best at having like 80% of it be new information or diving into specific areas. And then if there are areas, what we're going to do today is it's kind of like a buffet of all the different things that C15 does from at a system level.

So red blood cells and liver and metabolism and heart. And so I'm going to walk through those in a relatively rapid manner, but just to give you an idea of kind of where at the sciences in these different places.

And if there's one that you really love, it's like, man, I would love a whole webinar just on this stuff, just on fatty liver disease or just on fructosis.

Make a note for us and that will help kind of help guide what we'll do in the future ones is we'll pick individual topics and then really do deep, deep dives on those.

Um, so with that, I will start moving. And then you just know to go ahead and write in the chat, um, questions that come along as we, uh, as I move along here.

All right. So we got to start with dolphins cause like we've got the dolphin card. So we're going to use it every time. So, you know, just as a reminder, this, uh, discovery as Danielle just shared, right.

All came, um, as me as a veterinary epidemiologist working for the Navy's Marine Mammal Program. and caring for a growing population of really geriatric dolphins that don't exist in the wild.

And so it was really thanks to taking care of these dolphins, the Navy having a 50 year archive right of blood samples of samples routinely taken from the dolphins as part of their routine care, having the foresight to archive that serum throughout their 30 to 50 year lifespan in freezers and then also have their tissues.

So as you can imagine, the power of this population paired with the fact that we were discovering that as dolphins got older, about one in three were developing aging-associated conditions like insulin resistance, fatty liver disease, high cholesterol, and we'll talk about even the full suite of changes consistent with Alzheimer's in a paper that just got published today.

C15 Discovery in Dolphins and Essential Fatty Acid Background

So this is literally hot off the press information on the cognitive health front, but just wanted to make sure I tip my hat to the dolphins with regard to kind of the foundation of the initial discovery.

Since then, 10 years of Office of Naval Research-Funded Studies, as well as teams around the world independently working on C15, amassing over 100 peer-reviewed papers on C15's benefits.

All of these papers are at discoverc15.com. So just as a reminder, C15 is special, right? And it's emerging as the first essential fatty acid to be discovered in over 90 years.

An essential fatty acid being a nutrient that our bodies require. And if we don't have enough of it, our baseline physiological health will decline. And our bodies don't make enough of it, which means that we have to get certain amounts of C-15 from our food.

Now that was a huge stake we put in the ground with Dr. Ed Dennis, the editor-in-chief of the Journal for Lipid Research for 15 years. Ed and Rick and I published that paper here in 2020 in Scientific Reports.

Since then, there's now been validation from multiple teams throughout the world who skeptically looked at that same assessment and they came back with the same conclusion, including gold standard studies that were done to initially discover the first two essential fatty acids, which are linoleic acid and omega-6 and alpha-linoleic acid and omega-3.

thrilled to report that C15 continues to pass the test with regard to being an essential fatty acid. And then there's our, we have a cute little picture of our, of C15, pentadecanoic acid there.

As a reminder, it is a dietary odd chain saturated fatty acid, which means it has no double bonds. It's just 15 carbons, an odd number of carbons. That makes this a stable fatty acid with no double bonds and it's one of its main functions is it strengthens, it goes into the cell membrane.

to strengthen cells against lipid peroxidation, this age-driving lipid peroxidation, and helps keep them strong. We'll talk about other, there are lots of things that C15 does, but it's one of the most ownable and special aspects of C15.

all while being anti-inflammatory, while a lot of the even-chain saturated fats, right, that also have no double bonds, but an even number of carbons, like C16, C18, are pro-inflammatory, especially C16, and continue to be associated with an increased risk of type 2 diabetes, heart disease, fatty liver disease.

So we're seeing very opposing roles just based upon one carbon, which is, we can go into a whole webinar just on that topic. So like I said, we're going to talk about C15 and whole body and mind health.

And so there are a lot of broad health benefits of C15 that start at the cellular level. So we're going to cover how C15 helps with red blood cell health, liver health, and iron metabolism, which are intricately linked with red blood cell health, as we'll show, metabolic health, heart health, gut health and cognitive health.

So we're going to cover a bunch quickly and I'm going to go relatively quickly because again, it's kind of like your preview of the buffet, but I also want to make sure that you have plenty of time to ask questions because some of the feedback we got in the last webinar is we really want lots of Q&A time, which I love that.

So in order to really understand how C15 has these broad benefits, we really need to start with ferroptosis, right? And we talked about this in the last webinar a bit, but it's really worth re-emphasizing.

And ferroptosis is a newly discovered form of cell death that was discovered by researchers Dixon et al at Columbia University back in 2012. So we know there's apoptosis, necrosis, autophagy, right?

Three ways our cells die. they discovered a fourth, and they called it ferroptosis. Now, what ferroptosis does, and all of these images are from my Ben Watson paper last year in metabolites, and again, all of these are at discoverc15.com, that what Dixon showed was they found that cells had fragile fatty acids in their cell membrane that led to increased amounts of lipid peroxidation.

That lipid peroxidation then combined with intracellular iron, which no one was right quite clear why that iron was there. But when you combine lipid peroxidation with this intracellular iron, you get massive reactive oxygen species that then attack the mitochondria and take them out.

So when you look at these cells under the microscope that have died from ferroptosis, the cell membrane is intact, but it's slightly thickened. The nucleus is intact and it looks like an otherwise healthy cell, except for the fact that all the mitochondria are dead and the cell itself is dead.

So that is the definition of for apthosis. It then this then happens in the body and it results in systemic as it advances systemic lipid peroxidation, systemic ROS, and systemic iron overload.

Ferroptosis and C15 Deficiency Mechanism

This, what we show, and what the dolphins showed us, is it starts at the liver, but it then expands to the pancreas, to the heart, and what I should have added here, and to the brain.

So if you do a Google search or PubMed search for iron overload, right, and people, you'll see that the behavior of publications have evolved from being a hereditary iron overload or hereditary hemochromatosis to now this non-hereditary form of iron overload.

Last year, 2024, there were over a thousand papers published on iron overload and the impact it's having throughout the body. That's there when you can just see kind of the results of iron deposition and fructosis throughout the body.

Over 10,000 papers have been published on fructosis since 2012. We know that it does all of these things based upon those extensive studies. What nobody could explain was where ferroptosis was coming from.

Why were we having this new form of cell death? And that's where we showed in the metabolites paper, really the accumulation of the last decade of studies showing that C15 deficiency causes ferroptosis.

So briefly, we, um, in the metabolites paper, we walk through all of the science showing the following that if we have less than 0.2% or probably to be even more accurate, if we have less than five micrograms per mil of C15 in our red blood cells, that results in cell fragility and lipid peroxidation and all these components we know now, which is for optosis.

Our, the fragility of our cells includes our red blood cells. And so when those red blood cells become fragile, which is one of the first things you see in a C15 deficiency, the macrophages in the liver, which are called Kupfer cells, engulf those red blood cells.

And that then results in what you would see in your patients as accumulating iron overload. So if you have a lot of red blood cells that are getting engulfed, by the liver that results in overwhelming the liver with iron in which it's not able to recycle that out, it just starts accumulating.

So what you would see in your patients is you would start seeing high serum ferritin or hyperferritinemia not caused by just inflammation with a normal transparent saturation.

It then progresses to fructosis in the liver and then results in all these downstream complications of which you all know well are intricately linked.

So then what we also know is, and we've been able to demonstrate, is that all of this is fixable with C15, right? is such a clean and clear picture that that's why we talk about a nutritional deficiency.

It's like vitamin C deficiency in scurvy and vitamin D deficiency in rickets, right? That if the issue is truly caused by a deficiency, when you put that nutrient back into the system, you see the reversal of the syndrome.

And that's what we've been able to show. So you put C15 back into the system, whether it's an animal model, or a recent clinical trial in humans, you give them C15 supplementation.

It gets the C15 levels above that five micrograms per mil. You see improved hemoglobin, red blood cell stability, a lowered red blood cell distribution width.

You see attenuated engulfment by the macrophages, lower liver iron deposition, reverse for apoptosis components, and then evidence of all the downstream benefits.

So I just kind of wanted to cover this. We're going to talk system by system. But really, I think what's really important is that there is a core underlying factor that I'm not even going to talk about when we're going to talk about these systems from a very specific a mechanism of action that mechanisms of action with which you're familiar, but I just don't want the crew to forget this underlying component of fructosis and C15 deficiencies.

All right, so here we go, whirlwind tour. So let's talk about C15 and red blood cell health. And we know that C15 plays a role in stabilizing red blood cells.

So the addressable issues in your parents, in your patients, and probably your parents, are that you see evidence of fragile red blood cell membranes.

This would be declining hemoglobin over time. increasing red blood cell distribution with, and that's because the cells are dying prematurely. It's forcing the body to make more red blood cells to compensate.

You get more red blood cell variability, and that results to this increasing RDW. Over time, that progresses to anemia. The beneficial mechanisms of action, like I shared, for red blood cells, it's super simple.

C15 goes into the red blood cell membrane, which you can measure, and that helps to protect against lipid peroxidation and increases the stability of the cell.

When we look at in vivo studies, we know that models of anemia that are due to fragile red blood cells, that C15 supplementation stabilizes the red blood cells, it increases hemoglobin, decreases red blood cell distribution with, and alleviates anemia.

And then in humans, there is a study by Sobolewski et al that showed people, specifically children, with higher C15 had lower lipid peroxidation levels systemically, and they had red blood cells that had longer lifespans.

Probably just as importantly, in a placebo-controlled clinical trial by Robinson et al, they showed in which two out of three participants met the criteria of being C15 deficient, those people who took C15 and achieved over five micrograms per mil had improved hemoglobin concentrations.

So I picked a couple of my favorite graphs for each of the parts that we cover. And this one was the initial one from the dolphins. So we gave dolphins a higher C15 fish diet that significantly improved their red blood cell C15 levels And as a result, you can see here that within bi-month three, every dolphin that had anemia, which is below this black line, every dolphin had resolved anemia.

So this was, they were given fish, so it wasn't just C15, but it really got us thinking because as we looked at what were the independent predictors of this improvement, of the entire metabolome, it was C15 concentration in the red blood cell membrane that independently predicted this improved outcome.

And what we showed is really a threshold effect, that once we got C15 greater than five micrograms per mil in the dolphins, when they were low, C15, which this would be one to two micrograms per mil, and we gave him the modified diet and got them up to five micrograms per mil, there were no longer any dolphins that had anemia.

So again, it was a very clear picture for us from this very clean phenotype in the dolphins. And then we've sensing supportive information in controlled animal models, like I shared, and now evidence in humans.

So with that, we're going to move to the liver. Because once we have fragile red blood cell membranes, like we talked about with ferruptosis, those get engulfed by the copper cells that increases liver iron deposition and ferruptosis.

And that leads to what's called dysmetabolic iron overload syndrome. That's another term that, if you haven't heard before, you can look up on PubMed and is gaining attention.

It falls right in line with the iron overload conversations. We know that if a person has iron overload and they have

fatty liver disease, they are much more likely to advance to mash or nash steatohepatitis.

And again, what you would see here is hyperferritinemia, which is measuring iron, tissue iron deposition, as well as elevated liver enzymes. And this is what we saw in the dolphins, by the way.

So the beneficial mechanisms of action is that C15, again, stabilizes those red blood cells.

Red Blood Cell Health and Anemia

It attenuates DIOS. for eptosis and lipid peroxidation. We also know that C15 activates PPAR-alpha-delta, which has been one of the top mechanisms that drug developers have used, have been targeting as a means to treat Masold and MASH.

And it's because PPAR-alpha-delta agonists help to lower inflammation, including IL-6 and TNF-alpha. You're going to hear those cytokines a lot. with regard to C15, and it directly decreases fibrosis in cell models, disease cell systems, mimicking fatty liver fibrosis.

So in the in vivo studies, that translated well. So in models of DIOS, Masold, and MASH, we saw alleviated DIOS, so no more iron deposition in the liver.

It lowered liver lipid peroxidation, lowered liver inflammation, and fibrosis. And in human studies, Jeff Schwimmer helped with both of these studies.

He's a leader in fatty liver disease in kids, and he showed that the higher the C15, among 372 kids, the higher their C15 levels, the lower their liver fat.

He then followed up with this placebo-controlled clinical trial, and he showed that, again, of the people who got up and over that five micrograms per mil, They saw not only improvement in hemoglobin, but more importantly, because they started with high liver enzymes, they had lowered liver enzymes, specifically ALT and AST.

So here are a couple data points from these studies. This was that animal model. that has NASH with anemia and liver iron overload. So basically the dolphin model, the theroptosis phenotype, and we were able to show that when these were Japanese rabbit models, when they were given a C15 supplementation for three months, that we saw the liver fibrosis, this treated group compared to the non-treated group, saw their fibrosis score go down, iron staining basically went to zero.

and hemoglobin went up as well, and the red blood cell distribution width went down. These are some of the data from Jeff's study from the clinical trial in young adults, and he showed that of those, again, who took C15 supplementation and had this increase, we had to break it into a sub-threshold group.

Those who were in the C15 treated group, but didn't see an increase versus those who did. And of those who did, they saw an average increase of two micrograms per mil.

These are the ones that had that improvement in ALT and AST and increase in hemoglobin. When Jeff went back and looked at the study group, he did say, listen, these are 18 to 24 year olds during COVID.

So he's like, this is probably one of the worst adherent groups you could ask for. And they did show that adherence to the protocol was a strong predictor of whether or not they had raised rates.

They didn't explain for all of it, but we also have to account that did they accurately report that they stuck to the protocol or not. So just keep that in mind.

More questions will be answered, I think, as we move forward with more studies. With regard to, I think what I'm going to do is I'm going to get through as much as I can and I'll pause in 10 minutes so that we have time for Q&As.

So when we talk about metabolic health, we know that C15 activates AMPK and AKT to improve glucose control. That's an easy, addressable pathology, right?

So your patients that have insulin resistance and are at risk of type 2 or have type 2 diabetes, AMPK activation, obviously it's the mechanism that Metformin uses, which is one of the top first-line treatments for managing type 2 diabetes.

And it also activates AKT, which can improve insulin sensitivity. So that explains how in models of obesity and type 2 diabetes, C15 supplementation effectively lowered glucose, as well as adipokines in pro-inflammatory cytokines, and it lowered cholesterol, And in human studies, there haven't been clinical trials specifically looking at people with glucose, with hyperglycemia.

But there are so many studies, multiple meta-analyses of prospective cohort studies repeatedly showing that people with higher C15 levels have a lower risk of developing type 2 diabetes.

And then Lee et al. published a paper in 2022 showing that for every 50% incremental increase in the percent of C15, the risk of type 2 diabetes was reduced by 8%. Again, association studies, not intervention studies.

So here are my favorite graphs from these studies. This shows how C15 activates AMPK. Interestingly, we'll be diving more. We'll probably do a whole webinar just on different behaviors of C15 at different concentrations.

But here it shows that at a 40 micromolar level, it had that significant ability to activate AMPK. Interestingly, this is a foetal study. It showed that C15 was just as good at helping with glucose uptake of cells as insulin.

So it actually had an insulin mimicking effect, not just an insulin sensitizing effect.

Liver Health, Iron Overload, and Fatty Liver Disease

More to get played out in the human realm, but thought that was really interesting. and as well as showing that C15 increases insulin sensitivity. So if you give it to cells, you give it insulin, it does better.

If glucose uptake, you give it insulin plus C15, and the cells did even better. And these were muscle cells. When we move to heart health, we know that C15 in multiple studies has been shown to lower cholesterol and vascular inflammation.

So addressable pathology is obviously lowering cholesterol. I am super interested in it. Does it specifically lower oxidized LDL cholesterol or small and dense LDL?

And then it also can lower a vascular inflammation in IL-6, MCP-1, TNF-alpha, and addressing comorbidities of heart health, which include type 2 diabetes and Masold.

People with Masold are are more likely to die from heart disease than from actual direct liver disease. So these are very closely linked diseases. The beneficial mechanisms of action, we talked about it activates AMPK and PPAR-alpha.

Those are also mechanisms that can lower LDL cholesterol. It also inhibits Jakstat, which helps explain how it lowers these pro-inflammatory cytokines, specifically in human cell systems that were mimicking vascular inflammation.

So in vivo studies, they're limited to obesity and type 2 diabetes. So these were not just heart disease models, but again, lowered these pro-inflammatory cytokines, lowered cholesterol.

And then in humans, people with higher C15 have a lower risk of developing heart disease, coronary heart disease, including a 17% lower risk for every 50% incremental increase in C15, and heart failure.

And all of these are meta-analyses as well. And then in a control clinical trial by Choi et al, they showed that women who were supplemented with C15 for 12 weeks had lower LDL cholesterol.

My graph pick here was from True et al. This was a large prospective study with over 5,060 year olds that they then tracked for 20 years. and looked for incident cardiovascular disease.

And what they showed was the higher the C15 in their blood from 0.2 to 0.5%, there was a linear decrease in the risk of developing heart disease. Okay, I can do this.

Okay, so with regard to gut health, these are newer. So what's newer to the scene are gut health, and then I'm gonna talk, just touch on cognitive health, because we're gonna do a whole webinar on just that topic.

But with regard to gut health, there are some really interesting studies that just came out in this last year showing that in mouse models, it attenuates leaky gut, lowers inflammation, and supports a healthy gut microbiome.

The addressable pathologies are folks with leaky gut syndrome and intestinal inflammation. The beneficial mechanisms of action are strengthened cells.

So we get back to those stronger cells. This includes epithelial cells in the gut, and it closes up that leaky gut issue. And it also inhibits JAK-STAT, which again gets back to lowering gut inflammation.

In in vivo studies, multiple models of inflammatory bowel disease, mouse models have shown that C15 improves the integrity of the gut lining. So a less leaky gut, it decreases gut inflammation and it's supported intestinal healing.

And in human studies, there's just one hint toward gut health because they didn't focus on gut health in these studies, but the Choi et al. clinical trial in Singapore showed that women, this involved all women, they showed that women who supplemented with C15 for 12 weeks had in addition to lower LDL cholesterol, improved growth of bifidobacterium adolescentis, which is a great mama microbe.

It's probably one of the most important microbes from an abundant standpoint that helps not only support healthy metabolism, but has actually been shown to improve longevity in worms.

So keep that in mind that it was in worms. So my favorite picks from the gut health, this one is from Singh et al. showing that when they gave mice C15, it had a short term benefit.

All these things you see, they gained body weight. So these were mice that were given DSS to induce colitis. And so it had these benefits of basically improved tissue health, improved body weight gain and evidence of less leaky gut in the short term.

In the long term, C15 had a benefit on the model of ileitis. So more to be determined with regard to short versus long term effects and specifically which types of inflammatory bowel disease.

This is a separate study that was put out by Wu et al. Again, in mouse models of inflammatory bowel disease showing that this is the healthy control This is a mouse that was exposed to DSS to induce colitis and inflammatory bowel disease and leaky gut syndrome.

And then this shows the model that had DSS and C15. You can see where the tissues on both of these are looking a whole lot more like those controls than versus the DSS.

This is in part not only to, like I said, improving the cells, but reducing inflammation including TNF-alpha, IL-1-beta, and IL-6 in these models. Okay, last one.

I did it. Okay, so C15 in cognitive health. This paper just came out today in the International Journal of Molecular Sciences, where we focused on cognitive health.

And what this paper shows is that C15 improves cellular signaling related to improve memory and cognitive health. The addressable pathology that we talk about in the paper is, you know, we know we have declining function with age.

that this declining cognitive function is linked to all the comorbidities I just talked about, fatty liver disease and heart disease and type two diabetes, that it's all that type three diabetes, right?

Which includes neurodegenerative diseases. We know that MAOB and FAAH increase with age. And again, I'm saving this to go into detail in the future. So just consider this a little bit of a teaser of talking about what MAOB and FAAH are.

But we know that C15 inhibits both MAOB and FAAH, and these activities are relevant, if inhibiting these are relevant to neuroprotection, lowering neuroinflammation, and amyloid beta plaque formation in animal models.

improved mood and lowered pain, and the paper goes into detail about these activities. In in vivo studies, not with C15, because nobody has done in vivo studies on C15 for cognitive health, but in in vivo studies of other compounds that have these same activities of inhibiting MAOB or FAH have shown that that can lower amyloid beta plaque formation to decrease inflammation, and improve cognition in animal models.

So again, just note my caveats there. In human studies, there is an interesting study that was Shen et al, showed that people with type 2 diabetes, again, who are at higher risk of developing cognitive decline, people with type 2 diabetes and higher C15 had better overall cognition scores, and improved recall and memory.

So a hint that we may be heading in the right direction. Just a quick little peek at the paper. This is where we showed that the dolphins, that older Navy dolphins get amyloid beta plaques.

We're not the first to show it. This has actually been demonstrated in probably six different cetacean, wild cetacean species to date. What was new is we actually looked at the amount of amyloid beta plaque deposition as well as neuroinflammation and activated microglia by brain region.

Metabolic Health and Glucose Control

And we looked at six different brain regions. And what we showed was specifically in the hippocampus that older dolphins had higher amyloid beta plaque density, very similar to humans.

We also showed with regard to neuroinflammation that dolphins, absolutely older dolphins are susceptible to neuroinflammation. And when we look specifically at activated microglia, CD68, that the hippocampus had the highest amount of activated microglia.

So when you bring these two together, The hippocampus is so important in human pathophysiology of Alzheimer's as well as other diseases, because again, this is our memory center, this is our happy center, and so when that gets compromised, it impacts our mood and memory.

And so these are just the two graphs showing that we had C15 tested against 39 different targets. This screen is specifically to look for therapeutic candidates for Alzheimer's disease, and it's from urofins.

And so we said, gosh, let's test C15 against this, knowing that dolphins get The full suite they get, now we know they get Alzheimer's like disease in the brain, they get fatty liver disease, they get iron overload, they get insulin sensitivity.

So let's see what C15 can do specifically for cognitive health. And this is where you can see a very, this is like a drug, right? So a dose dependent increase in FAAH inhibition, really heading between starting at one micromolar and working its way up at maximum activity at 50 micromolars and then seeing the similar thing with MAOP.

So we did run five other what we thought promising candidate molecules that we had discovered from the dolphins

against this panel and C15 was the only one that not only hit one but two promising receptors.

Uh, so with that, uh, there's a book so happy. Um, and if you helped make this a number one bestseller on Amazon, thank you for that. And, um, uh, well, it's, it's a great.

It's a great resource for, and I think it's going to be used a lot from like, um, kind of your go-to, um, from with a healthcare professional mindset of, you know, the book has tons and tons of, of references.

But then what I'm finding useful myself is the index. So you can basically look up any term or word you want in the index and be like, okay, what does C15 have to do with blah?

And so I am now using it every day to help answer questions. So hopefully, I hope it's useful for you too. So read the longevity nutrient. The other team's here, the RCP team is here to support you with regard to fatty 15. And again, all the science that I talked about and more, including the paper that just published is on discoverc15.com resources.

Okay, I'm going to stop talking. Danielle, did you have something to share? Thank you. Yes. So thank you so much. That was, like Dr. Steph said, that was just a buffet of some of the information she has.

What a breadth of information, you have, and if we can go deeper into each of these topics as we progress so our goal is to do one of these with Dr step a quarter we'd love for you all to join us and invite your colleagues to come and learn as well.

you know, each of these areas in more depth, but to learn a little bit of each was awesome. And so we, and thank you all for participating in the questions.

We have several for you and we'll get started on that. I do want to give you a discount code. Now note this discount code is actually non-functioning.

So if you go to our website and try and use it, It won't work because of it being a wholesale offer. So you have to reach out to your representative. If you don't know who your representative is, please reach out to hcp at fatty15.com.

We'll put that in the chat. And that's an email address to where you can reach your rep. But the code is book 15. And the offer is we're going to give 15% off your wholesale order and give you a free book.

With any purchase you'll get 15% off as well as a free longevity nutrient book and every chapter I read I felt like I should just add even more to what I'm already taking.

I do have all of my dogs, my daughter, myself, my husband, and my parents now taking it. But as you all read, I think you'll feel compelled and confident to recommend it to more and more patient types.

So with that, we'll get started on some of the questions.

Heart Health and Cholesterol

So the first question, beginning with dosage. So the question was, is there a maximum dose limit? Is it safe for children and during pregnancy? So from a tox standpoint, we have not seen a maximum dose.

So we've not seen evidence of toxicity. This includes, we have now, you know, almost a hundred thousand active subscribers. We've been around for four years.

And we've done the, with regard to the rodent studies that were to get the grass status that put it at a hundred times in which they were tested a hundred times of the dose.

of that 100 to 200 milligrams and saw no toxicity at those doses. So that it makes sense given that we're not really giving hyper doses that what we used to get from our diet used to be between one to 500 milligrams a day.

But what the Navy wanted to fund us to do was to say, hey, can you give that amount, but in a pure bioavailable form, that isn't having to compete with all of the pro-inflammatory fats that C-15 is in with food.

So we were literally funded by the Navy to help develop this pure C15 ingredient in fatty 15. So, and all the science I shared is on the pure ingredient.

There was a study, for example, done by Bishop et al, right, that I think we talked about in the last one, where if you give a mouse C15 in an obese type 2 diabetes mouse C15, as you saw, they got better.

If you give it milk fat, it got worse. So fatty liver disease got worse, inflammation and whatnot. So a need to be able to Truly, you see 15 have this as a supplement as a way to supplement our diets so that we can help people really leverage the benefits.

So no evidence of tox children. The current grass approval is for which is really for ingredient versus supplement. But being the most conservative, it's for children who are for and up.

And not only is it considered safe for pregnant women and nursing moms, Um, we've just got an approved claim that it supports healthy pregnancy. Um, and again, that could be a different topic.

Um, but we were very happy to get, we have an independent third party who's been reviewing our claims and everything you just saw today. Um, again, not disease treating with regard to structure function claims, um, has, uh, has been approved by, by that, that third independent party.

Amazing, thank you. And another question was, should FATI-15 be used as a foundational therapy or targeted therapy? Yeah, that's a great question. So depending upon where your patients are at, right, can dictate what dose they need.

So the good thing is that there is C15 testing. So you can, that's, C15 has been tested for decades using GCMS. So if you use a fatty acid panel, just check and make sure that they have C15 included.

It's not a given. that they do, but increasingly it's C-15 is being included in fatty acid panels. Ideally, you get concentration. So often they report percentage.

You want to get concentration of five micrograms per mil. So if the goal is to not be deficient, right, then you can dictate the dose. based upon where their baseline levels are and get them up and over that dose or up and over that level.

If the goal is, for example, we didn't talk about this today, but again, tapping into the activities, the mechanisms of action that are antidepressant and anxiety lowering, which we hear from a lot of our consumers, customers, that that is a lower dose that you don't need, they don't need to have a lot.

While others like this cognitive health side, you saw that the maximum cognitive health signaling activities was at 50 micromolars, which would be equal more to that 300 a milligram dose.

So it's really tailoring. And so what we're going to do, we've been working with our team to develop a guide that based upon the need, and we'll put it in structure function claim form, can help dictate what levels you would want to use for dosing.

And it's a really fascinating part. We published that in PLOS ONE. We did cell-based phenotypic profiling of C15 in which they profiled it across 148 different biomarkers.

Gut Health and Intestinal Inflammation

And then they compared that to 40,000 other compounds that profile. And it showed that at low doses, it mimicked bupropion. At middle doses, it mimicked antimicrobials and anti-inflammatories.

And at high doses, it mimicked anti-cancer drugs. So very different behaviors at different doses and no evidence of toxicity. at those doses. Thank you.

And I can't wait to get that guide and share it with you all as well. Another question was, is deficiency relative or absolute as it's related to an excess of other fatty acids?

Oh, that's an interesting question. I think it's I think because the most reliable data have come from the actual concentration, I think that's where we're going to get.

You're going to get the best information that they need five micrograms per mil or higher to get those benefits. I think where it's going to land is they need X amount in their cell membranes versus a percentage, right, which is relative.

Right now, we're looking at they need to be above 0.2 percent to not be deficient. But the numbers, it's just it's less reliable. But if that's all that you have access to, then the relative can work with regard to.

We know that if they're in the 0.3 to 0.4 percent in most of the assays, they're in pretty they're in non-deficiency territory. At that point too, it's kind of like just, it's like you're standing on the edge of a cliff and you may be easily pushed over or you're fine if you're just standing there and nothing pushes you.

So that was kind of a long-winded answer to- Oh, that was great. To be able to answer that. So I would go with actual amount that you need versus relative.

Yeah. Amazing. And something I like to remind myself of also is there's a difference between not being deficient and being optimal. And there's probably different patient types in your practice.

There may be the people who just are aiming to not be sick and to not be deficient, but there may be those that are optimizing their health and that may be a whole different dose, right?

Yeah, that's right. Okay. And then another question, how long before you see changes in blood work? So reliably, uh, between three to six months and blood work.

And some of that has to do with, you know, like there is a test that measures C-15 in red blood cell membranes. And that's another thing to pay attention to because it'll measure it either in serum or plasma, which you'll see an immediate increase in C-15.

But again, it takes three to six months for that to translate. Um, consistently to whether or not a person's gonna benefit. As we saw in Jeff Schwimmer's study, if they really need to be taking it, taking it reliably, because if they don't, obviously their numbers aren't gonna move.

But yeah, yeah, that's it there. Great. Any data on C15 autoimmune disease and in menopausal women? Um, yeah autoimmune disease is the next to last chapter next the next last chapter in the book.

When we did the human cell system studies I guess is a robust panel called bio map. That is. very good at predicting what a molecule is going to do. It's usually for drugs.

We've been able to use it very well for C15, and thus far it has panned out. It predicted it would do this, have anti-cancer properties at 50 micromolars.

Now there are groups that have shown that C15 decreases tumor size in mice with breast cancer. So We see that translation happening over and over again.

So we saw that in those Biomap systems that C15 lowered over 18 pro-inflammatory cytokines, including in cell systems mimicking autoimmune diseases, specifically lupus, rheumatoid arthritis, and psoriasis.

So cells, these are cells. We do, I myself have atopic dermatitis, I can tell you for sure that fatty 15 helped me calm, helped to calm that down, but, and we have, you know, individual anecdotal evidence, but, so I'll leave it at that, but no clinical trials to date have tested C15 for any specific autoimmune diseases.

Okay. And then, can C15 be absorbed when people have hypothyroid or other conditions affecting fatty acid metabolism? It should be bioavailable. So the benefit of one of the things maybe wanted us to address was to provide a fatty acid that was highly bioavailable.

Cognitive Health and Brain Signaling

So in food, C15 is attached to triacylglycerides, which are not absorbable. So when you get it from dairy fat, which is our primary source of C15, We need digestive enzymes to basically cleave C15 off of that trace cell glyceride to be a free fatty acid that we could then absorb.

So we cut to the chase and the C15 ingredient we have in fatty 15 is a pure free fatty acid, C15. We were lucky in which there were extensive clinical trials done with this exact molecule and ingredient.

And it was done for patients. They did it in a bunch of healthy people, different ages, genders, including children. And they wanted to see if C15, this pure feed fatty acid, could be a marker of absorption of fats, specifically looking at people with cystic fibrosis.

But what they showed was C15 was not a good biomarker because it was 100% bioavailable in both the healthy population and the population with cystic fibrosis.

So they actually ended up having to use a different molecule that wasn't as well absorbed as C15 in people with cystic fibrosis. So it's to date, understanding those populations in those studies, most people should absorb C15.

as long as they're taking it reliably. Yes. Okay, awesome. And what has changed about our food supply that C15 levels have decreased so sharply? Yeah, that's a great question.

So first, you know, our primary dolphins got C15 from certain types of fish. And one of the reasons they became C-15 deficient, one in three of them, is because the Navy lost Yulicon, which was a super fatty fish in which all dolphins were getting lots of C-15 in the 1990s.

And then it got mixed. Some dolphins were getting some high C-15 fish and others were not, hence the one in three. For us, our primary source of C-15 is from dairy fat.

And I will mention the fish, the dolphins are getting C-15 not only from fatty fish, but it's specifically in the skin and the heads of fish. So that's why, in general, fish is not a reliable ongoing source of C-15 for us, unless you eat the whole fish.

For us, dairy fat is such a reliable marker of dairy, of dairy fat that it's been used as a biomarker for dairy fat intake for decades. Nobody knew that it was actually beneficial, but what they did know is C15 accurately reflects how much dairy fat you eat.

So, sorry, do you know what was in the quote? What was the question? Oh, that's okay. So what had changed about our food supply that C15 levels had decreased so sharply.

So that was a long lead up to as we've decreased in 1977, congressional recommendations came out, now shall not eat saturated fats. And the main way you can decrease your intake of saturated fats is by stop eating butter and the whole fat milk.

Those are, yes, those have lots of pro-inflammatory saturated fatty acids, but it was also our primary source of C15. So as generations have past have have continued, they are remarkably decreasing not only their intake of whole dairy fat, but they're just avoiding cow's milk altogether.

So much so that the USDA released a report of the concerning avoidance of cows milk in generated like you guys created this. So it's very that was not they did not intend for people to stop drinking milk all together.

So C15 levels have gone down. When you add on top of that in the 1990s, pediatricians really doubled down on kits. So when kids turn two years old, every parent is given a piece of paper that says, regardless of their health state, that they should go move off whole fat cow's milk and go to low fat or non fat cow's milk.

Further, if mom is C15 deficient, that then makes her milk C15 deficient. And there are a whole set of really fascinating studies showing that the more C15 mom eats, the more C15 baby gets in the womb and through her milk, the better that baby grows, the better that baby's development and following in the Eden cohort study in France, the better their cognitive development.

So there's mom. And then the other is that cows, we now know that cows that are fed grass have twice as much C15 than cows that are fed corn. So industrial, changes are going to influence how much C-15 is even in whole fat milk.

So lots of these, you add up all these reasons together and that's resulted in lower C-15 levels population-wide, especially among kids that were born in the 1990s.

These people are now in their thirties and we're now seeing what? A rise in coronary heart disease, fatty liver disease, which didn't, non-alcoholic that didn't exist before 1980. We're seeing a rise in these conditions.

in these populations. So one of the hypotheses are C-15 deficiencies that they've had since birth may be contributing to this rise. Awesome. Okay, and then is it possible that C-15 could affect the action of insulin in the type 1 diabetic?

That's a good question. You know, it used to be so clean where type 1 diabetes was juvenile onset diabetes and type 2 diabetes was adult onset diabetes and never the two shall cross.

Q&A, Dosing, Safety, and Product Information

We're increasingly seeing phenotypes of type 1 diabetes that also come with type 2 diabetes-like issues and I'm not sure about that quite vice versa. So I would say among these, not kids, but people who have this mixed type 1, type 2 phenotype, that that's where C15 may come in and help improve insulin sensitivity issues.

There is one study that showed that moms who had higher C15 were less likely to have children that not only developed, didn't, less likely to have children that developed food allergies, but also type 1 diabetes.

Association study, one study, so keep that in mind, but looking at that autoimmune component is interesting. But most, almost all the studies have focused on type 2. Okay, thank you.

And then I guess one last one, is fatty 15 able to cross the blood brain barrier? And if so, what benefits regarding brain injury, post-concussion syndrome, diabetes type 3, meaning Alzheimer's or senile dementia, a post-stroke could be expected?

We're learning. This paper's a good start. It does cross the blood brain barrier, so it's a small molecule. that makes it across. There are multiple fronts, right?

So not only knowing that C15 can inhibit FAAH and MOAB, which we will talk about in the next webinar, which are neuroprotective, anti-inflammatory, helps to sustain the amount of dopamine in the brain, like all of these good core functions of protecting brain function.

But also, right, our bodies use C15 to make a second molecule called pentadecanolcarnitine. We published that in 2022, I think. And pentadecanolcarnitine is the second ever discovered full acting endocannabinoid.

So this metabolite of C15 fully activates CB1 and CB2. That also crosses the blood brain barrier and would provide the endocannabinoid-supportive, brain-supportive activities that we know of, anandamide and 2-AG.

That took us a sec. There's so much detail here. Yeah, there's so much detail. I'm really excited to dig even deeper. I think our next quarter, we're gonna focus on cognitive health in more depth.

So please join us if you're interested in that. Thank you so much for engaging in all your questions. I know there are several that did not get answered.

We will be exporting the chat and we will get you your answers. We will follow up, your sales rep will follow up with detailed study links. in depth answers as much as we can get to you.

So we really appreciate your participation and your time tonight. Again, the code to give to your sales rep is book 15, book one five. It does not function when you check out, just email it to your sales rep and let them know and they will apply the discount 15% off and a free book.

So thank you so much. Thank you, Dr. Steph. Thank you all for attending. And we look forward to seeing you again. Yeah. And thank you for your evening, everybody.

Your time is valuable. Thanks for spending it with us. Great. Thank you.