Latest Updates on Glyphosate: A Special Interview With Dr. Stephanie Seneff

By Dr. Joseph Mercola

JM: Dr. Joseph Mercola
SS: Dr. Stephanie Seneff

JM: Hi everyone, this is Dr. Mercola helping you take control of your health and I am at an Autism event in Atlanta, Georgia called TACA, which is The Autism Community in Action. They had some great speakers here and I was able to capture one of them. Who you may recognize as Dr. Stephanie Seneff, and she's going to give us her latest updates. If you don't know who she is, she is a Senior... What is it?

SS: Senior Research Scientist.

JM: Senior Research Scientist at probably the most prestigious academic institution in the world, at least by some agencies that evaluate that, which is MIT. So her initial work was in artificial intelligence.

SS: Yes, and the precursor to Siri and the Amazon Eco. The new development or spoken computer dialogue systems.

JM: She's got a great brain. And she's refocused her interest more in biology in recent years, and there's a fascinating story about that. And she's really a champion now for helping to understand how glyphosate is an issue, and she's presented some of her new findings here at the conference and I just wanted her to have her share them with you. So welcome and thank you for joining us.

SS: Thank you so much for having me.

JM: So it's been a while since we talked or at least recorded you on an interview. You were at my house not too long ago.

SS: I know. We had a great time.

JM: Yeah. So why don't you give us an update? What's going on now with... Well, yeah. Tell us what's new and then I'll try to play the role of the person watching this and interrupt you when I think that it needs some type of clarification.

SS: Right, because things can get messy. Science is hard and that's one thing I'm realizing. Glyphosate is an absolutely fascinating molecule. I've become hooked on it so to speak. And I just love the research, I love the puzzle. And glyphosate is the mother of all puzzles in my opinion. I believe I'm zeroing in on the mechanism of toxicity, and it's unique to glyphosate, and insidious, and cumulative.

SS: So it's extremely dangerous in the sense that it doesn't bowl you over. You get small exposures to glyphosate all day long in your food, in the air, in the water. Probably breathing the air from the gasoline tank. We don't know. But it's pervasive in the environment so we can't avoid it. And the United States has the highest... we use the most glyphosate per person per capita in this country.

JM: Interesting. What is that number?

SS: Oh, I don't know the per capita number. And I don't even know the number because it's just a huge number.

JM: I think it's like... isn't it like 4- 5 billion pounds a year?

SS: It's mind mindbogglingly huge.

JM: Worldwide?

SS: Yes, worldwide. And the U.S. I think uses 20% of the worldwide, which is a lot compared to our population which is maybe only 5% of the total population of the world. So we're using much more. Canada also uses a lot. And both of those countries have a lot of heart health issues, high Autism rates, lots of autoimmune diseases, food allergies, Alzheimer's is going up dramatically. Of course diabetes, obesity, all these things are going up dramatically in our population. We don't know why. We see that glyphosate is perfectly correlated with many of these diseases. It's also going up exactly in step with these diseases, and there's many, many plots that I've put together in collaboration with other people. We've published many papers. We've shown... I mean, it's just amazing.

JM: Yeah, one of them is Dr. Anthony Samsel.

SS: Yes, right.

JM: Who I introduced you to.

SS: Yes, I know and that is such a great story really.

JM: Yeah, at the time it wasn't about glyphosate it was about Vitamin D. We were promoting your thesis of Vitamin D sulfate. And then he would come on and just give us the hardest time. Stephanie, you got to come on and defend us.

SS: Yeah, I know. And you know, I had forgot that that was how we had met. But then he insisted it was. Then I checked our notes and sure enough it was. I knew that you had introduced us, but I hadn't remembered that it was because of the sulfate issue. I had thought it was because of glyphosate because it was so perfect. And he had identified as a problem because of his own personal experience with it.

SS: And now he has this organic garden that he grows and feeds his own family off of his own food that he grows. He's very self-sufficient.

JM: Yeah. It’s a great collaboration between the both of you.

SS:
He's really interesting. He's a fascinating person. Very, very brilliant and I've learned a lot from him. And certainly he got me started with glyphosate. I'm just hung up on this molecule. It is so fascinating. As I said I love puzzles and I love biology.

SS: And biology has so many complicated metabolic pathways you just get lost in it.

JM: Yeah, and just a little tangent here to augment what you just stated is that you wrote a fiction book. [inaudible 00:04:35] and I actually wrote the forward for it. And it really describes your story and process. It's just absolutely fascinating. I hardly ever read fiction but I read this one. What's the name of it?

SS: Cindy & Erica's Obsession. And then it has a big subtitle about solving all the issues.

JM: It's a brilliant [crosstalk 00:04:53] story.

SS: Yeah. And I twas fund to do because I put a lot of science into it and I wasn't sure... I was trying to not be off putting because I know people, their eyes glaze over with science. But I worked it in.

JM: Oh, it was good.

SS: Yeah. And of course it was also my journey to try and figure out how cholesterol sulfate... you know, it was so interesting for me because when I first started out maybe 10 to 12 years ago looking into Autism and looking into heart disease because of personal reasons. And the two converged on the same thing which was sulfate deficiency, and I identified that before I met glyphosate. I didn't even know the word glyphosate. I saw sulfate deficiency. And then I could see how glyphosate causes.

SS: And this is what is amazing to me. Glyphosate causes sulfate deficiency in so many ways. It's almost like it's a perfect storm.

JM: What's the mechanism? Because you wouldn't necessarily think that because it's normally a chelator, it chelates these trace minerals out.

SS: Yes, and that is trouble.

JM: And it affects the microbiome and the pathway that you and Don Huber described of the shikimate pathway.

SS: Right.

JM: But, how does the sulfur enter into it?

SS: Yeah. It took a while to figure all that out, and I twas sort of teasing me all the time with little bits and pieces. And now I really got it worked out I believe. And I think a key thing is sulfite oxidase.

JM: Is that SuOx?

SS: Yeah, SuOx and actually Bob [inaudible 00:06:15] talked about SuOx in his talk. Very, very interesting. Sulfite oxidase is a super important enzyme that, as you might imagine, it takes sulfide as input and it turns it into sulfate.

SS: So if it's broken you get too much sulfide and too little sulfate. Very simple.

JM: Ah, I think I'm beginning to see the connection now.

SS: Yeah, so that's pretty much straight forward. And sulfide is extremely toxic. It's a very reactive molecule like ONOO.

JM: Yeah, peroxynitrite.

SS: Peroxynitrite. Yeah, it's right up there with peroxynitrite. And people who have a defective version of sulfite oxidase have the incredibly serious genetic disease.

JM: So genetics snip for that.

SS: Yeah, they have a version that's got a problem that doesn't work because of genetics. Their life is a mess. They get all kinds of metabolic problems, they get Autism, they die early, by the age of five they're dead. I mean, it's a really devastating disorder. So that shows you it's extremely important and also it's connected to Autism.

SS: And I've identified heparan sulfate deficiency in the middle of the brain. In the ventricles, you know in the cerebral spinal fluid in the ventricles. Super, super important heparan sulfate in there. And they've identified through multiple studies both in mice and humans that heparan sulfate is deficient inside that area of the brain, which is critical for developing the brain. The neurons grow out of that area from these precursors, and that whole process is orchestrated by the heparan sulfate. So I think that's key, this heparan sulfate deficiency, in the developmental issues that can go on in utero and probably even beyond as you're maturing as a small child.

SS: So in the gut, the microbes and, of course also human cells, are converting sulfite to sulfate using this sulfite oxidase. So, if that's busted you can't do that. Sulfite piles up. Now you've got to get rid of sulfite some other way. And what happens is you get an overgrowth of bacteria that's specialized in reducing sulfite to hydrogen sulfide gas.

JM: Which is pretty good.

SS: Hydrogen sulfide is very, very interesting.

JM: Yeah, but it has some benefits.

SS: It's got a bad side and a good side like with many things. And its good side is extremely interesting to me, because what I see happens is you get too much hydrogen sulfide gas in the gut, and hydrogen sulfide can diffuse very easily through everything. It can go straight through cell membranes. It doesn't have to go along the blood channels. It could just... like a gas. It is a gas. It can just spread anywhere, right?

SS: And so it goes elsewhere in the body from the gut. And when it gets to a destination site it gets oxidized into sulfate there. This is what I think is going on. And to do that you need oxidizing agents. You need an oxidative environment. You need inflammation. So now we have systemic inflammation. And that's behind so many modern diseases. That's something that many people are admitting. Systemic inflammation.

JM:
Can we take a step back because I'm still a little bit unclear. Maybe you're getting to it and I apologize if I interrupted you prematurely.

SS: No, go ahead.

JM: But, the increase of the sulfite is related to either a defect in the production of the enzyme. But how does glyphosate... does glyphosate affect that SuOx, sulfite oxidase directly?

SS: Sulfite oxidase, yes.

JM: That's what I was thinking because the [inaudible 00:09:34]

SS: Yes.

JM: Okay.

SS: It's very interesting. Actually multiple ways. One is hemme. Glyphosate has been shown to disrupt the very first enzyme in the synthesis of a component of hemme. So glyphosate messes up hemme synthesis. That's known from studies. And hemme is a critical component of sulfite oxidase. It's one of those enzymes [inaudible 00:09:52] and that means it depends on iron. Which glyphosate chelates so that makes the iron a problem as well.

SS: More than that it depends on [lipinon 00:10:00] which is another mineral that glyphosate could be keeping away. Because glyphosate binds to these minerals and makes them unavailable. So it leaves both iron and lipinon. And it's got multiple sites of extremely, highly conserved glycine residues. Where, if those glycines are mutated to something else. These mutations that cause these horrible diseases, some of them are glycine mutations. So there's certain glycines in SuOx that are absolutely critical for it to work properly. And this is where my idea of glycine substitution comes in.

SS: Because I believe that in certain proteins, in certain spots, glyphosate is able to get into the protein by mistake in place of the amino acid glycine. And to understand that glyphosate is a complete glycine molecule. It's a perfect match to glycine. Except that it has extra materials stuck onto its nitrogen atom.

SS: So when the protein that's going to recognize glycine in order to put it into DNA has to leave the nitrogen atom outside of its pocket because the nitrogen has to hook up with the next amino acid. So the fact that the nitrogen has some stuff on it doesn't matter to it. It says, "Oh, I have to fit exactly glycine very tightly." Glycine is the smallest amino acid. And in order to distinguish glycine from all the other amino acids all I need to do is make sure that I make a tiny space that fits only glycine. Alannine won't fit because it's got an extra methyl group attached to the carbon. Glyphosate will fit because it's a perfect glycine molecule. Except the nitrogen is sticking outside of that pocket so that it could hook.

SS: So the extra stuff on nitrogen is not constrained. This is important because I think a lot of people think “Oh, it can't happen."

JM: Well, I was going to mention that. This is a really highly controversial issue. And even in people who are in our camp because of the biochemistry of it. Well-respected research scientists disagree pretty strongly with you, and this isn't proven. There's no science to it, It's theoretical. But it makes a lot of sense.

SS: It certainly does and it makes more and more sense the more I study. First of all from the standpoint of which enzymes get disrupted by glyphosate. And I can find these glycine places where it would substitute in a cell. Including of course, EPSP synthase. Which is the enzyme that... this is how I started with it, because EPSP synthase is the enzyme in the shikimate pathway that glyphosate disrupts. Famously disrupts. They know that. And they've studied it. There's lot so of papers on it. It's very, very interesting.

JM: Monsanto knew this for many years.

SS: Monsanto has done those papers. And the papers are amazing because it... and when you add it all up it just seems... I don't understand why they're not saying, “Oh, of course this is why. This would explain everything that it's substituting for that glycine.” Because, there's a glycine residue in that molecule. This EPSP synthase. At the place where glyphosate disrupts things. And that glycine formed particle shape. And that shape fits a phosphate anion that's part of a precursor, a sub strait called PEP, phosphoenolpyruvate. So the phosphate, phosphoenolpyruvate fits into a pocket that the enzyme forms out of this particular amino acid.

JM: Which is what again?

SS: EPSP synthase. And so the enzyme forms the shape which fits the phosphate. And glycine is part of that shape. And there are also other amino acids that have positive charge strategically positioned to attract the phosphate. Phosphate has negative charge. So those other amino acids inside the enzyme help to hold the phosphate in place and attract a negative charge. So when the enzyme is being assembled-

JM: It's the ionic attraction networks.

SS: ... yeah, ionic attraction. [inaudible 00:13:46] but, it helps to bring it in. So what's happening I think is when the enzyme is being assembled it gets to the point where it needs... the enzyme is built like beads on a string. And it shapes as it's being built. So it's shaping into a perfect shape say, “Okay, we can attract negative charging.” And glyphosate is just like a pig in shit getting into there. Because it's like, “Okay. There's my glycine match. There's my negative charge. I need to put a phosphate back. I can put my methylphospho group right comfortably where that phosphate is supposed to go and I am good to go.” So it'll go right into that enzyme and then build it beautifully. Except that there's no spot for the phosphate that is in the sub strait to fit.

SS: So now the enzyme is dead in the water. It can't do its job.

JM: That's how shikimate pathway gets disrupted.

SS: Yes.

JM: And perhaps some people watching this aren't familiar with that. So maybe you could describe a little bit about shikimate pathway.

SS: Yeah, so the shikimate pathway is a really important pathway in plants and in many microbes. It produces the aromatic amino acids. And those are among the amino acids that are the building block of proteins. Just like glycine, tryptophan, tyrosine, and phenylalanine. Super, super important. They're not only part of the building blocks of proteins which would already be pretty drastic. But they're also precursors to all the neurotransmitters. Dopamine, serotonin, melatonin, melanin. Skin tanning agent. They're also precursors to certain B Vitamins like folate and I think niacin.

JM: Well, specifically NAD+.

SS: Yes, NAD+ comes from niacin which comes from tryptophan.

JM: [inaudible 00:15:16] pathway.

SS: Yes, tryptophan.

JM: It's a small percentage. But I didn't think it was clinically significant but it turns out it is because if you're low on NAD+ which is... we're not going to go off on a tangent but many people are for a variety of reasons. I think EMF is a big one.

SS: That's another one that I can talk about.

JM: Yeah, oh we can go into this but when that goes down then maybe... you talk to me. Then the body grabs the tryptophan and all these other important benefits of tryptophan get diminished.

SS: That's right. And of course-

JM: Are you the one that told me that?

SS: ... I might very well be. Yeah.

JM: Okay. [inaudible 00:15:48]

SS: Back when I told you. So tryptophan of course then producing serotonin and melatonin. That's why you get sleep disorder. Sleep disorder is one of the diseases that's going up exactly in step with glyphosate usage on corn crops, because of the melatonin problem I suspect, in part. There's other reasons too because of the sulfate deficiency. That's going to disrupt sleep as well. So, anyway, getting back to this EPSP synthase it's really fascinating.

SS: And the researchers found that there were... the way they discovered the version of EPSP synthase that they insert into the GMO crops..., so they make these Roundup Ready crops, glyphosate resistant. And they do that by inserting a bacterial version of EPSP synthase.

JM: Oh, that's what makes it resistant.

SS: Yeah, and that bacterial version has alanine instead of glycine at that spot.

JM: Interesting.

SS: Very, very interesting.

JM: And powerful confirmation for your theory.

SS: Absolutely. And there's a recent paper that's really fascinating comes out of Dow DuPont. I was really blown away by this paper, because it talked about they're doing CRISPR technology. This is absolutely terrifying. CRISPR technology to tweak the genes of the plant so as to make it glyphosate resistant without having to put a bacterial gene in there.

SS: And in CRISPR they've concluded that... regulators have concluded that CRISPR is not a GMO technology, which is pretty shocking to me. And therefor they can do CRISPR, and they can produce a non-GMO glyphosate resistant crop by tweaking its own genome and not bringing in somebody else's genome. And the first thing they did in tweaking it was to get rid of that glycine residue.

SS: They knew, “First we've got to get rid of glycine.” And then that takes a hit on the enzyme. The enzyme doesn't work as well because it's got alanine there. It's got that extra methyl group that's in the way. The same problem that glyphosate causes. But it's a much smaller problem because methyl is much smaller. [inaudible 00:17:44] so it's a smaller problem. So then they design... it's fascinating the paper because they said-

JM: Is this a recent paper?

SS: ... yeah, a recent paper. Now I don't know the title and I don't know the author.

JM: [inaudible 00:17:56] we can put it in our notes.

SS: Yeah, Dow DuPont, really fascinating.

JM: Are they the same company or are they two different ones?

SS: I think it was the two of them together. I think they joined.

JM: Dow DuPont is unquestionably probably as evil as Monsanto.

SS: I know I'm learning that.

JM: Oh my gosh there's some incredible... they made Teflon essentially. Actually they didn't make it. I think it was Dow that invented it. And then Dow had some integrity and realized, "No, this thing is too dangerous. We have to stop making it." So DuPont bought the rights to it or license and started making it. They're the ones that are perpetuating this craziness.

SS: Wow.

JM: Yeah, but anyway. I'm sorry.

SS: That's interesting.

JM: It's interesting.

SS: Yeah. So anyway, so they figured out that if we change some other amino acids over here and make a little more room to account for the methyl that's in the way... because the enzyme was only hurt by a small amount. Glyphosate was completely dead in the water because glyphosate could no longer substitute, whereas the enzyme just had a little bit less room. So it's not nearly as drastic for the enzyme as it is for the glyphosate. And so the easy explanation is that the glyphosate could no longer substitute because there was alanine instead of glycine. There's no code. It can't work. And that's also true for E coli. E coli had a natural mutation where they lost the glycine. And there's a lovely paper that shows that glyphosate at incredibly high levels, they had a whole range, and the enzyme just worked beautifully all the way through. It never got affected by glyphosate, whereas the original molecule with the glycine there got clobbered by the glyphosate, and very consistently worst with more. So it just basically out competing the glycine in assembling in that protein to mess it up so that it can't work.

SS: And they also had another paper talked about 1,000 different analogs of glyphosate. They basically change one atom and put something else in there and make a new molecule. And made all these different molecules that were very similar in shape and biophysical characteristics to glyphosate.

JM: I'm going to edit this out. I'm just wondering. Where's your microphone? Did it slip? Okay. Yeah. That's okay. We'll see, hopefully. Sorry about that. [inaudible 00:20:13] I thought, "I don't see her microphone." A little bit over here so I can still see it. If I can just move this up there. I think maybe the problem is you put it in a little place that it could slip. Put it on here. All right. Okay.

SS: So they did a thousand different analogs of glyphosate and none of them worked. Only glyphosate worked. And they were puzzled. I would think they would have said, "Hey, maybe substituting for that glycine..." it's like it's screaming at them and they don't see it. That's what's so fascinating to me. So anyway, so once I realized the significance of this... and I even found a paper that talked about phosphate binding... there's a lot of interesting papers out there. It was specifically interested in the big class, there's several different classes, of proteins that bind phosphate, because phosphate is super important. It's in a lot of different molecules like ATP and ADPH, FAD, FMN, GTP I mean it's just... glucose-6 phosphate, G6P. All these different... and PEP of course which is the one that gets in the EPSP synthase. In fact, phosphate is added to molecules to make them more reactive. It's sort of a step that's done in biology. Like glucose-6 phosphate in order to be able to make them more reactive so they can go on do other things.

JM: And plants do it too. It's typically one of the minerals that are added in fertilizer.

SS: Phosphate is definitely a very important fertilizer, which shows you that phosphate is super, super important in biology. And ATP of course has three phosphates attached to the adenosine. And it's that third phosphate, the Gamma phosphate that gets taken off to make ADP, and that's an energy driver for many. For example, myosin. The muscle protein. Myosin contracts by converting ATP to ADP. So it binds the phosphate of ATP. And at that binding site it has a highly conserved... actually it has three highly conserved glycine residues. But the middle on in particular very, very important. If that gets mutated the protein is dead in the water it can't contract. Same story all over again for myosin.

SS: Myosin is an important contractor protein or move the feces through the gut. So if myosin gets paralyzed you're going to get peristalsis. You're going to get CBO because things get backed up. Small intestine bacterial overgrowth. You get a lot of problems with your gut because the myosin is not able to contract. You get constipation of course. And these are all connected to Autism, these problems. And so I think the myosin in the gut is being poisoned by the glyphosate in the same way that the EPSP synthase in the shikimate pathway is being poisoned. Because of this glycine at this place where phosphate is supposed to bind sets up a beautiful environment for throwing glyphosate in place of glycine in the protein itself.

JM: Well, I just want to interrupt here to not get you too discouraged because there are some good things we're going to talk about. But it's important to understand the basics of the problem, the foundational reasons why it's brought so we can understand solutions.

SS: Yeah, right. So that's myosin. I think myosin even in people who have chronic fatigue syndrome I'm suspecting glyphosate is getting into their muscles and disrupting myosin. So you can start to think about-

JM: Have you discussed that before?

SS: ... I don't know.

JM: Yeah, because you neglected to mention that you're writing a book about this.

SS: I am writing a book. I'm having a good time. It's a very big challenge to try to write it. I want to cover the chemistry, and I want to keep it from being so difficult that nobody wants to read it. So it's a very difficult task that I have.

JM: But it's important to get that right. Because you have to understand that there's strong reality behind these assertions.

SS: Right, I mean, I think if I don't cover the chemistry then I'm not doing justice with it.

JM: There's not many people who can cover it as well as you.

SS: Thank you. So it's fun, and I'm very preoccupied with the book. I'm having lots of fun with the research because I'm still not done with the research of course, and I keep on coming upon new things. But there was this one paper that looked at all the different proteins that bind phosphate, various kinds of phosphate-containing molecules. And looking at the site that binds phosphate to see if there's a generalization of what goes on there. And what they found was glycine. Absolutely at least one sometimes three glycines, really important to give room, because glycine is a small molecule. You need to have room for that phosphate. And then two was to have strategically positioned positive amino acids. Amino acids that have a plus charge so that they can attract that phosphate to come into that side. So those two principles apply to so many different proteins. And in all of those cases it's a set up for glyphosate. So I think to look at those particular proteins that bind phosphate and that have that highly conserved glycine, and you can look for mutations in the glycine. Sometimes you don't find any because I think it's so essential that you would die.

JM: Yeah, you're dead. You don't get that one you don't come out alive.

SS: Right, exactly. And then you look for different diseases that are connected to defective function of that particular protein. And that's how we can discover a lot of... you can form links to explain why glyphosate would cause various diseases such as Autism. Autism is a good example.

JM: Well that's got to be a challenge doing that type of research because... I mean, in some ways it's easier than the others because you're building on other people's advancement of the science. But you're discovering new science. It's a whole different level of investigative research journalism.

SS: Yeah, and it's interesting to do it entirely by reading papers. And I think not enough people are doing that work. I think that kind of work is not easily funded to just pay somebody to read papers and write papers.

JM: No. I mean, because everyone has responsibilities. And it takes a lot of time. It takes hours a day every day.

SS: But also, they always want you to be doing new research. You're in a lab, you're doing some study. But there's all this research out there and all these papers published. If someone would step back and really absorb all of that knowledge and then synthesize a story out of it you can really make a lot of headway on explaining things in how biology is working. It's a very good example with this phosphate binding thing because this gets into the NADPH oxidase problem. And what's really interesting to me... and this is also linked to Autism... the red blood cells have a tremendous responsibility to keep NADPH in its reduced state. They use glucose-6 phosphatedihydrogenase. G6PP.

JM: Well, let's stop here. Because this is really important topic. And you and I... I mean, we could keep on going forever on this stuff. But most everyone watching this doesn't know what NADPH is.

SS: Kay, good.

JM: So let's just sidetrack there and help them understand and appreciate this. Because it is just about as important as it could be.

SS: Yes, absolutely I agree. And of course it goes to glutathione as well.

JM: Well you need it to catalyze glutathione.

SS: Yeah. So glutathione and Vitamin C are really-

JM: And recharge it too.

SS: ... exactly. So these are important antioxidants, glutathione and Vitamin C. They're really important for mopping up all these free radicals that are going to damage tissues, a and they have to be interest reduced state in order to be able to work. In order for them to get into the reduced state you need to oxidize NADPH.

JM: And reduced state means they have a surplus or at least an electron they can donate to neutralize free radicals.

SS: Right. And so you need to give that electron back to them so that they can donate it. Ti's like they gave away the football. You've got to give it back to them before they can give it away again. It's the NADPH that keeps them in that reduced state by essentially giving up its electron to them by becoming NADP+. And then it's the responsibility of glucose actually, the sugar, to bring it back down, because glucose is a really good reducing agent. So it's going to take the NADP+ and turn it back into NADPH.

JM: Through?

SS: G6PD, glucose-6 phosphatedihydrogenase. Which is super important.

JM: And that enzyme defect genetic snips is the most common genetic disease in the world.

SS: I know that and that is extremely interesting to me. It makes sense because it's so so important and because it's under siege. I am suspecting that mutations are happening more often in proteins that are under stress. That there's some mechanism that's able to recognize this protein is in trouble, and it needs to be tweaked. And so to sort of up the ante on the [inaudible 00:29:10]

JM: Interesting theory.

SS: Yeah, and that's particularly interesting because I found a paper that talked about the mutation rate of all the different amino acids. And they found two... I forget. I think it was aspartate that was the most commonly mutated. And they could explain it based on the DNA that there were certain cytosines that were more susceptible to mutation. It could make sense in terms of the DNA that it would be more mutated because it was code.

SS: And then they say glycine was number two. It had the most mutations out of any other amino acid. And they didn't know why. They couldn't explain it. And furthermore they said next to glycine next to glycine, two glycines in a row, was especially susceptible. And that's very interesting because glycine is small. Glyphosate has to have room-

JM: It's the smallest amino acid.

SS: ... yes. And glyphosate needs room because it's got that extra methylphospyl group. So, if you've got glycine next door you've got more room to push your methylphosphyl group. It's going to be more susceptible to glyphosate substitution.

JM: Let me just add to the other important variables with NADPH is that it's absolutely required to make fatty acids. Making fatty acids is the biggest consumer of NADPH. And then nucleic acids, the elements of your DNA, and cholesterol. You can't make cholesterol without NADPH.

SS: You are telling me things I didn't know and I need to go back and-

JM: Oh, it is just so massively important and almost virtually no one appreciates.

SS: ... so interesting.

JM: Yeah. And you know, I think this is maybe one of the reasons why statins work, because they lower cholesterol synthesis by inhibiting HMG-Coenzyme A Reductase. So you're not making this cholesterol and you need NADPH in that reaction. So you actually increase NADPH.

SS: You get more NADPH by virtue of-

JM: Which is a good thing.

SS: ... right. Which is such a good thing that it's worth sacrificing your cholesterol, right?

JM: Well, I mean I don't think it's a good thing overall. But some people do appear to receive clinical benefit from taking statins. But it's not at all related to cholesterol reduction.

SS: No, I know that. In fact, they always say it's because of the antioxidant capability. Which would be true if-

JM: Yeah, that's my theory. I haven't read it. I just put it together.

SS: ... I love that theory a lot.

JM: And then Steve Montgomery goes into the inflammatory component with the toll receptors. And Malcolm Kendrick believes it's by increasing nitric oxide. So, I mean, there's a lot of ways. I don't think any of it's proven one way or the other, but we know it doesn't work by lowering cholesterol.

SS: I know.

JM: And that's how you got into this through your husband, right? He was put on statins.

SS: That's right. Yes. And he's been very well without the statins for many years. So that's the thing.

JM: Yeah, so let's get back to the NADPH.

SS: Glyphosate gets into the blood, that's big.

JM: What's the connection between... is it the Nox in the red blood cells or is it the NADPH?

SS: Well, so the-

JM: Oh, the Nox we could... Nox is NADPH oxidase, and it consumes NADPH's fuel. So it's a good thing. You need it, if you don't have it you're going to be dead from some type of infection. But if you have activated too much boy you've got big problems. No more NADPH.

SS: Right so if Nox is running too much it will.

JM: So is Nox the issue in red blood cells?

SS: Well it's the G6PD because it's your glucose-6 phosphatedihydrogenase is big time in the red blood cells. And I think it's their big responsibility to keep all those antioxidants in their reduced form.

JM: And do you know why that is?

SS: Do I know why that is?

JM: Yeah.

SS: You seem to know.

JM: Well, I think because red blood cells are one of the only cells in the body that don't have mitochondria.

SS: Oh yeah, so [inaudible 00:32:40]

JM: So the only way they can create energy is through anaerobic fermentation or glycolysis which is where G6DP comes into play.

SS: Yes, that's right. They manage glucose. It's interesting they have a lot of oxygen. But they don't use it. And they don't have any mitochondria, which is so fascinating. And that's probably because it's too dangerous to have a mitochondrion that's doing what they do, and that's why they do what they do. You know? So it makes sense.

JM: Because it is odd.

SS: Yeah, glucose is a very good reducing agent. So glucose is busy turning NADP+ into NADPH through this enzyme. Which has two phosphate binding sites.

JM: G6PD has two phosphate binding sites.

SS: Yeah, it's really interesting. It has the spare. It has a... it binds to NADP+ to get rid of NADPH and then it has a second NADP, plus it binds on its tail. It's really interesting. I found a wonderful paper where they were puzzling over that. It's like, “Oh, why is it binding over here?”

SS: And they had a theory which was cool that it's sort of a pipeline. Sit got the other one ready to go. And as soon as it got this one done it could kick it off and get this one on. It's go tit ready to go. So it's sort of like a staging room where the second one comes and on so that it's very convenient to get it where it needs to go to very efficiently convert. And so it's got two places with the potential to get messed up by glyphosate.

JM: Wow, interesting. Man, I did not realize there was yet another mechanism of actually lowering NADPH.

SS: Yeah, right. It's huge I think.

JM: Yeah, but virtually no one would get it because they don't know what NADPH is, or the significance of it.

SS: I know. It's not spoken about enough. Ti's really a very, very important molecule.

JM: And part of that reason... if I can just give you a little backstory because I'm investigating this really carefully for one of the next books I'm writing which is on EMF and it plays a big role... is that it's been known for... Actually Otto Wherenberg. I think he got one Nobel Prize. But he was nominated for many others, but he got one in 1930. He discovered NADP.s it's been known for a long time. You know, 80 years we've known about NADP an NADPH. But, it didn't really start to come into clinical appreciation until the 21st century when they discovered the [inaudible 00:34:58] longevity proteins which consume NAD. So then all this focus has been on those molecules. So it's not really widely appreciated because of that. Because it's just, "OH, it's just NAD. You need it in mitochondria. It doesn't do anything else."

SS: It's interesting how certain molecules get so much attention and certain other ones can get neglected. Biology is just such a huge space, and it seems like the research community gets hung up on one thing and drills down deep and neglects something else. And that's certainly true for sulfate. I felt sulfate is definitely underappreciated.

JM: Do you have any reasons why you think it might be?

SS: I think it's a paranoid reason that Monsanto knows that glyphosate messes it up.

JM: Well, that's a corporate financial motivation. But what about the broader research community also doesn't have this appreciation. So why do you think that is?

SS: I don't know why. Do you have a thought?

JM: No. I mean, I'm not... I don't know. Maybe it's just because there's so many things going on that they're out in their own little world and they just don't get it. And there really hasn't been any breakthrough development in technology like autophogy or the discovery of nitric oxide or this Nobel Prize work. So there's a lot of focus on that, where there really hasn't been something comparable on sulfur.

SS: Yeah, I mean it should be because I find it really fascinating. And what I learned early on, again before glyphosate, was that there are all these molecules that are sulfate in transit. Really important biological molecules that are sulfate in transit. [inaudible 00:36:30] is one of them. [crosstalk 00:36:33] and cholesterol sulfate of course is one that I'm very fond of. And then there's all these neurotransmitters. Dopamine sulfate.

JM: Do you think that their primary role I to just transport? Or is there another benefit or role for that actual transport?

SS: So one thing is when you sulfate these molecules they become more water soluble. It's categorically true for all. These molecules are all sort of not very water soluble. You put a sulfate on and water... so that makes sense. So in a sense they said, “Oh yeah, well this is why. Therefor that's done. We understand."

JM:
So it can travel in the blood and get distributed.

SS: Yeah, so you can move it around without you having to package it up inside LDL or something like that because it can just go freely in the blood if you sulfate it, which is important, which is great. But I think there's another purpose which is to transport the sulfate. That they are actually carrying sulfate on their backs and delivering it to wherever they go. Because they have to let the sulfate go before they can go into the cell. They're not active when they have sulfate on them. Pretty much all of them become inactivated when they have sulfate.

JM: So it's dual purpose.

SS: It's a beautiful mechanism because basically sulfate's hard to transport because it gels the blood. That's the feature of it. It gels the blood along the lining of all the capillaries. It's stuck to the capillary lining all over the blood vessels. You've got these sulfates sprinkled all over. And those sulfates are creating this gelled water like gelatin that coats the edge of the blood vessel and make sit very slick so the red blood cells can just slide right through the capillary without resistance. It's very, very important.

JM: How does heparin come into this?

SS: How does what?

JM: Heparin.

SS: Heparin, yeah. Well, heparin sulfate is what it's in the lining of those blood vessels. And heparin is the most highly sulfate molecule known to biology.

JM: Interesting. That's a nice little tid bit.

SS: Yeah. And heparin can actually supply sulfate to the capillary wall. It actually can stick there and supply it. So heparin becomes important in that respect. But the sulfate transport then... so I think the neurotransmitters I think one of their purposes is... and perhaps even one of their most important purposes is to transport sulfate can carry it to the site where they drop it off. And their signal is, “Hey, I'm going to give you your sulfate” in a sense.

SS: And so all these sex hormones, you know testosterone, estrogen, estrone sulfate, testosterone sulfate, melatonin sulfate they're all sulfated in transit, of course cholesterol, Vitamin D. So it's all of the sterols and all of these derivatives the aromatic amino acids that glyphosate is disrupting. So you're going to have a deficiency in the aromatics because of the blockage of the pathway and the bacteria. And that's going to have a deficiency in sulfate transport.

JM: Man, that just ties it all together. Okay. Little break, switch this [inaudible 00:39:36] jean jacket no problem. Just make it... that's good. Okay now you can go back. That is brilliant. It makes so much sense. Just like you said earlier it's a giant puzzle. It's a 10,000 piece puzzle and you just have to put all the pieces in and starts to make sense.

SS: It's very thrilling. People who loves puzzles can appreciate the thrill of when I find a new paper and it's like, “Oh my God, this fits so perfectly.”

JM: Wow, man. Are there any more puzzle pieces you want to provide us before you go into some of the exciting solutions? Because it's important.

SS: Yeah, I can tell you one more thing about the sulfate. So sulfate gets activated by a process using an enzyme that converts it to something called PAPS, Phosphoadenosine phosphosulfate.

JM: PAPS.

SS: Phosphoadenosine phosphosulfate. PAPS. And that enzyme-

JM: I'm assuming that's adenine, correct?

SS: It's got ATP. So that enzyme binds to ATP molecules. That means it binds to phosphates, right? It produces something that's got two phosphates in it, PAPS. It actually takes... it steals the terminal phosphate from one of the ATP molecules to make it ADP and it sticks it onto the other ATP molecule. And it takes off two phosphates and sticks sulfate in place of it. So it does a lot of stuff. It's got two parts to it, that enzyme. And it's got a binder region. And so some animals have those two enzymes separated. But we've got them combined in one giant enzyme.

SS: PAPS synthase, which has all kinds of incredibly important glycine residues in it many of which are associated with these phosphate binding sites. So that enzyme which makes the activated form of sulfate is likely disrupted by glyphosate for the same reason as the G6PD would be disrupted. And therefor if you can't turn sulfate into PAPS you can't add sulfate to anything.

JM: I think I may have missed... what does PAPS do?

SS: PAPS is the activated form of sulfate. What happens is when something... so there's these sulfitases. Those are enzymes that take sulfate off of things. And then there are these sulfotransferases which put sulfates onto things. And in between the sulfate gets converted to PAPS. Before it can get put on it has to be energized by being bound to ATP in order to be able to get onto the...

JM: So this would be crucial for the sulfate transporter.

SS: ... sulfate transfer, yes. If you transfer from point A to point B is going to be blocked-

JM: If you don't have PAPS you're in trouble.

SS: ... yeah. If PAPS isn't working. And that's very, very interesting. Another thing that's interesting is there's a paper on E coli. And they exposed these E coli to glyphosate, using... they did a big meta analysis of their different proteins to see which ones were suppressed and which ones were activated. They had a big appendix with a whole list of things of all these proteins that

were affected.s many, many proteins were disrupted by glyphosate in terms of changing their activation state. Whether it made it or not.

SS: And a whole bunch of proteins that were ATP binding sites there were about maybe eight or 10 of them. ATP binding sites were stimulated by glyphosate. They increased production, which is because, I think, they're defective. They're not working because they can't bind because of the phosphate problem. And therefor you have to make more of it. So I think that's very interesting too. It's just another little piece of the puzzle that supports the idea that enzymes that bind phosphate are in trouble with glyphosate categorically. And I haven't found... I don't think I've found any enzymes that bind phosphate that don't have at least one glycine at that phosphate binding site.

JM: Wow. You've really expanded the understanding of the damage that glyphosate does, because it's not restricted to the shikimate pathway. I mean, that's part of it for sure. And it's not restricted to binding these trace minerals. Especially molybdenum. I didn't understand the connection.

SS: Yeah, well I'm not sure about molybdenum because I have not been able to find a paper that explicitly says it binds with molybdenum. However, molybdenum [crosstalk 00:43:53]

JM: I thought glyphosate was known that it was one of the main ones that bound.

SS: I don't think that it said.

JM: No, it's zinc.

SS: But, that doesn't mean it doesn't. It just means they haven't looked.

JM: Okay. So what is acknowledged?

SS: The category is +2 cations and molybdenum is +2 cation so it's in the category. But no one has explicitly said, as far as I can tell, that it is one. But, I've looked because I've wanted to find it.

JM: So, that other +2's would be calcium, magnesium.

SS: Yeah, magnesium, zinc and cobalt and manganese are most affected.

JM: Do you think the fact that calcium magnesium typically... I mean, you had mentioned in any of your preceding information it's not an issue because we have so much magnesium and calcium?

SS: Yeah, I think those are sufficiently-

JM: Sufficiently in excess.

SS: ... yeah, the more migrant ones like cobalt and manganese are ones-

JM: Oh manganese. I was confusing manganese with molybdenum. Manganese is the big one. That one is well done.

SS: ... that one is really interesting.

JM: What's the connection with manganese now? Because it's obviously through some critical enzyme function that we're not appreciating.

SS: Yeah, manganese is really, really interesting. It starts with the lactobacillus. Because lactobacillus are unique and interesting in that they use manganese for their antioxidant activities. Manganese is an important part of their antioxidant management, protecting themselves from oxidative damage. That's unique to them. They don't use iron for example. They use manganese instead of iron, whenever iron is normally done.

SS: And they get clobbered by glyphosate. Bifido bacteria and lactobacillus are very susceptible to glyphosate.

JM: Wow. So that's independent of the shikimate pathway.

SS: Yes.

JM: Wow.

SS: And of course lactobacillus are really, really important in the infant because they drink milk basically. And it's been shown that when you have low lactobacillus you can have anxiety, depression. These things are connected to lactobacillus deficiency. Manganese is what's a catalyst for the conversion of glutamate to glutamine.

JM: Okay. Yes, so this is the big one. So listen carefully.

SS: Yeah. And Autism has been very clearly linked to high glutamate and low glutamine in the blood. There's a study that looked at all the different amino acids in the blood in autistic kids compared to controls. And they found everybody was statistically insignificantly different. Except for two. Glutamate and glutamine. Way too much glutamate, way too little glutamine, for the autistic kids. Which suggests a manganese deficiency problem. Because that could explain it because glutamate is not getting converted to glutamine.

JM: How about the other way around? You would think most of the glutamine is coming from the diet. I believe it's the most common amino acid in our diet is glutamine.

SS: Oh really?

JM: I'm pretty confident it is. But you're discussing the reverse. So what about glutamine to glutamate? Is that an issue too?

SS: I don't know. I haven't researched that.

JM: Okay. So it's the other way around. So how do we get the excess?

SS: And the excess glutamate is a neurotoxin so that's a problem.

JM: Right. Normally you have the glutamate... and we'll talk about how we get the glutamate... but it's converted to glutamine which is beneficial. Unless you get too much because it activates [inaudible 00:47:03] too which inhibits autophogy. So the timing is key, so key. But if you... it'll increase the glutamate because it's not being degraded. But how do you get too much glutamate.

SS: How do you get too much?

JM: Yeah.

SS: You mean like in your diet? Well, the MSG.

JM: Well, I get that. T's one obvious one. But is there some metabolic pathway that accumulates?

SS: Well, if you can't turn it to glutamine... in fact in the brain-

JM: But if we're eating MSG it's not going to be an issue?

SS: No, no. It's in all the proteins. There's plenty of glutamate in the proteins.

JM: Glutamate is in the proteins?

SS: Yes. Glutamate and glutamine are both amino acids that are coding amino acids.

JM: Oh man. I feel foolish. Glutamate is an amino acid?

SS: Yes.

JM: It's not one of the 20.

SS: It is.

JM: Glutamate?

SS: Yes.

JM: How can I not remember that? Glutamate is an amino acid.

SS: Yes, it is. And glutamine is too. Both of them.

JM: Okay. Wait, does this have another name? Glutamate.

SS: Glutamic acid.

JM: Glutamic acid. That's why. Okay. Like aspartic acid, glutamic acid. So that's where I was messing it up. It's like, “You know, I don't remember that from biochemistry."

SS: Oh, that's right. Glutamic acid and aspartic acid. Both of those are negatively charged. Both of those are actually [inaudible 00:48:19]

JM: Now I'm getting it. Okay. Now it makes sense. Because I mean, I didn't make the connection that glutamate is just another term for glutamic acid.

SS: Yeah, that's confusing it depends on the ph as far as whether it's an acid or not, you know?

JM: Oh okay. So that's how we get it.

SS: Yeah.

JM: That makes sense.

SS: Right. So it's interesting with the neurons because the neurons use glutamate as a neurotransmitter. It's very, very important for transmitting signals. They release the glutamate into the synapse, and then the [inaudible 00:48:47] very quickly take the glutamate out. They clear it really fast. And they convert it to glutamine using manganese.

JM: Is there a specific enzyme they use?

SS: Yeah. Glutamine synthase. And I think that glycine uses that as well. But id have to go back and check.

JM: Probably.

SS: Yeah. Many of the enzymes do. Some of them more than others. Anyway, glutamine synthase produces glutamine. And then the glutamine is shipped back to the neurons. They take up the glutamine and they convert it glutamate and hydrogen inside of these little canals to protect, because they know glutamate is very reactive. So they have to keep it safe. So there's a whole mechanism that prevents glutamate from being out in the general environment because that would be bad. And that's through the astrocytes. They convert the glutamine back and it gets converted inside the neurons. So, if the astrocytes can't convert glutamate to glutamine you're going to get neuro excitor toxicity. And glyphosate has been shown to cause that in the hippocampus in the mouse. They've done studies where they've exposed them to glyphosate and they've even done in vitro studies. In both of these, in the animal as well as in vitro, they've shown that glyphosate excites the [inaudible 00:49:57] receptors. Causes this excess glutamate production.

JM: And doesn't empty it.

SS: And blocks the conversion, yeah. And blocks the conversion of glutamate to glutamine.

JM: So all because of the manganese.

SS: Yeah, the manganese blocking the-

JM: The chelation of manganese from the body tissue stores and also from the diet. Then this glutamine synthase will not work well.

SS: Right. And it's even more interesting to me because Anthony and I did a paper on manganese, which is really fascinating to read about manganese. There's a condition called manganism, which is very much like Parkinson's disease.

JM: It's too much manganese.

SS: Too much manganese in the Brian stem. It's caused by lack of [inaudible 00:50:37] breathing this air that has this manganese. It goes into his nose, it goes up into his nasal cavity and it travels long the olfactory nerve into the brain stem. And of course then it's too much manganese. It's very, very interesting. Manganese has an unusual property that it can travel very well along nerve fibers. And so, I think when you look at glyphosate disrupting the... so first of all glyphosate disrupts the [inaudible 00:51:01] enzymes in the liver [inaudible 00:51:02] enzymes which are really important in making bile acids.

JM: And detoxification.

SS: Yeah, they have a lot of specific... also Vitamin D activation. So that's a big problem with [inaudible 00:51:12] enzymes. That's been shown in rat studies a tremendous drop [inaudible 00:51:15] enzymes glyphosate. And the enzymes have glycine defenses. They've got this beautiful paper that talks about this sequence. Ti's got three glycine-

JM: It's this beautiful puzzle.

SS: ... it's really amazing. They also have hemme. So that's another issue with the hemme supply.

JM: Just a really good analogy here is a favorite of Bob Miller's is like playing 3D chess underwater.

SS: Yes. It's so amazing you just go down these threads, you go down these rabbit holes all over the place. You know, it's really quite incredible. So let's see, where was I going with that? Yeah, so then the bile acids can't be synthesized.

JM: Yeah, so this was from excess manganese now?

SS: Well, we're getting to manganese. So we start with bile acids. Bile acids can't be synthesized because the [inaudible 00:51:55] enzymes are blocked. And if the gall bladder is getting exposed to glyphosate that's going to disrupt myosin, which means the gall bladder can't contract, so it can't release the bile acids. So you get bile acid deficiency. You get insufficient bile acids. And that's

been shown to actually be true in mouse [inaudible 00:52:13] they have low bile acids. So that works, and that connects with it. Manganese normally goes to the liver, binds to the bile acids and gets carried back into the gut bound to the bile acids. And then it eventually gets taken up into the [inaudible 00:52:27] and delivered through the lymph system eventually to the vein here, gets into the heart and goes through the general circulation.

SS: So the chylomicron is a transporter of manganese in the blood to deliver it to the entire body. But it depends upon the manganese going into the bile acids, which won't happen if the bile acids aren't flowing. So you get the manganese building up to toxic levels inside the liver because it's not moving out. And then the liver ships it out on the vagas nerve up to the Brian stem and causes ADHD and Parkinson's disease. I suspect from too much manganese in the brain stem, and too little in the cortex because it's not coming in the blood supply. So you get an imbalance where the manganese is distributed.

SS: And I even suspect that Lyme disease species likes manganese. And I'm suspecting it makes a positive role to redistribute manganese. It's available in high concentrations, for example, in the gall bladder. Picks up the manganese in the gall bladder and ships it around to the body helping to distribute where it needs to go. But of course causing disease at the same time, which is often the case. I think a lot of these pathogens are often doing something useful for us in the process of causing symptoms.

JM: And then when we try to eliminate them it's actually counterproductive.

SS: Yes, you're right.

JM: It's so complex. So this is beyond fascinating. It's amazing.

SS: It is amazing. I mean, it really keeps me going. I'm so depressed about the world's situation right now in so many ways. But at least I have my papers that are so much fun.

JM: Yeah. And it is depressing. Especially everything that's going on. But we do have some good news. Maybe this is an appropriate time to discuss the good news that happened with Bayer that purchased Monsanto.

SS: Absolutely. Bayer doesn't think it's good news.

JM: It's good news for those who value health, that's for sure. So, as we're reporting this they just had a recent victory in litigation that essentially resulted in a cumulative drop in Bayer stock by one third. They have lost nearly $30 billion, with a B, $30 billion. So that's a big number. I think it's either... I think there's 11,000 lawsuits pending.

SS: 11,000 more.

JM: Yeah, they're pending. So it is potentially possible... unless they pull... I mean, they still have $60 billion so they can buy off a lot of juries and legislators, who knows. But it's potentially possible they can go bankrupt.

SS: I think so.

JM: It is definitely on the brink of possibility. Because you've done a magnificent job of compiling the damage of glyphosate. Not world is beginning to understand this widely. So eventually glyphosate's going to be gone. Just like DET.

SS: I sure hope so.

JM: It will.

SS: I hope it's in my lifetime.

JM: But it doesn't matter because the same evil companies can come up with a derivative of it and go out and take another 10, 20, 30 years.

SS: I really hope we can get the message out so well that people would recognize that we need to go back to renewable, sustainable-

JM: Regenerative agriculture.

SS: ... yeah, it's absolutely the solution.

JM: 70% of the food that's produced in the world is produced by small farmers.

SS: Yeah, that's good news. Since most of them aren't in the United States.

JM: No, but you can do it yourself. We had victory gardens in the United States in World War II.

SS: It's great. Ti's really a super movement. I really feel like any young person who's looking for a career they should go into regenerative agriculture. It's so important. Absolutely, if we don't do enough of it we're going to be gone. Our species is going to die out.

JM: Yeah. Well, in so many different ways. Especially the planet with global warming. A lot of people may criticize me for believing it but I do. Regenerative agriculture is a proven strategy, really one of the most effective to store the carbon in the Earth where it belongs.

SS: I need to talk to you about global warming and glyphosate. Because [Robisco 00:56:30]

JM: Robisco?

SS: Robisco. Have you ever heard of Robisco?

JM: No, I have not.

SS: It is the most common protein in the world.

JM: Robisco?

SS: Robisco. And it stands for something and I don't remember what it is.

JM: Okay. And it's a protein people eat?

SS: It's a protein that is in plants.

JM: Oh it's in plants. Okay.

SS: Not in humans. Humans don't have Robisco. It's a plant protein. And it's a crucial protein in the pathway that takes carbon dioxide out of the air and turns it into organic matter.

JM: Wow, the mechanism of how we prevent global warming.

SS: Exactly, and glyphosate has been shown in multiple papers to suppress it. And that makes sense because it binds phosphate.

JM: Gosh, you just keep oncoming up with those.

SS: That's what I mean. It's like, "Oh my God, this too." So I think glyphosate is a big player in global warming. It's preventing the plants from taking carbon out of the air.

JM: Well, it's 5 billion pounds a year. That's a lot of glyphosate.

SS: Yeah, and every plant that's affected the Robisco gets disrupted and carbon dioxide... whenever an enzyme is not working well it sub strait builds up, right? So all these plants are not able to use their Robisco effectively. Carbon dioxide builds up. It's simple math. You're going to have higher carbon dioxide in the air.

JM: So let's discuss remediation. So glyphosate is in the soil. Thankfully I believe the micro rice and fungi tend to destroy it and metabolize it, get rid of it. Have you looked at the metabolism of glyphosate in the soil and its half life?

SS: Well it's interesting because there are very few microbes that can break it down. It has this CP bond that's very difficult. And it even-

JM: It's the carbon-phosphate bond.

SS: ... yeah, carbon phosphate. There are some enzymes that can break carbon phosphate bonds into other molecules but not necessarily in glyphosate as it's an unusual version of a carbon phosphate. It's so unique that the microbes don't know what to do with it. So it's only a few of them. One of the ones that can break down is aceto bacter. And aceto bacter is in apple can cider vinegar. So, I love apple cider vinegar.

JM: I have it every day myself. I go through about a quart-

SS: Oh, good for you.

JM: ... is it a quart or a pint? I think it's a quart every two weeks or so.

SS: Yeah. We make salad dressing. We have salad for dinner and I think it can actually help you to break down whatever glyphosate is in your mouth, because it will get right to work turning glyphosate into useful phosphorous. It completely gets rid of it.

JM: So the bacteria they use to ferment the apples to apple cider vinegar is actually still present if it's not pasteurized.

SS: Yeah, you have to get organic uncultured.

JM: Yeah, we make it too. We have something called keto cider vinegar. It's got the mother in it. It's not pasteurized. Those organisms can actually... I did not know that. Did not know that.

SS: Yeah, I think that's really interesting. So I suspect... a lot of people have said apple cider vinegar is helpful. I suspect one of the reasons is because of its microbes. So don't buy it if it's been pasteurized. Get it raw. It's really great. Interestingly like pseudomonas areuginosa is another microbe that can break down glyphosate. And it's a major pathogen in hospitals. I think it's flourishing because of this feature that it can break down. So it's actually helping you out by clearing glyphosate.

SS: I even suspect... I don't know if you've been looking at Cipro

JM: It's fluoroquinolone. It's the most common fluoroquinolone.

SS: Yeah. Fluoroquinolone is a class and fox... getting foxed? You know all this bizarre symptoms that are happening long after you've taken your antibiotic. They're getting all kinds of weird problems with their joints. Their Achilles tendon. They have the aorta breaking open. I mean, all kinds of weird things are going on. They look to me like glyphosate poisoning. The symptoms are showing up long after you've taken this medication. So my suspicion is, I'm trying to figure out how does that work?

SS: At first I was wondering if it was glyphosate in it but that's not going to be enough to cause any problems. Because that's just a short term [inaudible 01:00:40]. It's lasting long after-

JM: Yeah, yeah. It's definitely long term.

SS: ..., so it's some change happens at the time you take the antibiotic. Well, what would be changing? Well the antibiotic kills anaerobic bacteria. And those are the bacteria that happens to be [inaudible 01:00:54] that breaks down glyphosate. So it's preferentially killing off the bacteria that metabolizes glyphosate. Including Pseudomonas for which it's often used. Pseudomonas, those bacteria are not susceptible to many of the other antibiotics, which is why you have to use

something more powerful. So you're using it to kill off the pseudomonas, and if you succeed you've killed off the thing that's killing the glyphosate. So you're still getting the glyphosate because you've got the same habits. And you're no longer in the clear and therefor it's causing all these symptoms. That's a theory of mine.

JM: Wow, that's interesting. Any more puzzle pieces you want to share with us? Or can we go on to some of the-

SS: I think I'm good.

JM: ... yeah, so some of the exciting immediate things we can do. Obviously eat organic.

SS: Eat organic is absolute. That's the first thing.

JM: That's the foundational basic, because you've got to limit the input.

SS: Right. Getting it eating is a good way to get it.

JM: Before we go there though, what about the fungi? You mentioned two bacteria species. The aceto bacter and the pseudomonas so what about the... have we looked at that? Because I thought the fungi were more effective at breaking it down than the bacteria.

SS: Could be, could be.

JM: I could be mistaken too. That was just my general impression.

SS: I haven't found that, but I should look for it, because I know that fungi actually are very powerful, and they have so many genes you would expect them to be able to do more.

JM: It's like 90%... I believe 90% of the microbial life in the soil is fungi.

SS: Yes, they are so fascinating. I think the microbe [inaudible 01:02:25] are being destroyed by glyphosate.

JM: What's the mechanism?

SS: Well, because they're susceptible. They're just getting poisoned just like everybody else. You lose the organic matter... I mean, I think the soil is losing its minerals, it's losing its organic matter because of the glyphosate. It's going to mess up the organic matter.

JM: Yeah, [crosstalk 01:02:44] it fundamental... which was the Robisco protein. So it can't convert the CO2 from the air in to the carbon in the soil.

SS: Right. It looks like it also messes up nitrogen fixation as well. So that's both carbon and nitrogen. And nitric oxide is a much stronger greenhouse gas than carbon dioxide. It's over 100

times as strong as carbon dioxide. Nitric oxide. So you're going to have more of that if you can't bring nitrogen in.

JM: Methane is in there too.

SS: Yeah, methane's a problem too. And I don't know how... I'm sure glyphosate messes that one up too, but I don't know the details of that.

JM: Well, before we go into how to remediate because I wanted to go back to the glutamate again. Because just a minor tangent, in some way it's important because it gets to NADPH. Glutamine is also essential role for glutathione.

SS: Well, glutamate is actually a component of glutathione.

JM: Oh, it's glutamic acid. It's not glutamate. It's glutamic acid.

SS: Bob Miller actually said glutamine, so you probably remember it from him. He made a mistake, because it's glutamate. Glycine is a member of the-

JM: And cystine.

SS: ... so glycine is important because it could be glycine.

JM: Oh, I know. I know exactly. We've talked about glycine. But there's this interesting..., so it takes glutamic acid and cystine and it makes glutathione.

SS: And glycine. Yeah.

JM: And glycine. So you've got all three. But the rate limiting enzyme I think is glutamate cystine synthase. .

SS: I think so too. Yeah.

JM: Yeah, so there's an interesting nutraceutical that actually activates that enzyme.

SS: Oh interesting, wow.

JM: [Safarpain 01:04:29]

SS: Oh that's fascinating. That is so perfect. That makes sense to me. So safarpain will allow you to build up your glutathione.

JM: Yeah, rather than taking very expensive glutathione supplements, which is really hard to get and very expensive you just take a little safarpain which is pretty cheap.

SS: I love safarpain. And I eat tons and tons of broccoli and brussels sprouts and all those cruciferous vegetables.

JM: Yeah that are full of glucosinolates that convert it with marasinase to safarpain. But that definitely gets to the glyphosate because that's part of the remediation. But I wanted to kind of-

SS: No, but frankly I will take that further because I have been studying glutathione. And glutathione... well, I consider certain molecules to double another role they play is as a storage form of sulfur. And I think that glutathione is one of those storage forms of sulfur because it has the sulfur-containing amino acid cystine. It's very interesting when you look at glutathione, there's an enzyme called Ggt, gamma glutamyl transpeptidase which breaks glutathione down into individual amino acids. And that enzyme is going up in the general population in step with the rise in glyphosate usage in our corn crops.

JM: And I think it's sometimes abbreviated to GGT for short.

SS: Yeah.

JM: Yeah. And hardly anyone tests for that.

SS: It's a really good test.

JM: It's a very good..., and it's cheap. Very cheap. Just a few dollars.

SS: It's actually interesting they don't test for it. [crosstalk 01:05:48]

JM: [inaudible 01:05:49] tested all the time.

SS: ... conspiracy theory because it's a really good... it's a much better predictor of heart disease for example than cholesterol. And it's a better predictor of liver disease than standard ALT.

JM: So why? What is your... and who was it?

SS: So it's fascinating. It tends to go further. So GGT breaks apart glutathione into three things. So those three things are very, very interesting because the cystine can be a source of sulfate if you can oxidize it. But you need to be able to oxidize it.

JM: SuOx.

SS: SuOx, right. So SuOx has hemme, right?

JM:
Yeah.

SS: And so the other two are the precursors to hemme. They make the [pyropane 01:06:27] that is the [inaudible 01:06:29] so you need to take the glutamate and glycine can go in to make the

hemme. Those are the two precursors to make the pyropane. So I think that the glutathione is a storage form of resources that can allow to make SuOx because you need the hemme. And to have the cystine that's a source to oxidize with SuOx to make sulfate. So I think glutathione is a storage form of sulfate in that respect.

JM: That is so beautifully elegant. That's like a roll of glutathione never discussed. We conventionally think of that as the primary way yo detoxify and neutralize these free radicals is glutathione. The most important intracellular antioxidant. But this whole other role is just magnificent.

SS: Yes. So then you have sulfate deficiency because sulfate oxidase is not working in the gut, you know? And then you've got glutathione gets burned up making Ggt I order to try and get the sulfate back. So you wind up with a glutathione deficiency.

JM: Wow. So this is maybe part of the strategy... I'm not surprised you weren't aware of... I just found out yesterday because... I wasn't aware either until yesterday but [inaudible 01:07:44] There happens to be one of the exhibitors who's a really bright guy. His name is Brian [Cornblat 01:07:51] he did his PhD with Jeff Fahey at Johns Hopkins, who is the world's expert in Safarpain. Top guy in the whole world. He was explaining to me because I was told him [inaudible 01:08:02] I thought it was [inaudible 01:08:03] too. But no, no he's talking about the glutamate cystine synthase.

SS: I did not know that.

JM: Yeah, so it's got to be part of this overall strategy. I didn't appreciate is the fact that glutathione is so central in this whole role for glyphosate.

SS: Absolutely. And this makes sense why so [inaudible 01:08:21] saying, "You don't need to because I'm here, and I can become sulfate." So it's becoming a supply for sulfate.

JM: Wow, because there's a lot of anecdotal approaches in this. Some of the presenters here are sharing stories about [inaudible 01:08:36] they're getting incredible results in autism using safarpain.

SS: And I talked with some people here who said that they had people who were sensitive to sulfur that are not sensitive to safarpain. Which makes me think that it is making sulfate through a pathway that doesn't involve sulfide oxidase.

JM: Interesting. So fascinating. Because definitely we're going to discuss sulfur augmentation. But it appears so far maybe a strategy.

SS: I think so. And I really **(I'm really) glad to know that it's something I did tons of [inaudible 01:09:03]

JM: Wow. That is just... that's just [inaudible 01:09:11]

SS: That's really great. Well, we covered a lot of territory.

JM: Yeah. So what we didn't cover is that the exciting. We shared some of the exciting news that Bayer is going down.

SS: Yeah, and this was the lawsuit which he won just a few days ago.

JM: This is not Duane Johnson. This is the one subsequent to that. I don't think it was even the same jurisdiction.

SS: Totally different. Yeah.

JM: It was in Missouri I think.

SS: Yes, I'm not sure.

JM: Yeah. But there's 11,000. So anyways, people are exposed to it. Everyone is. Not everyone can afford to eat organic, which I think... I mean, yeah it's true. But that's a justification sometimes.

SS: I think they have to afford it.

JM: You just have to. Yeah. Or grow your own food

SS: Yeah. There's ways to do it

JM: Buy it from the marker's market. You've got to be careful. You've got to check the sources and stuff. So, Safarpain could be a good source of sulfur. Let's talk about sulfur and then we'll talk about some big clyocine components. So the sulfur?

SS: Yeah. Garlic is something I love.

JM: I didn't realize you were a fan of garlic.

SS: Yeah, garlic is an excellent source of sulfur. Again, it's sulfate sulfur, which I think is healthier than other forms.

JM: Okay. I know you're not a big fan of some of the things that some [inaudible 01:10:26] like MSM.

SS: Yeah. I haven't-

JM: Or DMSO.

SS: ... I just don't... I think they're good actually. I've heard good reports of people taking them. And actually my husband takes MSM. I don't generally likes drugs as you know.

JM: Yeah. Well it's not a drug.

SS: I know. Anything that's not in a recognizable form is kind of a rule of thumb I have. I think I would take MSM if I were very sick. I wouldn't rule it out. And I haven't been able to figure out the chemistry of it, and that's one thing that frustrates me I haven't been able to find the papers that show how-

JM: So it's still got this enigma around it.

SS: ... it turns into... because it's got two methyls, and a sulfur, which is awesome because methyl [inaudible 01:11:01] is an issue. Sulfur is deficient. So it's got all the right ingredients. Presumably it can do something with them, but I just don't understand the chemistry. I haven't been able to find. It's frustrating.

JM: Well you'll figure it out. I'm confident you'll figure it out.

SS: I haven't been able to find papers that talk about how it's turned into sulfate or anything.

JM: The way you inspired me to actually purchase a float tank, which is a giant tank filled with a thousand pounds of magnesium sulfate. You can take an Epsom salt bath. But why not be able to soak in it all the time?

SS: I love that. Have you been doing it?

JM: No, I'm still about two weeks away. I ordered it like six months ago. But it comes in real shortly.

SS: Yeah. I really like that Epsom salt baths.

JM: That's your favorite, right?

SS: Yeah, that's one thing I do. That's the only thing I do besides eating.

JM: And in the bathroom reading your papers.

SS: That's right.

JM: Are you really?

SS: I love a hot bath.

JM: Yeah, I'm just guessing it makes perfect sense because you've got to read.

SS: Right. You've got to have the time. You can't waste any time.

JM: So that's where you get the sulfur. But the other part of this is really crucial and ties directly into what you talked about initially, was the glycine. I wrote a book previously, it's called Super

Fuel with James DiNicolantonio who informed me of the value and importance of glycine supplementation's, which is the smallest amino acid, and it's actually... you can buy it in bulk for really, really inexpensive. It's like $20 is a year's supply. It's a... it sometimes used as a sweetener. It's actually sweet.

SS: That's really interesting.

JM: Yeah. So it can be a sugar substitute. You don't need a lot of it. Maybe a quarter of a teaspoon. If it was by the gram maybe [inaudible 01:12:52] maybe twice a day. But it's a magnificent source of glycine because it signals... I'm a little bit opposed. It's definitely not natural. The natural form ideally would be organic bone broth.

SS: Exactly. And we do a lot of that. I make a big pot of soup every weekend.

JM: Yeah. Because it then [inaudible 01:13:10] connective tissue.

SS: Right. Absolutely.

JM: This may be the problem I just realized is that eating so much meat can be problematic is because that has methyanine. And you have really high levels of methyanine and they're not eating the connective tissue, so they get low levels of glycine. It's this glycine to methyanine ratio that's so critical. So, if you have to connect you eat nose to tail. And you have the whole animal, including the organs. But the connective tissue you can balance out the methyanine to glycine ratio.

JM: Or you can take it as a supplement. By why don't you talk about some of the dangers of... you know, collagen is another way to get this. And there's a lot of collagen supplements on the market.

SS: Collagen is dangerous because of glyphosate.

JM: Yes, that's what I want you to expand on. Why is it dangerous and how do we find a collagen protein that isn't?

SS: Right. So you have to get grass fed. And of course grass fed cannot be guaranteed. That's a problem because collagen has a huge amount of glycine in it. And I suspect collagen is getting disrupted by glyphosate and that's the reason why we have an epidemic in all sorts of issues with bone and joint pain, we have back pain, neck pain, knee pain, foot pain. All of these people with foot pain. They're just having so much trouble with pain.

JM: Gosh, you know it never occurred to me. It makes perfect sense. But this is major public health warning. Major public health warning because collagen protein is in the news. Everyone wants to try to take it. But in my experience most of it is garbage. It's from Cafo chickens in China. And you are actually trying to do a good thing by getting yourself glycine but that glycine is loaded with glyphosate.

SS: Right, it's a very big problem. In fact, Anthony went to the butcher and got a bone. He tested-

JM: Just a regular.

SS: ... he made sure that it was Cafo. He wanted to make sure it was really low quality. And he went back, and he processed it and he tested it, and he found glyphosate in it. You have to break down the proteins first. So he has these enzymes. He applies hydrochloric acid and enzymes. He has a whole process he's working on a procedure. That's another thing about glyphosate is you can get a false test, or a protein because it gets stuck inside protein, and you don't see it. So you need to process it in order to get it free in order to be able to see it with the methods that they use.

SS: So collagen... and he tested gelatin. It had glyphosate in it. And of course he tested vaccines and found glyphosate in them.

JM: Oh yeah, before we leave we definitely want to... because we're at an autism event and obviously there's a connection with vaccines and autism, so this is fascinating information. Maybe some of you have heard a bit before. But if you haven't... but why don't you expand because you really uncovered a good one here.

SS: Absolutely. He tested a whole bunch of different vaccines, and he consistently found glyphosate in the live virus vaccines as opposed to the ones who were just... the just put antigen in it and they put it in some [inaudible 01:16:10] and stuff. There's two kinds of vaccines. But the ones that are live virus are the ones that contain glyphosate in them. And the highest level was found in the MMR vaccine. And the MMR vaccine is the one that's very controversial with the idea that it causes autism. And the pushback on that idea. Andy Wakefield wrote a paper, published it in 1998 in the Lancet that said he was observing that these kids that got this gastrointestinal disorder after getting the shot. And then they regressed into autism and there were maybe 12 kids that he had in this case study and these kids that were exposed to the MRM vaccine and got autism.

JM: Well, that's an interesting theory. And I'm no fan of glyphosate and no fan of vaccines. Especially MMR. But, just to play devil's advocate here, I mean, he published the paper in 98. Clearly the vaccines had to be produced before then. Probably in 97 and 96 and glyphosate wasn't widely disseminated at that point.

SS: Yeah, it was around.

JM: It was definitely around.

SS: There were only a few of them.

JM: So the concentrations had to be relatively low.

SS: Right, and it has gotten a lot worse.

JM: Now today there's no question. But in 96?

SS: Right. So maybe MMR still causes the problem without glyphosate, which is possible.

JM: There's so many other mechanisms. That was an interesting theory and certain it could be valid today.

SS: Yeah. MMR is much worse today than it was before. I took the VAERS database, vaccinated [inaudible 01:17:36] reporting system and I divided it up into two piles. The data before 2002 and the data after that date. And did a statistical analysis of the symptoms that were reported. You saw a whole bunch of symptoms that were pretty serious that were much more likely to have occurred in the data after 2002 compared to before, and things like swelling in the brain and-

JM: And why did you pick 2002?

SS: ... it was a midpoint of the data set. So it was arbitrary to cut it in the middle. And it happened to be a time-

SS: Let me interject here. This is her specialty. She got her PhD in doing this type of analysis. So you know how to do it.

SS: Stephanie: ... yeah. Went through a lot of data in both piles, so I just took the whole average over the time period. And very, very big differences. And autism was one of the things that showed up statistically significantly more often after 2002 compared to before.

JM: Interesting. So supporting your hypothesis about glyphosate.

SS: Yeah. And so the hypothesis is really interesting because that MMR is live virus grown on gelatin, which is dried from all those ligaments from those pigs.

JM: And that's the key because it's live it has to have the fuel source and they used gelatin contaminated with glyphosate.

SS: Yeah. Contaminated with glyphosate I'm assuming, and then they introduced protein.

JM: They're not going to have organic grass fed collagen source. No.

SS: So the microbe takes up the vaccine, puts it into its own proteins. And in particular there's a protein called hemoglutanin that's produced by the measles virus and that's the protein that you need to react to in order for the vaccine to take. So the purpose of the vaccine is to get you to produce antibodies to hemoglutanin. And so it turns out there's a group of people who have been studying MMR in connection for many, many years going back to the 1990s.

SS: Professor Singh is the lead, So-I-And-G-H from Utah State University. Very fascinating work. They showed that the autistic kids... they had a large group. Something on the order of 100 autistic kids and 100 non-autistic. They showed that the autistic kids many of them had a really high response. They did a really good job of responding to the hemoglutanin produced by the measles virus and had a really high antibody response. Those who had the high response almost all of them had an autoimmune attack on the myelin sheath because of something called molecular mimicry. A particular segment of the measles hemoglutanin peptide sequence that resembles very closely,

about 75% the same, as a peptide sequence on myelin basic protein. And so the immune cells got confused. First of all the brain got infected with measles, the brain's immune system responded. And then the brain's immune system started attacking the myelin sheath through this mimicry process. And it turns out that I've found a book that has a table where they showed a particular peptide sequence that is similar between about 12 amino acids that are very similar between myelinic basic protein and measles hemoglutanin. And three of them on both of those molecules were glycines.

SS: So they have three glycines that line up that are the same on both molecules. Any of those glycines could be glyphosate, which could cause the immune system to get specially upset and be hyperreactive to the molecule and then cause this autoimmune attack on the myelin basic protein. So that's my best explanation for how MMR could be causing autism.

JM: Let's take it to the other end of the spectrum. Maybe you haven't looked at this. A good possibility you haven't. Another epidemic we're facing is Alzheimer's.

SS: Oh yes.

JM: So, you know you talked about myelin basic protein, so we've got beta-amyloid in here and then [inaudible 01:21:14] proteins. I'm wondering, in the brain, which... I mean, I think it's an artifact. I don't think it's the cause. But nevertheless, there's a clear association there. I'm wondering if you looked at any relationship between glyphosate and Alzheimer's?

SS: Well, I agree with you that it may precipitate it plaque is not the cause. But there's a toxic form of protein that is dissolved that's soluble in the cytoplasm. That is probably the cause and also-

JM: And what is it? The beta-amyloid?

SS: ... well, the beta-amyloid normally goes to the membrane. And it's not clear what it does but-

JM: I thought it was extra cytoplasm.

SS: ... it goes into the membrane of the cell. It might be excreted by the cell and gets broken. I'm not sure. But it ends up in the extracellular space because the cell collapsed.

JM:
But it goes in the membrane.

SS: It's supposed to go in the membrane. And there's a particular pattern of membrane penetrating proteins that's well established, which is called a GXXXG, three X's between two glycines is a pattern that shows up in the transmembrane part. Ti forms a helix that goes like a screw into the membrane. Amyloid beta has a GXXXGXXXGXXXG protein.

JM: Say that three times fast.

SS: Four glycines equally spaced. It has this long [inaudible 01:22:34] that causes it to go into the membrane. And they had shown that, that motif is a piece of the protein that's problematic. And they've shown-

JM: Oh, so it doesn't go in the membrane. It goes extracellular.

SS: ... if you change the glycine into something that's more water soluble such as glyphosate it becomes unable to form... the glycines are critical for forming the helix that holds the thing together in the membrane. So the whole thing falls apart if you start throwing in glyphosate that's got this extra bulk and this extra negative charge. It's not going to be able to form the helix anymore. It forms beta sheets instead. They've shown this, not with glycine but with other substitutions, they've shown that those alpha helix's turn into beta sheets. Those are soluble, and then enough of those beta sheets gather up and form these fibroids, which you have to take out. And that's true also for some of the prion proteins. You know there's a deer disorder, chronic wasting disease in America. CWD. It's really fascinating. There's a prion protein that's associated with that. That prion protein I looked at it. You can find these proteins by the way. You can search them you can find the whole sequence.

SS: It has something... I can't remember how long it goes. It's way beyond Alzheimer's almost twice as long. GXXXGXXXGXXXG it goes on for seven or eight glycines. Huge long length of that stuff. So it's getting affected by glyphosate and causing CWD.

JM: So is it the same mechanism?

SS: I think it's the same thing.

JM: The beta-amyloid not being able to integrate into the cell membrane.

SS: Same thing. The prion protein [crosstalk 01:23:58] substitutes for glycine.

JM: So it is the... so the observation with Alzheimer's and beta-amyloid is that it is extracellular. But it's supposed to be in the cell membrane. Not fact that it's not in the cell membrane is what's causing the problem.

SS: That's right. And it's not doing whatever it's supposed to do when it's in the cell membrane because it's not there. So it's both messing up its real function and causing this new function. It's catastrophic. So, I think that's... and Alzheimer's goes up exactly in step with glyphosate just like with autism.

JM:
Wow. Have you talked about that?

SS: I have. I've even written a paper actually that Anthony and I... multiple papers.

JM: Is it published yet?

SS: Yeah, we've published papers, more than one that talk about it. I did a whole analysis of ALS too. A whole separate paper.

JM: Really? That's a tough one. A lot of times people think there's an infectious component to that.

SS: Oh yeah? Interesting. I think it starts in the gut. ALS starts in the gut. But it's a disruption of the gut microbiome by glyphosate. Very controversial story but there's this protein called TNF-

JM: Very tough to treat. That is one of the toughest to treat.

SS: ... I know. It's impossible.

JM: It's not impossible, but it's one of the most challenging. So many diseases that conventional medicine is just clueless on how to treat it. Fundamentally so... I mean, they're easy as can be. Almost 100% of them. But ALS is not one of them.

SS: Right, I know. It really seems so irreversible. TDP43 is a protein that is associated with ALS for people who have genetic mutations in that protein. They have an increased risk, and they can get it early. And TDP43 has this glycine rich region. We have a picture of it in our paper. It's amazing because it's this whole section that has tons of glycines in it. And there's a whole bunch of mutations that happen, that are causing this increased risk to ALS that are occurring in that glycine rich region. And many of those mutations are glycine substitutions for something else. It's all concentrated in that glycine rich regions because that's where all those mutations are that are causing the ALS. It's really interesting that protein could be affected by glyphosate to cause ALS.

JM: Excellent. Well, this has been a wealth of knowledge and information.i really appreciate this. Wow.

SS: [crosstalk 01:26:17]

JM: Ye so you did. When does your book come out?

SS: I have a deadline to have the draft ready but yeah the end of January next year. So I have some time.

JM: Okay good. Yeah, you have plenty of time. We'll definitely have you on again. And maybe be able to squeeze an interview in too. Because we're both going to be speaking at Bob Miller's event.

SS: Right, in September.

JM: September in Colorado.

SS: Yes.

JM: Which should be really good.

SS: Yes, that will be fun to do another interview. Hopefully I'll know more by then. We'll have more things to talk about.

JM: Yeah. We'll do something. Might even do a little better audio. I apologize for the... we only have one camera. I had brought two cameras but I couldn't figure out how to get the other one to work at the same time. But we'll have two cameras next time.

SS: Andrew may have trouble with my microphone.

JM: Yeah, we'll figure it out. This information is so fascinating.

SS: It's so interesting isn't it?

JM: Yeah, and people are going to love this. This is one you're going to have to listen to a few times. And thank you so much because you really helped clear up some fundamental misconceptions and misunderstandings. But it ties it all together. Once you follow it, it's just like – oh my gosh.

SS: Once you know enough then everything else becomes a piece that fits into the puzzle. That's when it gets really exciting.

JM: So thanks for all you're doing. It's just magnificent. We need more people like you.

SS: I know.

JM: You've actually been an inspiration to me too because I've been able to offload a lot of my work responsibilities to essentially do what you're doing in a different area where I'm reading... my goal is to read about 2,000 papers a year. The whole paper. Not just the abstract.

SS: Right, right I know. I do that too all the time. Ti's a major part of my time is spent reading papers.

JM: You know, it's one of the... I mean, it's a great thrill to treat patients and help facilitate their improvement. But I'm telling you when you're in this discovery mode, and you read a paper, and you realize something that's never been realized before.

SS: It is so exhilarating.

JM: It's like finding a golden... a chest full of gold coins. And it's like, “Oh my gosh."

SS: I'm so glad to hear you say that because my husband doesn't appreciate how exciting it is when I find out these things.

JM: Ti's like, “Wow.” And it's not every day. But you know, on a regular consistent basis you're going to get these epiphanies.

SS: It's a real adrenaline rush. It really keeps you going. Especially with the world that's so depressing. It's what gives me joy.

JM: There's a real important need because there's so many puzzle pieces. Someone's got to put it together. It's kind of hard to follow. It's going to force you to consolidate it and make it concise and clear and walking through it like you did in your book with Cynthia?

SS: Right, Cindy.

JM: Cindy, yeah. All right. Well good. Thanks so much.

SS: All right. Thank you.

JM: I appreciate everything you're doing.

SS: It was great. Thanks.

JM: All right.

[END]