Speaker 1 (00:00):

<affirmative>, you know, um,

Speaker 2 (00:01):

Can we talk about the origins? Like, so in, in, in, in your assessment, you know, how do you see the origin of this virus? What do you think it is?

Speaker 1 (00:09):

Right. And if you're, and if your listeners are interested, there's a book that we, we published in September called is COVID 19, a by a weapon, a scientific and forensic investigation where I lay out many of the D tells for people. So yesterday or the day before. And I'm not sure when this video will come out, but yesterday or the day before NIH finally admitted that it had been funding gain a function research. Um, and I'm, you know, they're throwing people under the bus right now because it's very clear to them that they've been caught on this. And we've been talking about it for a while now. So if you go back, you can see what's called Gaina function research that the United States has paid for. In fact, the department of defense has done has paid for more than half of it with, uh, a gentleman by the name of Peter Deza at EcoHealth, who made it possible to funnel federal monies, uh, from N I I D and NIH and department of defense and civil of federal agencies, uh, to Ralph Burke at the university of North Carolina and people at university of Texas, Galveston, university of Iowa, Wisconsin, you name it, there's a lot of players that have, that have been involved with this.

Speaker 2 (01:14):

Can you briefly explain what, uh, gain of function is just

Speaker 1 (01:17):

Briefly, right. Right. So gain of function is where you take something like a virus and you change it to either make it more effective or more dangerous. Now, the premise is what you wanna do is stay one step ahead of a problem. And if you do that, you know, what's coming down the line. The reality is, however, if you look at the two decades worth of research and funding, and even the patents that have come out of this, you see that this has not been a, let's just stay ahead of it. Phenomenon. This is just, how far can we push the envelope type thing? Can I

Speaker 2 (01:52):

Ask a question, cuz this is interesting to me. Um, and I know you also have a degree in law. Yeah. So maybe yeah. In your spare time. So, but, but you know, obviously, you know something about intellectual property, you know, based on, on, on that experience, I'm sure when you say patents, um, are you saying that they patented viruses that they created or what what's the patent,

Speaker 1 (02:14):

Right. So that's one of the big misconceptions that people have. Um, uh, you, so if you wanna know what a patent is, you basically look at what's called the claims. I mean, there's a lot of stuff that we put down, but the claims are what you're claiming. So when you look at all of these different things with the viruses, they're not claiming they can make a virus. What they are claiming is that they have developed a method to change viruses. Okay. And that's, and that's what these patents are. So, uh, a lot of people have read through those patents and said, oh my goodness, they're patenting life. And that's not what

those patents say. Those patents clearly state that here's the process. One of the patents, very specifically states when you get to it that, you know, N I, I D <laugh> Anthony P group and NIH funded research very specifically for the gain of function or alteration of the spike proteins of coronaviruses. And it doesn't get much more specific than that.

Speaker 2 (03:16):

Was it recent or was it year, years ago that, uh, that Fauci his team have that patent, uh, that you just described?

Speaker 1 (03:24):

Um, that patent is sometime within the last 10 years, last five to 10 years. Right. Um, if you look at, and, and if I think it's always interesting, you know, people look at my history and they think, well, this guy's all over the place where if you really look at the threat of my, I research, there's a continuity to it. And there's a continuity to Barack's research. If you look at Barack's research, he began with what was called transmissible gastro VIR virus, or T G F. Uh, so it was a, a virus that affected the gastrointestinal tract that was easily transmissible. And, and that's really where his work began. But then over time as you track him, he, he merged into this area of coronavirus is about the same time that she Lee did back in 2004. So the bashing of me by the federal government and the court case, um, relates to the fact that the neuro five AC receptor that I was working on for heart disease happens to be the same receptor that was critical for shes, a Lee, to take her glyco, her HIV, glyco protein, one 20 inserts into the coronavirus.

Speaker 1 (04:33):

That's where that attaches reality is you wouldn't want somebody like me working on, even though I didn't know what they were working on working on that same field, because it's a problem for what they're doing, but it turns out it's the exact same receptor. And then the quantitative work I did in nuclear imaging exposed, big formalize and the, and, and, and I came out, you know, they came after me with a vengeance, which is fine. I'm still here, still standing, still sharing the information about what's really going on, which is what's critical. That was the timezone that shes a Lee and everybody else, uh, was working on the beginning of this coronavirus. We were completing the human genome project at that time. And so it, wasn't known to researchers that all viruses can, can, uh, reverse, transcribe or get into our DNA. And the way they do that is, uh, about 18% of the human genome are genetic code, uh, has what's called long interspersed nucleotide elements.

Speaker 1 (05:38):

And those elements allow viruses to reverse transcribe. So DNA to RNA is transcription mm-hmm <affirmative> and RNA to protein is translation. So DNA to RNA is transcription. So RNA to DNA is reverse transcription. Okay. So it's the ability to take an RNA virus and insert it into DNA. And so it, we now know that this virus SARS SCO two does this in all, but three human chromosomes. Um, it's also been known in cardiology for some time that platelets, which are part of the thing that help your blood decline are very rich in reverse transcriptase. And we know that CD four helpers cells, which are a type of white blood cell that deal with infections, including SAR, SCO two and HIV, um, are rich and reverse transcript a, which is how I gets into our, our what's called our T helper or CD four cells and hides until it repos itself, which is why it's a problem. Well, it turns out it would appear that Shely thought that she needed the HIV glycoprotein one 20 to reverse transcribe and to do attach to the cell, which is how it initially attaches to the cell. And Luke done a wonderful job of showing that there's a tremendous

amount of, and which is SIM deficiency, virus, or equivalent to HIV human immuno deficiency virus in this SAR. Two,

Speaker 2 (07:03):

You brought up Luke Montier, which who's a Nobel Laureate. Um, right. And, uh, so I'm glad that you kind of cite the, you know, where his work is, is directly in this arena. I mean, it's very relevant to what's going on today. Do you agree with the sentiments? I mean, he's needs to be really alarmed with, uh, what we've been doing. Uh, so are you, do you agree with the sentiments around, uh, the COVID

Speaker 1 (07:25):

Response? Yeah, so I know professor Monier, uh, Monier MC out of who's one of the head, uh, experts in the, in the world, in Japan for, for, uh, MCAC research and myself provided the three affidavits for the international criminal court case. So we're the ones who are providing the expert testimony behind the scenes. You know, I, I, I chuckle when people CRI criticize Monte because that's kind of, you know, this is, it reminds me of every student who ever comes into the classroom who tells the professor they're running <laugh> it's like, okay, I don't, I don't have time for that. When people come in and do that with me. Right. And, and Montier, doesn't have time for the inter we just kind of chuckle. It's like, you need to learn a lot more before you get to that. So, um, he is appropriately, uh, alerting people to concerns about these vaccines.

Speaker 1 (08:19):

We, which, you know, we, we have data now that I talked about extensively that shows that the vaccines actually impair the immune system. And one thing I started to mention earlier was that in 2017 BARR and his group stumbled across open reading frame 10 for a coronavirus. And if you insert open reading frame 10 into SARS two, it will shut down the human immune system with open reading frame 10 in that genetic code, you cannot make interferon, or you do not make interferon. So there's no innate immune response to protect the person. And that's the baseline. So you have innate immune response, which are the T-cells that we talk about. And then you have adaptive, uh, humeral antibody response, which is layered on top of that. But you have to have that innate system or the antibody system doesn't work. So we know that there's much more dangerous things that they can bring out. And in fact, they boasted back in around 20 17, 20 18.

Speaker 2 (09:17):

When you say they, who do you mean they, uh,

Speaker 1 (09:19):

They being BARR and she Lee, that they could make a more infectious virus than they had. So this is

Speaker 2 (09:26):

University of North Carolina and the Wuhan lab, basically

Speaker 1 (09:29):

Collabo Huan Institute of virology, right? And, and

Speaker 2 (09:32):

They're, they're collaborating on this gain of function research. And the boast was what exactly

Speaker 1 (09:37):

That they had, that they could make a more infectious, more dangerous pathogenic virus, period. Wow. Yeah. And BARR was very honest in a nice Italian interview that we've shown several times where he says, look, if we, if we don't sign our signature to the genetic sequel, it comes to the bar lab. You won't know mm-hmm <affirmative> <laugh>, these are nucleotide bases. Um, FYI, let me hit this because somebody asked me a question about this earlier. Anybody who thinks these viruses do not exist is mistaken. Okay. This is not 18 hundreds, Europe, where we're using cooks past to get bacteria. Okay. This is 2021 where we understand that every living organism is made up of nucleotide bases to make its genetic code. You define it by its nucleotide basis. Viruses live inside of cells. They, they infect cells and then they live inside of cells. So we, and you sample out somebody, for example, from the lungs, what's called a Broco Alvi or lavage.

Speaker 1 (10:42):

So you go in and you do washings with saline and suck it out. You then have to treat that material to get the garbage out mm-hmm <affirmative>. And then you look for the genetic sequences and you pace those sequences together. And, you know, what's there. And so who thinks that this virus or these viruses and these variants have not been isolated is simply still living in 1860 and still working with bacteria. Although I would argue that certainly those, those postulates have been satisfied from all the studies and, and, uh, with the last animal, being the humans, receiving the vaccine, we've definitely demonstrated that we can now transmit, you know, if there was any question about that, we kinda, I think we've more than buried that. So that's the function of gain a function research. The problem is if you get too far ahead, uh, of, of the curve now you got something that's, that's not an E change that's gonna happen.

Speaker 1 (11:39):

You know, evolutionary change occurs one nucleotide base at a time. So for example, sickle cell anemia is a single nucleotide base and it changes a red blood cell into a sickle cell. Now, the reason why it still exists is because there's an advantage for that. If you're in an area where there's malaria, malaria, can't live in sickle cells, so it's a survival benefit for infection. However, you're certainly not gonna be an Olympic athlete. So there's a trade off there, right? Mm-hmm <affirmative> so that's one nucleotide base. There is 1,770 HIV and SIB based inserts in this thing. And one of the other critical things is called the fur and cleavage site, which is, uh, for amino acids, Prolene, arginine, arginine, and alanine, and amino acids are given a letter of the alphabet so that we can short, you know, right shortcut, uh, and writing. And that's called P R R a Prolene arginine, arginine L and that's each amino acid has three bases.

Speaker 1 (12:43):

So that's 12 bases, not one base. And it turns out that that insert is critical for, uh, this, the virus to who attached, because this virus attaches by that glycoprotein one 20 to the neuro five AC, and then it links to the ACE two receptor it then is, is, starts to be brought into the cell by what's called the transmembrane protein series two or TMP R SS two. And from there, it goes to the pure cleavage site, this P R R a site, and then it goes to neuro one and it's brought into the cell. So it's a little more complicated than just ACE two, but when you start looking at it, that glycoprotein one 20, shouldn't be on there, the pure cleavage site shouldn't be on there. Um, so if I

Speaker 2 (13:33):

Just try to understand the implications of this. So we could say that basically, uh, and obviously this is sort of a, um, you know, advanced, uh, biology that you're, you're discussing here, molecular biology, but, uh, but the conclusion of what you're observing and what you're sharing here is what is that? This is a manmade virus. And is that demonstrably true? Or is that speculatively true? Well,

Speaker 1 (13:58):

If you do, if you do the math, if you do the statistical analysis of the probabilities of these inserts is like 99.99, 9%. Okay. If you do the other approach, which is to say, well, if it's naturally occurring, there must be an animal carrier. Mm-hmm <affirmative> well, there is no animal cure that's been found. There's no animal cure that's been found for SARS co one from thousand two. OK. If you listen to, uh, Leema yang, who is the virologist from Hong Kong, who got out of there and is kind of been hiding in the United States right now, she worked with people that worked with SARS co one. And she said that was a bio as well. So SAR COVID two is applicably named because it's kind of version two, it's an upgrade if you will. I think the confusion, when we talk about bio weapon is people think that a weapon should kill the enemy.

Speaker 1 (14:49):

Right? Well, I'm old enough that I'm of the Vietnam era. And I will tell you that when we went to Vietnam, munitions were changed, uh, to a smaller size bullet so that we didn't kill the enemy. We just named the enemy. And the benefit of that is that if you kill an enemy, that's one person off the battlefield, but if you name enemy and they have friends, their friends will come and drag them off the battlefield. So now you've taken two or three people off the battlefield, right? So it's a more vicious way if you will, of, of playing war. And if you're gonna develop a bio weapon and remember the D D paid for more than, uh, half of, of, uh, of this freeze search for, for days act and provided David Fran, who'd been a former commander at the do D as an advisor for days act.

Speaker 1 (15:38):

So again, the DOD doesn't work with the girl Scouts and the boy Scouts. So you have to look at what they're doing and anybody who thinks that Fort Dietrich is not involved in this is, is a little bit delusional. So let me me just kind of throw it out there for you that I received two emails requesting me as a physicist to know if I would be interested in working at Fort Dietrich on funding for viruses supported by N I a I D so, you know, would I like as a physicist to be involved with the imaging of, of, of coronaviruses at, for Dietrich paid for by research from N I, um, okay. I don't know how much, you know, I mean, I really wanted to say, why did you send me these requests? I mean, are you not aware that I'm one of the people looking at you and saying me, you shouldn't have been, when were

Speaker 2 (16:33):

You sent those requests

Speaker 1 (16:35):

Actually in, in the spring of this year? Wow,

Speaker 2 (16:38):

No kidding. Uh, so that, that is kind of bizarre.

Speaker 1 (16:42):

Uh, it is <laugh>. Yeah.

Speaker 2 (16:46):

Uh, well maybe you should accept it and, uh, found out what was going on over there.

Speaker 1 (16:50):

<laugh>, you know, so that's where I think that, you know, it's one thing to get it. And it's another thing to say, yeah, let's play this game. And, you know, I mean, I don't, I don't wanna do that. I I'm very upfront. I'm very honest. I'm very trying to address conflict of interest. I do everything imaginable to not play these games that other people play. I mean, there's enough people playing the games. You don't need me and their playing in the sandbox too. Right. Um, I'll just go swim in the pool. Yeah.