Planet Waves FM Interview with Stephen Bustin, conducted by Eric F. Coppolino

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Transcription by Joshua Halinen for Chiron Return

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Hello world. My name is Eric Francis Coppolino, the host of Covid19 News and of Planet Waves FM. Both are located on the website planetwaves.net. You are about to hear my interview with Dr. Stephen Bustin in the UK recorded -- Monday, February 1, 2021. I had originally called him to speak about the reverse transcript days phase of the polymerase chain reaction assay that's being used to maintain the case count. As you will hear we got into some other subjects including the Euro-surveillance controversy over the Corman-Drosten paper. This lasts about 75 minutes. Here you go.

Dr. Stephen Bustin: Hello.

Eric Francis Coppolino: Hello.

SB: Hi there.

EFC: Is this Stephen?

SB: Yeah, good morning…

EFC: Yeah, it just struck 11 this moment.

SB: Just 11.

EFC: Yes. Okay, so hold on. You're coming over the public address monitors. Okay, turn that off. So, thank you so much for helping me out with this.

SB: That's okay.

EFC: I'm determined to understand this the whole way.

SB: Right.

EFC: Not have any gaps… I mean this is so complicated. It's so astoundingly complex that I mean I'm sure I'll be learning for a while.

SB: Well, it isn't that complicated really.
EFC: Oh please. [Laugh] Please do. I mean, you know, wash your hands before you make dinner. I mean, that's what…don't put butter in the washing machine. I mean…

SB: Well, it's not quite as simple as that but the principle is actually quite straight forward. And…yeah. But let's discuss things and then see how we get on. [Laugh] **|00:01:59**

EFC: Right. So, a lot of people know the New York Times reported that 90% of these so-called PCR confirmed cases would disappear if you dropped the cycle threshold down to 30.

SB: Yeah.

EFC: And then what the New York Times has not admitted and I have personally tried to shake their tree a little bit – is that they admitted that in 2007 that there was a 100% false positive incident at Dartmouth-Hitchcock. Do you know that incident?

SB: I don't know. I have looked at the article you referenced, the Planet Waves article. I have looked at that.

EFC: Oh okay, the one about Love Canal? Or the Reality Check?

SB: Mhmm. The Time for a Covid Reality Check. Yeah.

EFC: Yeah yeah, that one. So, you know, the 100% false positives in 2007. No cases of pertussis. 90% false positives. How is this possible is what we're looking at I think, right?

SB: Yeah. Okay, I think we need to go back to basics. When you go back to basics and that's explained to you how the whole thing works you can see what the problem is because the technique itself is not the problem. It is the way it's being executed.

EFC: Yeah.

SB: So, first of all the thing to remember is there are two kinds of results. One is simply the qualitative result. Is it there? Is it not there?

EFC: Right.

SB: And a lot of pathogenic detection is based on yes or no answer. And there's absolutely no problem with that because things like RNA quality and how much or how little is there doesn't matter quite so much if you're just trying to see whether it is there or not. So that's the first thing about quality of the PCR. | **00:04:09**

EFC: Was that Kary Mullis's invention? The original invention?

SB: Well, Kary Mullis's invention used to be run on gel. So you used to take your amplification tube and then run this out on a gel which was made up of kind of **____4_____**. And then look at it. So you saw bands on gel. That's the original PCR. Okay, that I call legacy PCR. Now the problem with that PCR is that there is very, very much to contaminate all over the place. Because you pick up your sample after it has been amplified and then you get aerosols generated and so from a diagnostic point of view that is a complete and utter no no.

So back in the 1990s something called real-time PCR was invented. The real-time PCR and the original PCR are exactly the same thing except that rather than opening a tube and running a gel you detect the amplification during the process itself. You never have to open the tube, so you minimize the problem of contamination.

EFC: Ahhh….

SB: But that's the key difference between those two methods.

EFC: Mhmm.

SB: Okay, so regardless of which method of PCR you use and when you're using reverse transcription PCR; which is for SARS-coV-2, it is essential you have two controlled, always two controls. One is a sample, [recording cuts out for moment] no template. The so-called no template negative control. And that always has to be negative. If it is positive, then you know you have contamination. And the second type of control you have to have is a sample that contains your DNA or your RNA, but you don't reverse transcribe it. So you just do a PCR. And that should also be negative. Now that can be positive but at a very, very high level because Taq polymerase does replicate RNA if only very inefficiently. | **00:06:16**

EFC: Mhmm.

SB: So the problem of course that can arise is if you have contamination and it's very low level contamination then you can get something at the very end of your run. But we can get on to that in a second.

EFC: Mhmm.

SB: So from a qualitative point of view for most pathogen detection you run your PCR in an instrument and you get a negative result. Your controls are negative. Your positive control is positive. You have to use that result. Now, what can happen is that you get the result that is at the borderline of detection and that is anything above…well…okay, let's go back another step. Now, the output of a real-time PCR experiment is the quantification cycle, the CQ cycle. The

problem with the CQ cycle is that different re-agents and different instruments and different operators can get significantly different CQs.

So I'm just publishing an article to describe this in more detail. And I think that's in my paper. I can't remember. But it's well known, we've mentioned it a lot of times that for example if you have a probe from two different manufacturers, then the background of the probe is different. And that then can change the threshold and therefore the CQ that you get at the end.

You can manually move the threshold up and down and change the CQ. And different instruments give you different CQs as well. So if you run the same test of the same sample on instrument 1 you get a CQ of 30 but on instrument 2 that might be 35. So that is the problem with fixating on the CQ. | **00:08:01**

EFC: (Gasps) Ohhhh…

SB: Now in general if you have a single molecule and everything's working the way it should be in your lab somewhere between 33 and 37 cycles would be the CQ that you get for single molecules. For example: in my lab, on my instrument I know that anything above cycle 36 is not real. Okay? So if you run your PCR and you get a CQ of 30, that's probably positive. 37, possibly positive. And that's another issue you have to worry about and that is in addition, because the detection of a single molecule at cycle 35 say is -- only valid if your PCR is 100% efficient and if there's no inhibition.

EFC: What's inhibition?

SB: Oh, for example: blood stops the enzyme from working. So if you have any **___8___00:09:04** in your sample your enzyme doesn't work as efficiently and what you might find with a CQ of 15 might suddenly be 25. That's called inhibition.

EFC: Okay, things that stop the reaction from working?

SB: Yes. So that's how you can get cycles above a certain threshold that are still positive. But if you have the proper controls and if you test your samples for inhibition then you should know about that and not get it. But let's assume that… Let me refer to my hospital because that's what I can tell you about because I know about that hospital. In my hospital they extract the RNA and I've tested about 100 of their RNA samples and there was no inhibition there. Okay? So I know from their samples that if the CQ is at 35 – that is very, very low levels of target in the samples. | **00:10:01**

Now again you have to distinguish the difference between target and the presence of viable virus. Just because you detect a fragment of RNA doesn't mean that you've got infectious virus present. And the problem here is that there are no certified standards. Some standards that tell us what the CQ actually means in a particular experiment. And if you look my paper that I've

published in Standard Report, we've tried to put in a standard that we've quantified. But that still only tells you that there are you know, 10 copies or 5 copies of a hundred copies.

We then need to relate that back within the clinical context to know whether that threshold of 30 say, does actually mean there's infectious virus. And that has not been done yet. So all you can say at the moment with these tests is that if you take a test and you have a positive result it is likely that you have virus present. In order to be able to say whether this is likely to be infectious you'd need to take the test again a day or two after you've had the first test. Because if it really is in the early stages of infection then you would get a decrease in your CQ. If you're at the late stage of infection when you're no longer infectious then it would either disappear altogether or the CQ comes down 25 to 30 or 35.

EFC: Mhmm.

SB: So, that's the qualitative aspect of it. People try and use it quantitatively, particularly to discuss viral load. And you just cannot do that at the moment. We have no idea what CQ means in terms of viral load. And then we can't extrapolate from that into a clinical context, ie. whether this person is infected or not. But in terms of detecting the presence of viral targets the test is absolutely reliable if you include the proper controls.

EFC: And if you have an authentic target? | **00:12:00**

SB: Well, yeah. I'm not sure why you think you wouldn't have an authentic target. Because we know that SARS-coV-2 is the agent that causes Covid19. That has been shown.

EFC: Can I ask you how we know that? Because there are so many different alleged manifestations of Covid19 that you would have to prove the connection causatively to each one wouldn't you? Blood clotting and you know ground glass pneumonia and bad toes…

SB: No, well… in terms of the molecular… you refer to Koch's postulates in your article.

EFC: Mhmm.

SB: Well, we've moved on from that really because you can't always fulfill those original postulates because you can get asymptomatic carriers of cholera salmonella and some organs just don't grow in cell-free culture. So, I think you have to move on from there. And the way we've moved on from that is if we can identify using molecular techniques the same pathogen or a particular pathogen in all individuals very particular to these than it is very likely that that pathogen is the causative agent. If we then isolate that agent and infect tissue cultures and we find that the cells die, or whatever, the virus starts replicating then we know that that virus is able to infect the cells. And if we then can identify the mechanism **_____12_____** that. And we know that the virus is binding to the ACE2 then we have a molecular pathway for a virus to infect an individual and cause the disease. And I don't think there is any question that

it's happening. Particularly as it is the third time it has happened. I mean we have SARS-coV-2002 or 2003, we have MERS and now there is coV2. | **00:14:13**

EFC: Right. Well, yes.

SB: And then if you find antigen in the viral protein and antibodies to that protein in those individuals and there is virtually conclusive evidence that that virus is causing that disease.

EFC: Can we come back to this at the end?

SB: Yes, of course.

EFC: Because I do have questions there. So, let's assume that there are authentic primers. Meaning they are working from a real sample. Right?

SB: Yes.

EFC: That's what that means? Doesn't it?

SB: Yes.

EFC: That they've got a clinical sample.

SB: Yes.

EFC: That they know from diseased people and they design that into the assay.

SB: Yes.

EFC: And then they tell the PCR go out and find it.

SB: Yes.

EFC: Like blood hounds having a sample of the person's hat or whatever.

SB: Okay. [Laughs] Yes.

EFC: Pretty much, I mean right? Keep it simple. So let's assume that's true.

SB: Mhmm.

EFC: And then we enter the RT phase. Right? The next thing that happens, we're looking for RNA. We enter the RT phase.

SB: Yes, that's the third thing you have to do. You have to convert the RNA into DNA.

EFC: Right.

SB: And you usually do that by using a specific, in fact, using one of the PCR primers. That's what you normally use. So we have the same primer as we have for the PCR. One of the primers will bind to RNA, the other one won't.

EFC: Could you say that a different way?

SB: Okay. RNA as you know it is single stranded.

EFC: Yes.

SB: DNA is double stranded.

EFC: Mhmm.

SB: So, for PCR you use two primers. One binds to one strand and the other one binds to the other strand.

EFC: Mhmm.

SB: Yes?

EFC: Yes.

SB: Because RNA is single stranded only one of the primers will bind to the RNA.

EFC: Mmmm… It's mirror won't bind. It's mirror won't bind.

SB: Say again?

EFC: It's mirror image, it's opposite won't bind.

SB: Yeah, the other strand won't bind. No. That just floats about. So, the first thing you do is add your primers to your RNA and use reverse transcriptase to extend from one of the primers. It's called the downstream primer.

EFC: Or the south primer? Is that sometimes called the south primer?

SB: Mmm… It must be…

EFC: North and South? Okay. We'll use your term.

SB: I don't use those terms.

EFC: Yeah, let's use your term – the downstream primer.
SB: _____14_____ 00:16:55. I guess it would be south. Yeah. And so, what happened is that primer is specific, or should be specific for the target that you're looking for.

EFC: Yes.

SB: So once you've converted the RNA into DNA you know have a single DNA strand to which the upstream primer can bind.

EFC: Mmmm. The other one.

SB: Yes.

EFC: Right.

SB: And now you start your PCR reaction and that upstream primer produces a strand of DNA from the strand of DNA that was made in response to the RNA. Are you with me?

EFC: One more time.

SB: Okay, let's start again. You have a single RNA strand and your downstream primer makes a strand of DNA from that. That strand now can be bound by the upstream primer. | **00:18:02**

EFC: Yes. Yes.

SB: Which produces a new strand of DNA that is complimentary to the other strand, which in fact the same sequence as the RNA was.

EFC: Mhmm. Right.

SB: And now both primers combines to that DNA. The option 1 has already bound and the one that produced DNA from the RNA can now bind to the newly synthesized strand. And that's how you start your PCR reaction now.

EFC: Is that stuff called C-DNA?

SB: Yes, the first strand you make is called copy DNA. Copy DNA, that's what it's called.

EFC: Okay, got it. That's what I figured from context.

SB: Yeah.

EFC: So, there's a lot of mystery in this stage. Everyone is telling me this is where things go wrong.

SB: Ummm… It depends. It goes wrong when you're doing quantitative PCR because the RT step is quite inefficient and high quite variable. But if you're simply looking to say if something is there or not it doesn't really matter.

EFC: Right. But for SARS-coV-2 they're using quantitative?

SB: Well, no they're using real-time PCR. And they shouldn't use it quantitative, they shouldn't make pronouncements based on the CQ.

EFC: Pronouncements of infection based on the CQ?

SB: Yeah. You shouldn't be doing… Well, if the CQ of 15 or 20, that's a very high viral load and you probably can say there was absolute infectious.

EFC: Mhmm. Mhmm.

SB: But if it's 25 to 30 then it becomes unknown at this stage.

EFC: And they're all running 37 to 47.

SB: Yeah, that's not something I would recommend. I mean to run anything above 40 cycles is pointless.

EFC: Even Fauci says anything above 35 is pointless. | **00:19:58**

SB: Yes, but bear in mind I talked to you about different people getting different CQs. So your 35 could be my 30 or someone else's 40, so we have to be somewhat circumspect there.

EFC: That's right.

SB: But yes. Once you start getting into that realm you have to be very, very careful. Yes.

EFC: So one thing I don't understand is the role of the probe. How would explain what a probe is.

SB: The two primers that you use for the PCR are very specific and amplify what we call amplicon, a fragment of DNA.

EFC: Mhmm.

SB: Okay. Now it is possible by, you know, pure coincidence that I design my primer inappropriately and it also happens to amplify something else. For example: a bit of human DNA or RNA or a totally unrelated or a different cells virus. That it's just possible. Okay?
EFC: Mhmm.

SB: And so, you might end up with the wrong amplification product because your primers aren't quite specific.

EFC: Mhmm.

SB: Right? So the probe is a third oligonucleotide which binds to the amplified DNA. So, it's essentially an additional level of specificity. So, not only do you have to amplify but you also have to confirm that the amplification probe is the right thing because it is virtually impossible, if you get the wrong amplification **____20____ 00:21:28** it also binds to the wrong probe. Because the probe binds to that as well. So, it simply tells you that what you've amplified is the right thing.

EFC: Right. Right.

SB: So, that's why all pathogen detection kits that are FDA approved have to be probe based. Because you can get false positives if you don't use a probe.

EFC: Okay, so what happens when you have partial annealing of probes and primers. And what is an auto-dimer. Auto-dimer. D-I-M-E-R | **00:22:00**

SB: Auto-dimer. Oh, dimer.

EFC: Yes.

SB: So, one of the critical… The most critical… I like to compare the primers to the tires of a car. The tires of the car are crucial to keep the whole thing on the road.

EFC: Yeah.

SB: Primers are crucial to keep PCR on the road. So, the design of your particularly your primers is critical sometimes people don't design them properly. And so, what can happen is that for example: the primer can bind to itself. So, it can loop back to itself and bind to itself. Or it can bind to its partner. If the sequence at the **_____22_____00:22:51** complimentary to the sequence of the other primer and then you can get an **____23____00:22:56** dimer forming. So, a primer binds to itself and can amplify itself.

EFC: Ah, it's like a feedback loop?

SB: Yeah, it kind of amplifies itself. Yeah. But you don't normally see that. Or you don't see that. The effect it has though is that it can reduce the sensitivity of the assay. So that you may get a false negative that way.

EFC: Right.

SB: But a properly designed test shouldn't do that.

EFC: Mhmm.

SB: And for FDA approval the manufacturers have to show the limits of detection sensitivity and the efficiency of the assays. And you would detect that at that stage. Now, I have looked at… Well, a lot of commercial primers of course aren't published. The original… on the 13th of January Drosten and also **_____25_____00:23:44** published assays targeting SARS-coV-2. And there was a mistake in one of those primers. I don't know if you were made aware of this?

EFC: Mhmm.

SB: And so that can happen. But I don't know, I haven't looked at the sequences to see whether they bind to each other or not. But that's the kind of thing you have to be very careful about when you're designing primers, that they don't bind to each other and that there's no mistake in the sequence. And that you also of course have to show that your SARS-coV-2 primers don't bind to any other SARS virus. | **00:24:17**

EFC: Or any other virus. Or any other normal RNA.

SB: Yes. That is less likely because it is a coronavirus after all. So, but it could bind to a human coronavirus for example. Yes, that's what could happen. And it's less likely to bind to a measles virus for example. But because there are human coronaviruses already that could be a problem. But I think more likely it would bind to some bats or some other coronaviruses that exist. That's why you do your in silico designs before you actually ever get to order your oligos and you go to the database to make sure that your primers don't bind to anything else.

EFC: That's the right way to do it. Yes.

SB: And I use a program called, it's called Beacon Designer by an American company called Premier Biosoft.

EFC: Econ Desire?

SB: It's called Beacon. B-E-A-C-O-N. Beacon Designer. And it's by an American company called Premier Biosoft. And I've used that for 10 or 15 years now. That is in my mind the gold standard. And which a lot of companies I would say use. So the problem is that typically… I

mean I design lots and lots of assays and typically I would take the assays that I get from the designer and I would tweak it myself.

EFC: Yes.

SB: So, assay design is one of those black arts you know.

EFC: [Laughs]

SB: I've looked at kits from several companies now and I guess I must say I've never looked for false positives but I don't have any other viruses but they all perform in terms of detecting SARS-coV-2 all reasonably the same. But you do get differences. So, for example, you might get a two or three CQ difference. So again, coming back to what you said about CQs if you use a test from company A, your CQ of 35 might be my CQ of 32. And then if I have a different instrument and use my threshold **____28____, 00:26:20** I could easily end up with 7 or 8 CQ differences. | **00:26:23**

EFC: Yeah.

SB: So that's why I say, talking about CQs is very misleading when one shouldn't talk about CQs at the moment. One should say it's there or it's not there. You shouldn't say it's infectious or not there. Infectiousness depends on other things as well then, that are outside of the test itself.

EFC: Mhmm. And all the paperwork admits this: CDC, FDA, WHO, the lab's own technical data admits this.

SB: Yeah, I think no one disagrees with that.

EFC: Well, but the New York Times calls every PCR a confirmed case.

SB: Yeah.

EFC: That's a lie.

SB: If you have a positive result there is SARS-coV-2 RNA in that sample. Now, how did it get there? It could be that the person it has been taken of has had or has the virus. That's one possibility. It's possible that during the collection process became contaminated. Or that it became contaminated during the extraction and testing process. So those are the three kind of possibilities. It is highly unlikely that this stage I think of the proceedings that the test itself is at fault. It is the pre-analysis that needs to be looked at and something could have gone wrong there.

EFC: Yeah, and yeah.

SB: And I think yeah, most people agree that just because you have positive results you have to then relate that to the clinical symptoms or whatever to say that this person is replicating SARS-coV-2 or not. | **00:28:08**

EFC: Right. And…

SB: And the only way you can really do that is by doing a second test a day later say or if you're lucky and the viral load is high enough you might be able to detect using antigen tests.

EFC: But that's not being done. Every time someone gets a PCR positive the New York Times and everyone else is calling that a confirmed case of SARS-coV-2, of Covid.

SB: Yeah.

EFC: I don't understand what the confirmation is. There's no split sample. There's no retesting. There's no symptoms.

SB: Well, I guess it's your definition of confirm. If you confirm that there is SARS-coV-2 RNA there. That's what I would say.

EFC: Right. But they're calling it a confirmed case of infection.

SB: Yeah. [Laughs uncomfortably]

EFC: And then they're saying there's like a quarter million of these a day. I don't know single person who is sick.

SB: You don't?

EFC: I don't.

SB: Well, we have…are ICU's are overrun at the moment.

EFC: Mmmm.

SB: And my daughter is a 5th year primary medical student and it's horrendous.

EFC: Well, I'd definitely like to see that data.

SB: And we've got 3.5 thousand people on ventilators. It's really bad.

EFC: Right. I want to talk about that in a second. Can I ask one question about the probes?

SB: Yes.

EFC: A person coaching me along in this named David Battini, an engineer biochemist in Italy said that one of the problems is probes partially landing in the wrong place but they still release the Taq Man flourofors and the positive signal is amplified anyway, even though in reality the relative cDNA amplicons are not duplicated. | **00:30:05**

SB: No, that's not a problem. In all my time… I've done real-time PCR… We have the first academic instrument in the UK back in 1998 and I have never ever had a probe partly binding to something and giving you a positive signal. It just doesn't happen. If it is properly designed, it just doesn't happen. Now, it is possible if you don't design the probe properly and don't check properly that it doesn't bind to anything else that part of the probe can bind. But it isn't really a problem. Much more of a problem is if there's a mutation in the probe… Okay, let me give you an explanation of something.

EFC: Yeah.

SB: We have… Have you head or SNIPS? Single Nucleotide Polymorphisms. Have you heard of that term?

EFC: I have not… Maybe I have. I don't remember, but let's say no.

SB: Okay, all of our DNAs are very, very similar. So you know we are virtually identical from a DNA point of view. But we have a few changes that make us different.

EFC: Mhmm.

SB: Okay. And some of these changes are quite characteristic of certain… it's not quite true but let's say eye color. Okay, there's a gene that determines eye color. There's more than one though but it doesn't matter for the sake that I'm going to try and explain to you. And you and me might be exactly the same except for a single base that's different. Let's assume that. Okay?

EFC: Okay.

SB: So we have a single base that is different. Are you with me still?

EFC: Yeah. A single base out of how many? How many are there? 32 Bases?

SB: Well, let's…no. Single base out of say a thousand bases that make up the gene.

EFC: Okay.

SB: Okay, the single base. Now we can develop tests, PCR tests where we have two probes and they will distinguish within a single different base so that my… one probe will detect my gene

and the other probe will detect your gene. And we call that SNIP detection. It's called genotyping. This is typically done for all kinds of different genes. I've just designed for example; you've heard of the new variance that has arisen. Haven't you? |**00:32:07**

EFC: What is it?

SB: The new variants that has arisen. The one that was first detected in the UK.

EFC: Yeah. I've been wondering a lot about that. Yes.

SB: Yeah. So these are single nucleotide changes that cause a different protein. Now, I and other people have designed assays that detect that single change. So what I am trying to say to you is the probes are incredibly specific. And if they detect something that is inappropriate than they haven't been designed properly.

EFC: And that's a variable, I would say. That puck is on the ice. That's in play right?

SB: It's possible but bear in mind you've got kits that are being used by companies that have gone through all kinds of rigorous testing procedures. I myself have looked at three other company's test and I have not detected… I haven't looked for that I guess so I can't say anymore. But I'd be very surprised if the kits that are used at the moment, perhaps initially, but the kits that are used today. By registered companies, by Thermo-Fisher or Takara or whatever. I think their probes would not detect non-specific amplicon. I would be very surprised if they did. But I can't say… I can say that the tests that I've developed, that we've published is completely specific.

EFC: Okay. Have you designed your own probes and primers for SARS-coV-2?

SB: I design all my own assays. Yes. Have you seen my paper?

EFC: Which one?

SB: In Nature Scientific Reports.

EFC: The one on SARS-coV-2.

SB: Yes.

EFC: I have not. Would you please send me a PDF of that? I cannot get in research gate.

SB: Yes, of course. Because we've also published all the underlying data that supports… So, I publish everything when I publish. So it's got all the data. Okay, that paper explains to you all about CQ and it also explains about probes. And what I've found… Because we've found that there was a few mutations in the primer and probe sequences and you can get permissiveness

of the probe. In the case that we've looked at, the probe will detect the target even though it has a single base mismatch. But I will send you those papers. I will also send you a pre-print of an article that I've authored which is just under review at the moment which discusses this whole issue of CQs and that will probably help you as well. Okay, yeah. I will do that. | **00:34:42**

EFC: So, one other thing that Battini said is that since April it's enough to have a positive signal. This is not in your lab, by the way. Somewhere else, everywhere else. If you add that since April it's enough to have a positive signal on a single gene of the nine most common targets normally used whereas at the beginning a simultaneous positive signal on at least three genes covering the entire sequence were used. Is that right?

SB: Not quite. The initial description of the assays targeted I think three different regions of the genome, yes. My test tells you also targets 3 different genes. I know of at least two companies, one is called Primer Design and the other is called Eurofins. Their assays target two different viral genes. Others, I don't know. No, in fact a third one, a Chinese one called Sun Shore also targets two. I think that most commercial assays target two genes. One in the **_____39_____** and one usually N or usually the N gene as well. So I think that as far as I'm aware, most but certainly the four kits I'm aware of, all targets two genes. | **00:36:18**

EFC: Okay. At least two…

SB: I think it is important to target more than one gene, yes.

EFC: I would say…

SB: You can get a mutation in one and then your primer doesn't work anymore. So I think two is the minimum. I would be happier with three. Yes. And that's why we have three.

EFC: So, but what Battini is saying that in most of these tests that are being used around the world, among other problems, is they're only looking for a positive signal on one gene.

SB: I don't think that is correct. But what is correct to say, and I know that from my hospital. If they have one positive and one negative, they call it positive.

EFC: I'm sorry one positive and one negative…?

SB: They call it positive.

EFC: Yeah. Right. Yeah.

SB: So, I think the way I would put it is that most tests do look for both targets but if one is positive and the other is negative you really should repeat that assay. But they don't have the time or I guess the man power to do that, so they just call it positive.

EFC: Right. I mean I'm trying to figure out… My background is in toxins. And I know how much care has to be taken and that really you only trust split samples. You only trust the sample… you know, you take soil and you shake it up and you send it to two labs, and you see what you get back from two unrelated labs.

SB: Yes, there's no question that… and this has been done against the criticism that… you'll see the paper I'll send it to you. About 15 years ago a study was done in the UK looking at influenza and they sent the same sample to 20 different labs and they got wildly different results. | **00:38:02**

EFC: Yeah.

SB: So that is definitely a problem.

EFC: Right.

SB: However, I would say that … Again, I don't know about the United States but in the UK after a lot of initial problems I think that the results we're getting back now are reasonably reliable.

EFC: Yes. The thing is that even if we don't use Koch's postulates we can at least consider the idea that you shouldn't be able to find the pathogenic agent in 90% of people who don't have any symptoms.

SB: No, because you can get asymptomatic transmission as you can with bacteria. You can have cholera and not have any symptoms at all. You can still harbor… Remember the typhoid Mary in New York at the beginning of the century. Remember her.

EFC: What do you make of the study that came out a couple of months ago in, I believe in November, in Nature, that was done in Wuhan studying 9.5 million people in Wuhan that said they could not find asymptomatic spread. Published in Nature, so far no issues.

SB: I didn't see that. Send me the link and I'll have a look at it.

EFC: Happily. I'd be happy to send that. So are you familiar with the paper by Borger et al, called the Corman-Drosten retraction paper.

SB: Yes. Yes.

EFC: What are your thoughts on that?

SB: I think it's a disgrace. We've reviewed that paper. The call for retraction. That's the one you're talking about isn't it?

EFC: Yeah, is it valid? Are the reasons in the call for the retraction valid reasons?

SB: No. It's an absolute disgrace. | **00:40:00**

SB: Hello.

EFC: Hey there. Hey there.

SB: I don't know what happened there.

EFC: Digital is not perfect.

SB: I can't comment anymore on that because I am part of a group of people who was sent that paper for reviewed and we've reviewed it.

EFC: Oh, that have reviewed the retraction demand?

SB: Yes. For that Journal. European… I can't remember what it was called. Euro… something or other.

EFC: Eurosurveillance.

SB: That's it yeah.

EFC: Oh, so you're on… Because Eurosurveillance was… they were… There was insistence that they retract that paper for diversity reasons, including the lack of peer-review. It was only published 48 hours after submission.

SB: Yeah. All I can say is that we reviewed that call for retraction and gave our opinion and in fact I was in contact with one of the co-authors who was on that paper and I said to him look, we've reviewed them and passed it back and Eurosurveillance said they would give you their information. So I have no idea, but I can't really say anything more about that.

EFC: That's fine.

SB: I can say to you that that is a disgraceful piece of work, that whole thing.

EFC: Okay.

SB: There's no truth in… yeah. Let's leave it at that.

EFC: So the thing I'm wondering is thought that everyone admits that in silico primers are being used because of a lack of clinical specimens. I don't understand that. Even the CDC says because there is no clinical specimens they are using a hypothetical virus.

SB: Recently? When?

EFC: No, it's even current on the website. It was current as of July, then it was republished and…

SB: But who? Who's using in silico analysis? | **00:42:01**

EFC: It seems to me like Corman and Drosten admit that they're using in silico and that even… Here's how the CDC put it… Let's see if I can find this. I've got to… CDC says… This is under the analytical performance, limited detection, and it's saying that since no quantified virus isolates of 2019-nCoV are currently available… And this keeps being updated. It's still current. Assays designed for detection of 2019-nCoV RNA were tested with characterized stocks of in vitro full length transcribed of gene bank a session MN908947.2.

SB: Mhmm.

EFC: And it seems everyone is saying that this is all in silico as was SARS-coV-1 was in silico.

SB: Well, the timeline of SARS I understand… as I understand it was but the 10th or 11th or 12th of January the Chinese published the sequence of the isolates that they have isolated from the patients in Wuhan. And from that sequence the original Drosten primers were designed in silico obviously because they had no access to any viral RNA at that stage. So the whole of the design was an in silico design that it was probably a mistake and I think that one of the… They also had redundant wobble… Okay, some of the seq... Normally you design a primer with actually sequences ACGTEE or rather ATGGAC and D. What they did was some of their primers had what we call wobbles, so they had more than one position possibility because they weren't quite clear about the sequence. I think that's how it works. And that's why the first set of primers for everything but the E gene have not been used anymore because they're not very good. And the reverse primer had a mismatch that reduced the sensitivity of the RT step. So, the first lot of primers were not a good set of primers, but they were the only ones they had based on the published sequence. But very quickly after that, for example: I became aware of that at the end of January and I had my primers for the… again for the published sequence in the middle of February. So at that stage all I could do was… And it's what I did. I ordered synthetic oligonucleotides that allowed me to look at my… the primer design without having any idea whether they would work in vivo – on real samples. It was only after we started getting cases in the UK and I was able to go to my local hospital and get samples of patients from the hospital that I could then confirm on real samples that my in silico designs and validated actually worked very well on the actual samples. So that is exactly the same with the Drosten primers.

EFC: Right. See…

SB: So I have no problem with the… that we all… if we don't have any RNA… if we don't have access to the actual material we have to design in silico and validate using artificial templates. That's the standard way of doing it.

EFC: Mhmm.

SB: And then once you get the actual sample of virus or bacterium or whatever, then you can test and hope that it works on the real thing. And if you have the right sequence it will work on the right thing. And then that's when they found that their primers didn't work very well because there was a mistake. | **00:46:06**

EFC: But this was used to declare a pandemic. So you're saying that they were wrong. It's kind of like Einstein's experiment, he got it wrong, but he got it right? With the eclipse, you know, where he's measuring the light bending around the star when it's occulted.

SB: There was a mistake in one of the primers. That's all… But the other to sets were okay. I'm not quite sure what the problem is because what was obvious was that the diseases, this pneumonia was being caused by this virus. And this virus started popping up where more and more people were coming down with the same symptoms. And these primers were detecting that virus.

EFC: Right.

SB: So, initially not terribly efficiently but by March it was completely clear… In fact, there were several kits out by then, including my own. I compared my assays to ones being used in our local hospital. So, I have absolutely no doubt whatsoever that what my tests and other people's test are detecting is SARS-coV-2.

EFC: Right. So, let's assume that's true. We still have an issue of 90% false positives being used for of these draconian measures to lock down civilization and make kids wear masks when they're swimming.

SB: Right. You see. I think this is what the problem is. You're now moving on from test results to a clinical…

EFC: Well social design based on them…

SB: They're not really false posi… False positive in PCR terms is a contamination or a mis-designed assay. So, you never ever get a false positive in PCR if you have all the controls present and your assays work properly. | **00:48:03**

EFC: But those include having your re-agents correct, it includes having quality control in the lab, it includes having competent personal, it includes…

SB: Yes, exactly. That's all important.

EFC: QAQC

SB: Now, if you don't have that you can get false positives. But I think what you're probably referring to is the fact that something of CQ of 35 or 37 is detecting something that isn't infectious so these people are not really transmitting the virus. So in terms of the PCR test they are not false positives, but in terms of the interpretations of the tests they are false positives.

EFC: Yes, that's fai… I will certainly grant you that. That this data is being radically misinterpreted and misused for political purposes it would seem.

SB: Well, that I can't comment on. But…

EFC: Yeah. I understand why. I understand why.

SB: I don't even think political… yeah… All I can say is that from a PCR point of view, they are real. From an interpretation point of view, they are open for discussion.

EFC: But how can you call somebody who shows up with a positive at 40 cycles, who has no symptoms what-so-ever, a confirmed case of an infectious disease?

SB: Yeah. I think that two possibilities that I said earlier, he could be about to develop the disease. So if you tested him two days later he might have a sore throat and his CQ now is 25. Or he had it last week and the virus is on its way out, and so it's no longer infectious. Certainly, at a CQ at 40 that person is not infectious. But it could be hours away couldn't it? It could be infectious in a couple of days' time.

EFC: But we're not seeing that. Really, I mean if there is a high case load in hospitals that's often the case. The spikes that we saw in 2020 were matched in previous years. It wasn't like there was some unbelievably shocking thing going on, only that never happened before and only happened in 2020. | **00:50:16**

SB: Well, in this country and in Italy and in other countries it has been unprecedented.

EFC: Well, there is also the comorbidities issue. Right? I mean when you've got 94% of the deaths are people with average 2.6 comorbidities and most of them are at life expectancy… you can't really say that that virus is the thing that killed them.

SB: Well, I have several colleagues in their 30s, 40s, and 50s that came down with this disease in March and several of them are still suffering from the aftereffects. One of them is a musician. She plays the clarinet and she can't play the clarinet. The other one can't go for a walk. She tires out within five minutes of walking. So, there's to aspects to this disease. One is that it kills old and vulnerable people. However, you define old and vulnerable. But the more insidious aspect of this virus is that it targets all kinds of organs. It goes to the brain; it goes to the heart, and it causes disease and what you call lung Covid. You know, where people suffer from it for months afterwards.

EFC: Mhmm. Right.

SB: So, I don't think anyone can deny the fact that this disease can be a serious disease. In most people it isn't but in a significant cohort of people it is a serious disease.

EFC: Right. I mean… and that's… you know, if we start from that premise then we might want to look at Roundup and Non-Hodgkins lymphoma, right? Because it's not like this kind of attention is given to all the known things that make people sick. They're spraying Roundup like crazy on highways now and nobody cares about that and people think it's wonderful to put it in their vegetable gardens. | **00:52:10**

SB: Well, yeah. I think, yes there are lots of ways of looking at it but I think we panic because we weren't prepared for this and because it was a new virus, it just jumped into human population it spread extremely rapidly and because we weren't able to contain it, certainly in the UK and **_____54_____ 00:52:33** Italy. Our hospitals very rapidly became overwhelmed and that's why we have to lockdown and prevent transmission because that's the only thing that stops the virus if you have nothing to fight it with. You have to stop transmission and that was very successful. And what is obvious every time you loosen the lockdown. UK is the best example. We opened up immediately. We had our 'eat out to help out.' Encouraged people to go on holidays and immediately it comes back and the only thing that stops it again is the lockdown.

EFC: Right. But you can't lockdown forever.

SB: No. But that's where the testing comes in you see. What you have to do is, you lockdown hard, bringing the virus numbers down and then you have to test and pray efficiently. And again, we haven't done that here. They have done that very efficiently in Korea, in Taiwan and that's why they've got it under control there.

EFC: Right.

SB: We haven't don't that and you haven't done that and that's why we're the dummies… We're both as bad as each other in that respect. Except you've got the additional political aspect which I don't understand. You know, why has this become a political issue.

EFC: Yeah. You mean why it's like a democrat virus and not a republican virus.

SB: Yes…

EFC: Only democrats believe in it and Republicans don't.

SB: Well, I think it's clear that your political leadership failed significantly on that.

EFC: I would say…

SB: That's another issue…

EFC: And who is Tony Fauci anyway? And why is this such a business deal? What do you think of the use on an MRNA vaccine? | **00:54:06**

SB: It's interesting, isn't it, that of the vaccines that have been produced, the MRNA vaccines are the most successful ones and the traditional ones don't seem to be working properly. So GSK, Merck, Sanofi, all those traditionally produced, and even the Chinese one are either non-effective or very low effectiveness. MRNA vaccine was always going to work. You are probably familiar with this, but we viewed MRNA for a very long time to try and deduce therapy. The problem has always been we can't target the RNA. If you want to correct genetic effect in a certain type of cells, you have to get the RNA into the right cell.

EFC: Hmmm

SB: And that's the difficulty: we can't really do that very well. It hasn't really been detected. With pathogens it doesn't matter, we just have to get into any of the cells. It will produce the viral protein and then they become neurogenic. That's what is working so well.

EFC: Is that really a vaccine?

SB: Well… its' viewed as a vaccine. We inject it and it raises an immune response, so it is a vaccine, yeah.

EFC: Okay.

SB: It's a new kind of vaccine. We are revolutionizing the world of medicine, in my opinion.

EFC: Yes. Well now we are in the uncontrolled phase of the experiment on the public and we have to see what happens over a period of years, right?

SB: Yes, but I think it's already clear that its' been influencing Israel. It's reduced the infection rate. In the UK it's beginning to now. I think it's you know, yeah.

EFC: Why did Fauci say, why are a lot of people saying, that it doesn't interrupt transmission; it only reduces symptoms?
SB: Well, I think we don't know that yet. You could imagine that you vaccinate, so you know you meet somebody who infects you. So you breathe in and your body responds to it by heating it up too much; it replicates in your nasal mucosa. | **56:03**
You might be infectious for a day or two, even though you yourself are being protected. We do not know whether that happens or not. It looks as though it doesn't happen but I think it will

certainly be… there's evidence that the fiber of the moderna and the academic of vaccines prevent emission but I think the jury is still out on that.

EFC: Right.

SB: Also, if you're naturally infected, the virus usually comes through your nose and you have a different kind of …

(lost connection)
EFC: can you hear me?

SB: …They are slightly different. It may be that a natural infection makes it less likely that we transmit in future than in vaccine but we just don't know yet about that.

EFC: Right. Right. I mean it seems like if there's something replicating in your nose, then Neti pot with zinc might help, like a nasal rinse with zinc. It seems like there's a lot of things besides a vaccine that have been shown to be helpful and preventive including zinc.

SB: Why wouldn't you want to have vaccine. Vaccines are the most easily administered and protective thing you can do. Why wouldn't you want to do that?

EFC: I would say maybe it depends on your orientation. I personally believe that Nature did a fantastic job with the immune system. We have this beautiful thing called cross immunity, T-cell immunity, B-cell immunity, memory-cell immunity: immunity of all kinds, all kinds of natural defenses the body has to help us.

SB: You wouldn't want to vaccinate Smallpox? You wouldn't want to vaccinate Yellow Fever? You wouldn't Measles or Polio? Vaccinations is the ONLY way to protect the populations from infectious diseases. You know, Ebola, there's now a vaccine. We have the vaccination for Dengue, and that devastates people in Bangladesh. The only protection against developing a disease is vaccinating against a pathogen. | **58:20**
I don't think you'd happily not have a Polio vaccination.

EFC: So my understanding of the history of Polio is that it starts to become an issue when our lives became more sanitary. When our lives became more sanitary, people were not exposed to the lesser forms of Polio and developed cross-immunity. This is according to the Viral Theory of Polio: that when our lives became more sanitary, and people were not exposed to dirt and grime and all that stuff, then the more pathogenic versions of the virus could jam in.

SB: No. That's not how things work, no. Let's take another example. Let's take Smallpox. Smallpox was the scourge before Jenner found Cowpox, I think that's there no question that, no one should really argue that vaccination isn't protective and helps us fight infection and disease particularly in the poor and poor countries where people are dying from all kinds of disease, including Typhoid and Salmonella, and God knows what else. That's my opinion and I could not

argue any different. Look at Measles; Measles are a killer. Measles vaccine protects against Measles.

EFC: But there's famous litigation in Germany from 2017 that has essentially proven that this virus has never been isolated and purified. | **01:00:06**

SB: Which one?

EFC: I forget the name of the litigants, but it went to the High Constitutional Court in Germany.

SB: Which virus?

EFC: Measles

SB: Yes, Measles has been purified. We can look at pictures of Measles virus.

EFC: Can you, when we correspond after this conversation, could you send me the paper showing the purification of Measles?

SB: Yeah, I'm sure I can, even if I need a certain amount of time to get them together, okay?

EFC: Yeah, it's not so much. There's the current paper on Sars-coV 2.

SB: Mmhmm

EFC: I'm going to send you the nature piece.

SB: Yes, please.

EFC: … on asymptomatic. You said you have something on purification of Measles, maybe.

SB: Well I have a, don't forget I have a, or you may not be aware of the fact that I was involved with the MMR Autism trial in Washington.

EFC: In Washington state?

SB: Washington D.C. Back in 2007, there was a case, which was held in front of the special court that we have in America.

EFC: Yes, yes, yes, yes, the Vaccine, the HHS Court of Claims.

SB: Yes, yes. So I was an expert with the Department of Justice. In fact, it also revolved around RT-PCR, showing that the RT-PCR results were showing the presence of Measles virus in the intestines of Autistic children were all false positive results due to contamination.

EFC: Ahh.

SB: That was my association. And from that I know that I have pictures of the Measles virus. I'll dig that out and send it to you.

EFC: Interesting. So one thing I'm interested in, and then I have one question about Spanish Flu, but the studies showing about purification and isolation of Sars-coV-2, I have not seen anything that to me meets the standard of purification and isolation. Everything's run through other cell lines and put into a broth, and then they PCR the broth and they find their target.

SB: They see it; there's pictures of it.

EFC: Right, but why is it that every government, when public records, including United Kingdom, and the United States, say, "we have no clinical samples of this"? Why do they all say that if it's not true?
| **01:02:15**

SB: have no idea. That's not my area of expertise but as far as I know the original samples from the first paper that was published in Wu, what was it? Wu… has all the purification, all of the evidence, including electro micrograph. And the first patient that was detected in the United States, his RNA or his viral RNA formed the basis of all RNA samples then sent out by the CDC to labs in America. So, it has definitely been isolated and cultured from a patient sample on numerous occasions including in the United States, but I don't know.

EFC: But there's two different definitions of isolation going around though. One is that you separate it from all else, and the other is that you put it into a broth and you find it.

SB: Yeah. Well, that's not really my area of expertise. As far as I'm concerned, I've read the papers and if that's the standard way of isolating a pathogen, so I have not problems with that.

EFC: Well, it's the current way that's used, I would say, that the idea of true purification you separate it into centrifuge, and you know you've got a sample of only that. And then that is the thing that is sequenced and then used to prime the PCR. It does not appear that that's what's happening. From my limited, you know, a year into this…

SB: Well, the way the sequence was established by taking the samples from the original patient growing up something and then sequencing it and then disassembling the sequence and what came out of that was the SARS virus. Which then very closely resembled a bat SARS virus. And was obviously a different one. So, that… Well, you know, this is a standard way of doing this so I really can't comment any further on that, except that to me that's perfectly acceptable and that's the way to do it. | **01:04:29**

EFC: Right, okay.

SB: You then want to take that virus… whatever you isolate then and infect a tissue culture. And they've infected animals, haven't they? And they've become sick, so I don't see a problem there.

EFC: Okay, I'm mean I'm just… You're the first convincing person I've heard argue that side.

SB: Mhmm.

EFC: You know, because very few people want to say much anyway, right?

SB: Right.

EFC: You know… So, that… you know, and I can bring you into that discussion with this other biochemist I'm working with who are saying, "well…, and doctors, we've got our own earlier definition of isolation which means, separating it from everything. And all the isolation that's happened with SARS-coV-2 has been through a process of crumbling it into a broth of other cells, an impure broth and then finding it, using Q-PCR.

SB: Okay, let me go back to the original Wu Paper and have a look at it again and I will get back to you about that as well.

EFC: I would love it.

SB: Okay, and I will give you my opinion, but I'm really trying to lay it out for you so that both you and I understand it.

EFC: I would love that. I would be so grateful to have that. One last question.

SB: Mhmm.

EFC: There's an August 2, 1919 paper from Jamma by Milton…

SB: 1919.

EFC: 1919. It's 101, ah, it will be a 102 on August 2nd. | **01:06:01**
Experiments determined the mode of spread of influenza. And they took a bunch of people who were sick with Spanish flu, as it was called.

SB: Yes.

EFC: And they took volunteers from the military and from prisons who were not sick with it and they couldn't… and they had them breathing down into the mouth. A sick person breathing into

the mouth of a healthy person. They were inoculating the persons nasal passages and throat with sputum from corpses and also actively infectious people.

SB: Yes.

EFC: And they couldn't transmit one case of Spanish flu.

SB: Really?

EFC: Yeah.

SB: Okay, I'm not aware of that.

EFC: And the doctor... Yes? The scientist…

SB: Bear in mind, I'm not a virologist.

EFC: Yeah. The scient…

SB: I'm not a virologist.

EFC: You're not… Ah… What do you consider your specialty?

SB: I'm a molecular biologist. I'm a… My specialty… well, nucleic acid detection, PCR transcriptional control regulation, that kind of thing. I'm not a virologist. Bear that in mind.

EFC: Got it. I mean this is why you're doing this… these molecular tests. This is not…

SB: I can give you very detailed and I think convincing answers to anything to do with the PCR and molecular technologies. And as far as anything else is concerned, I can give you an informed opinion based on my experience with these things. But I'm not an expert on isolating viruses. I just know how it's done and how pathogens are being isolated and identified. But I don't do that sort of thing myself. Bear that in mind.

EFC: But you accept running it through a cell culture as being a form of isolation.

SB: Yes.

EFC: As opposed to getting clinical samples from 500 patients with same symptoms and making sure they're all the same for example. | **01:08:04**

SB: Well, I think you want to do both, don't you? But what you'd normally do is you get the sample from the patient and then run those… Once you've identified what the pathogen is then you can do the PCR. You can't do it beforehand. That's the problem.

EFC: Yeah, you need… Right, you'd have a tautology. If you don't have the isolate first… If you don't have the known pathogen first the PCR can't be valid. It's like sending the dogs out to find the specific person without the sample of their scarf.

SB: But what you can do is, you can take the sample and sequence it. And they don't need any sequence information. And then you simply assemble the sequence and see what comes out. And then you find that there is some pathogen that you've never seen before. And I think that's how they identified the virus. But let me go back and… but bear in mind that sequencing is… PCR requires you to know how to sequence information. Sequencing doesn't.

EFC: Wait, say that again please.

SB: PCR requires you to know what the sequence of your target it is.

EFC: Yes.

SB: Otherwise you can't do PCR.

EFC: Yes. Yes. Yes.

SB: But if you want to sequence something, then you can take a sample and just blindly obtain sequences.

EFC: Yes. But how do you know which to target? There's so many things. If you cough into a tissue, how many viruses are going to be in there? How many bacteria are going to be in there?

SB: There'll be very few, very few. If you take a blood sample, there'll be no bacteria at all. And there really should be no viruses either. If you take a sputem sample there will be lots of bacteria. But then what you do is, you typically when you sequence and that's the reason why anyone does it like this way. You get, you know, lots and lots of sequences and you just plunk them into the computer, compares the sequences of database, and it assembles the sequences and tells you what's there. So most of these sequences will be human DNA and then there will be the odd bacterium… if it's a… there'll be lots of bacteria, but not necessarily any viruses. There could be the odd virus, I guess. But as something that's known. But suddenly what creeps up is something that you've never seen before. A coronavirus whose sequence has not been published and then you know you got something new. In this case coronavirus could something else. So you do not need to have purified sample to be able to identify a pathogen. | **01:10:26**

EFC: Okay, but you might need it to test on other beings, right? On animals, like how do you know what your inoculating your test animal with if you don't have a purified isolate?

SB: Of course. So once you know that there's something in your sample, then you would try to isolate it, yes. And then once you've isolated it, then you sequence it again, or PCR it up. Make

sure it's the thing that you think it is and then you would take that purified sample and administer it to whatever… yes… animals.

EFC: Right.

SB: And remember… I'm not sure how far they ever got with it, but there were going to be some challenge trials with the vaccine. Do you remember back in November or so?

EFC: Yes. Yes. Yes.

SB: Which was going to take the virus and inoculate healthy people… and yeah… I don't know if that ever happened or not.

EFC: I'm going to add that to my list of follow up things.

SB: Yeah, that would be the obvious proof that if you take a 100 people and give the virus to those 100 people and with and without a vaccine and the 100 people that have got the virus get it and the ones that don't, don't. Then it's clear that (A) the virus causes the symptoms and (B) that the vaccine protects you from it. Now, I thought that was going to be done but then there were some ethic… I think it is unethical to do that, but you know, I don't know what ethics were in the ends so I can't give you any more information about that.

EFC: Well, here in 1919… I'll read you the last paragraph of this paper. McCoy and Richie (doctors) did a similar series of experiments on God Island in San Francisco and used volunteers so far as known had not been exposed to the outbreak at all and who had negative results. That is they were unable to reproduce the disease when they exposed them to sick people. | **01:12:12**

SB: Interesting.

EFC: Perhaps there are factors or a factor in the transmission of influenza that we do not know. As a matter of fact, we entered the outbreak with a notion that we knew the cause of the disease and were quite sure how it was transmitted from person to person. Perhaps if we learned anything it is that we are not quite sure what we know about the disease. Period. End of paper.

SB: Yeah. I mean I really can't give you any more clarity on that, but I know that about 20 years ago they unearthed in a cemetery in the permafrost in Iceland or Norway, that's it, in Spitsbergen. In Spitsbergen it was. But there had been an outbreak of Spanish flu and they identified the actual isolate, the viral isolate, the influenza isolate from those frozen corpses.

EFC: In where was this. Where was this?

SB: Spitsbergen. In Spitsbergen, in Norway.

EFC: Yeah. Ahhh…

SB: So they did find influenza virus in these people who had died at the time of the influenza… Spanish flu.

EFC: Hmmm… That's interesting. Well, I'm sure the homeopaths will be glad of that because they only have 200c of that virus, because the samples are so distant into the past and perishable. Nobody froze them for a century. Right? Could you keep… you know…

SB: Yeah. I remember because when I worked at the Royal London Hospital are virologist was involved in that project. So… I think…

EFC: That is exciting. Does anyone know the name of that paper.

SB: Yeah. I'll try and find it for you. I'm trying to remember the name of the virologist. I will get the information to you. | **01:14:08**

EFC: But it was in Norway, right? That's what you were saying, it was in Norway?

SB: Yes, Spitsbergen.

EFC: How do you spell it.

SB: It's S-P-I-T-S, Spitsbergen, B-E-R-G-E-N. It's a group of islands in Norway.

EFC: Got it. Spits… The Island of Spit… (Laughs)

SB: It was a mining community. Yeah.

EFC: I'm kidding. Droplets. You know? Spitsbergen. It's a spit mountain.

SB: That's it like. Spit. That's it. (Laughs)

EFC: Okay. You know the last thing I'm curious about and I mean I'll send you the statement from CDC of why it is they're saying they have to use a mimicked clinical specimen to prime the PCR in the United States. That's the very language they use. In the absence of a clinical human specimens we're using a mimicked human specimen.

SB: Was this at the beginning of the epidemic?

EFC: No. No. Now. The paper is still up on the CDC website. They updated it in September. It was up in July. They re-wrote their document. They updated it in the autumn. The same statement is in the same slightly rewritten paper, but it's the same quote.

SB: Okay. If you send me the link, I'll have a look at it.

EFC: Happily.

SB: And I don't know why they'd want to mimic it. I don't know.

EFC: That's what they said. That's what… And this is why there's so much mistrust. I mean… and I have an obligation to report that they say that. Right? I can't just pretend they're not saying that.

SB: Sure. Sure.

EFC: And I'm glad I talked to you. And you know, you actually go and get samples yourself. I mean, that's my kind of science. You've got to go out with a shovel and your sample jar basically.

SB: Of course. Yeah.

EFC: And go digging. That's been the approach I've taken with toxins. If the state says it's safe. Okay, give me a jar and a shovel and I'm going to go get some and send it to my lab and then we'll talk. You know? Then we'll have a real conversation. | **01:16:08**

SB: Yeah. Okay.

EFC: Good. Listen. Thank you for that awesome writing in the 2017 paper. I'm going to print that out and hang it up in my office. It's good stuff, right? I mean, you guys write like writers. I greatly appreciate that. You're writing to be understood.

SB: Good. I'm always trying. I'm always trying to be understood. Yes. So, if you could send me basics that you want from me and I'll dig them up. Bear in mind I'm doing lots other things, so I'll get them to you as soon as possible. Okay?

EFC: You've been very generous with your time and I'll get these to you in an hour or so.

SB: Okay.

EFC: Alright.

SB: All the best. Take care.

EFC: Thank you so much. Bless. Bye for now.

SB: Okay, cheers. Bye.