

19: Bret Weinstein - The Prediction and the DISC

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(00:00:00)

Eric: Hello. This is Eric Weinstein. I'm going to be recording a short introduction to this episode because I think it's probably the most important episode of The Portal to date. That said, under normal circumstances, I probably would have either edited this heavily or not released it at all. It starts off quite slow and it gets quite awkward before finding its pace. Now what's going on is that the interview subject is none other than my brother Bret Weinstein. In Bret's case, you probably know him if you know him at all as the heroic professor who stood up against what can only be described—I swear I'm not making this up—as an Maoist insurrection at an American college in the Pacific Northwest, the Evergreen State College. It was a very strange situation because somehow the national media that we would normally have thought would have covered such a story—for example, the media that covered the takeover of Straight Hall at Cornell in the 60s—that media was almost absent completely. At least, they were absent for a very long time before they entered late in the game. And why is that? Because the story ran counter-narrative—that is, the students at the Evergreen State College who were behaving in a racist fashion were actually students of color, and this was an exactly counter-narrative story. And Bret, who stood up to this racist insurrection, was in fact somebody with a history of standing up against racism. He had, in fact, been a student at the University of Pennsylvania, my Alma mater, an Ivy league school, and had to leave because of death threats when he stood up for women of color who were being abused for the amusement and the sexual amusement of white fraternity students. So Bret was supposed to be familiar to many of you from that, from an old national news story, and he was also the hero of a book called The Tapir's Morning Bath.

But somehow, the news media, who chose not to report on the Evergreen story, was not very interested either in figuring out who Bret was, because the stories showed that there was a contradictory problem with the main narrative. In some sense, that's going to be recapitulated in this episode. There is an official narrative about what happened in the scientific episode, and there is a narrative which I think is much closer to the truth, which I happened to be one of a very small number of witnesses to this alternate story. Now the key question is whether to tell the story or not, and you're going to see that both of us have a certain amount of trepidation and energy around the question of whether or not to break a longstanding public silence. When Bret found himself as professor in exile along with his wife, Heather Heying, I had thought that the American biology establishment would realize that one of their own had been thrown overboard as jetsam, and that he would have been invited to many universities to give seminars in biology.

It took awhile for me to understand that, because he was found at Evergreen State College, the people who taught at highly ranked research universities thought that Bret was something more like a teacher rather than a researcher. In fact, he had been the top student of one of the most important evolutionary theorists in the United States, Richard Alexander at the University of Michigan, as well as a student of Bob Trivers, formerly of Harvard, arguably one of the greatest living evolutionary theorists, I think presently at Rutgers. Bret was somebody who had actually done really interesting work in his thesis, and for some reason, the system found it very disturbing to consider the full implications of his work.

I think in this episode we're going to do something interesting. I see Bret in two separate ways: On the one hand, I view him as a very heroic figure and he's an absolutely brilliant person. It's been a pleasure sparring with him throughout my life. However, I'm also his older brother and you're going to hear me at sort of my overbearing best, brow beating him a bit. Now the point isn't to push him down, but quite the contrary. I'm rather competitive as Bret's older brother and I don't want to compete with the weakest version of Bret, the professor and exile. Instead, I want him seated again inside of the institution where he always belonged. And in order to do that, I want him to tell the tale, not with embellishment, but as it actually happened, because I think it's one of the most fascinating episodes in modern biology that I've ever heard.

So I hope that you like it. We're going to put it in front of you as an experiment and we're going to test to see whether or not I'm correct that can be used to augment the usual channels.

I believe that a lot of us are sitting on intellectual gold. I don't think that the story that somebody's work didn't see the light of day, or got attributed to somebody else, is as exotic as the institutions would have you believe. In fact, I think it's quite common. I think many of us find that we don't have careers inside of science because something goes wrong quite early when we're quite vulnerable. And my hope is that some of you listening, who I know are struggling as graduate students or as postdocs or as undergraduates, will listen to this and find some courage to stand up for yourself, because, quite frankly, if you choose not to do it in order to make nice with your fields, the chances are you will probably won't have a career in the long term. You might as well swing for the fences and you might as well clear your throat and tell your story as it actually happened, without fear.

I don't know that this is going to succeed, but we're going to run an experiment and I think both Bret and I are up for it to find out wherever it goes. The one thing I would say is that if anyone else in the story wants to tell *their* version of events, it would be an honor to have you on The Portal. There are no bad people in the story, in my opinion; there are a lot of bad incentives. And if we're going to actually fix the system, we're going to have to look past the interpersonal. But the point of this, in my opinion, is that I think it's sufficient to open the case again and to seat Bret Weinstein inside of the university system—that is, the research university system, where he has always belonged. So have a listen, and I hope you like it.

(00:06:01)

Eric: Hello, you found The Portal. I'm your host, Eric Weinstein, and I'm joined today by none other than my own brother, Dr. Bret Weinstein. Bret, welcome.

Bret: Thanks for having me.

Eric: Okay, well what should we do? What do you think?

Bret: Wow. Well, I dunno. I would imagine a certain fraction of your audience is going through the usual sort of a—

Eric: Well they randomly call us either Bret or Eric.

Bret: Yes.

Eric: So far as I can tell.

Bret: Which our parents also did while we were growing.

Eric: I suppose that's true.

Bret: Including the pets names were also sometimes thrown in, if I recall correctly.

Eric: That's true. Okay. So if you don't mind, I was trying to think about the fact that we have an opportunity to do something that might be slightly different because you and I share a lot, and what I thought is that we should begin to really focus on areas of your expertise with respect to biology rather than the way in which many people have come to know you. So can I ask you to just quickly dispense with, in 30 seconds, how the world has come to recognize you if they recognize you at all?

Bret: Sure. To the extent that I am recognized, it is typically as a result of the meltdown at Evergreen and my stance—

Eric: That's Evergreen State College.

Bret: Evergreen State College in Olympia, Washington, where I taught for 14 years, along with my wife, Heather Heying, who taught there for 15 years. We faced a mob of people who accused me of racism. And these were students, they were students I had never met. And the event was so colorful, and eventually when the world caught on to the fact that the protesters, who became rioters, had uploaded footage to the net, and so the whole event could effectively be seen from their perspective, it raised interest in some other quadrants. So, for example, I ended up on Joe Rogan's program, which is the place I'm probably most recognized from. And

you know, my first appearance there, we talked about the Evergreen situation. And anyway, that's the bulk of how people know me.

Eric: All right. So you were a biologist teaching at a relatively obscure college that had previously been known for social activism. And I didn't love your introduction because when you say, "Well, the students accused me of racism" that leaves sort of a weird question. Like, "Why was he accused of racism?" Let me solve the puzzle just immediately—maybe you can't do this—because that was the closest we'd seen to a Maoist takeover inside of the United States of America, ever. Like, it was a case of mass insanity, and the videos showed it to be mass insanity, and unless you had been indoctrinated to believe that Maoism of some form, Maoist re-education, was normal, the rest of the world said, "OMG, what the heck is going on at this completely insane—". It wasn't just like one of these college craziness pieces. This is really an episode of broad institutional madness that was localized there. And I want to take it to be self-evident because it is self-evident. The video exists. And if you took the people who were trying to pretend that you were a racist in their own terms, that was sufficient to—it was like the unreliable narrator. They were, debunking themselves in the eyes of everyone who hadn't come under the spell of this particular kind of madness.

Bret: Well, there's a little more to it in the sense that they were entirely unprepared for a white guy willing to say, "No, I'm simply not a racist". And it just didn't occur to them that that was going to happen. And it didn't occur to them that my own students weren't going to flee to their side at the point that they leveled their accusation, because those things would have been normal in this environment. But, in my case, I grew up in a home—there were plenty of flaws in that home, as you know—but one of the places I don't think it was flawed was that it was very clear-headed about issues of inequality, race justice. And so I, I really have the sense that these issues were really not new to me, and I had a long history at the college, lots of students of color—

(00:10:43)

Eric: You're explaining too much. And I don't mean to be rude about it, but, they were just crazy.

Bret: They were crazy. But my point is, the accusation is in and of itself so powerful in modern circumstances that people, the idea of standing up to it doesn't occur to most people. And the fact is I was not well enough positioned. The thing descended into madness. It descended into literal anarchy with armed students, roving the campus, the same mob was looking for me, searching car to car, for example. It was a very dangerous situation

Eric: With baseball bats

Bret: With baseball bats. But what I'm getting at is I checked with myself and did not feel vulnerable to this accusation. I felt most people could not endure it, but I was in a position to, and in an odd way—

Eric: You'd been effectively driven out of your own university as an undergraduate, standing up against racism.

Bret: Indeed.

Eric: These people had flipped the script, and said, if you don't sign up for our racism, you're a racist. Yeah.

Bret: They did.

Eric: I don't, you know, here's the thing. I have two documents that I've studied that have a lot of longevity to them. One begins with, "We hold these truths to be self evident" and the other one begins with "In the beginning". And I think we've made a huge mistake taking this as an argument. It's a non-serious position held by morons and idiots, or people who've been indoctrinated and infected with an idea that there's something left-wing about being a racist. I'm not interested in it, and I also think that it's really important to stop giving these people their due. Like, it's really important to exclude them from the conversation, because if you have to have a three day symposium as to whether or not racism can be redefined in a way that makes it impossible for certain people to be racist but impossible for other people not to be racist, there's just no point. It's just needs to be thrown in the garbage because it just, it's a suicide idea that wastes everyone's time and plunges the world into stupidity, madness and hatred.

Bret: Well, you and I are in total agreement about the necessity to shut the bad actors out of the conversation.

Eric: Great.

Bret: I do have some concern about a large number of people who fall into one of two camps. They're either confused, or they suffer from so much cowardice that they will sign up for ideas that they ought to know are wrong.

Eric: Yeah. But I think you're not getting the message. We've made a huge mistake, and I refuse to spend time, because these people have decided that this is a tax that we should pay, that they have a serious point. It's a non-serious point. It's a terrifying moronic non-serious point that you can redefine racism to be anti-racism and anti-racism to be racism.

Bret: Nobody knows this better than me.

Eric: Great. Okay. Are we done?

Bret: We are.

Eric: Good. With that aside, my concern—you know that I play this game, which is called “What is the least interesting, interesting thing about X?”, where I take a person and I take their top characteristic. So for example, the least interesting, interesting thing about Dolly Parton is that she's busty. The *most* interesting thing is she's a genius level songwriter, and a fantastic singer, and an entertainer, and a great business woman. Doesn't matter. But the key point is we get hung up on some stupid superficial characteristic, and we don't see the actual interest or majesty in a person, and I feel like that has happened to you. I feel like, at some level, having known you for a very long time, you are an incredibly interesting person for totally different reasons than the reasons for which you have become famous. And I would like to use this episode and, by the way, you're welcome back anytime. Love to do a series with you. Love to, you know, make this a regular part of our lives if people like it.

(00:14:43)

Bret: Cool. I think you know, you and I both hear a lot of curiosity about what our relationship is like, and what our discussions sound like. And so I think there's lots of room for that.

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Eric: What I'd like to do is to try to be the foil for you that I don't think anybody else can be, because I was tracking the story very early. And by the way, when I originally tried to get you help and allies, I think almost the only person who could get what was happening at Evergreen State was our mutual friend, Sam Harris, who was willing to amplify and retweet this, because it was so confusing that most of the rest of the world had just never seen these kinds of arguments. And now it's much more common for people to be aware of these problems. But when it started happening, we didn't even have any framework for how to think about these things.

Bret: Yeah, and in fact, Sam, I remember even the content of his tweet where he entered this discussion, where he suggested that what was necessary was a deprogramming for these people. And from living inside of this very confusing scenario, to hear a message of reason from the outside, that it was visible how insane this was, meant a lot to me. It really, it changed things. It was like a reality check.

(00:18:00)

Eric: Yep. Sam's a real hero in that regard. It's just amazing that he got there early and he got there correct. And, you know, more power to him.

Bret: Yep.

Eric: Okay. As you know, I was not happy about you being at Evergreen State College, long before this problem was occurring. I viewed you as sort of retreating into this very obscure college and using the undergraduates as if they were graduate students, teaching very advanced concepts, and running kind of a weird Harvard-style program with very adventurous material, with no recognition that this kind of unusual educational environment was even occurring. Fair? Unfair?

Bret: Well, it's mostly fair. It was not really an appropriate place. I don't regret it. I think for the last year or two, Heather and I were living on borrowed time, that this could have come for us in a worse way, and it could've come for us at any moment. But the thing about the job I had was that it was the upside of a crazy experiment in education. The founders of the college had broken every rule of a normal university, and half of what they did in breaking it was crazy, and half of what they did was brilliant. Nobody ever bothered to separate the two from the prototype, and, you know, fix the broken part. Didn't happen. But, the administrators had no power, and very little knowledge about what was going on in the classroom, which meant that I could create

a learning environment that worked both from the point of view of students and worked from the point of view of me in my objectives to keep advancing a research program that frankly I would have had no way to keep on at a normal college. I would have been so burdened by teaching that I couldn't have combined the two things. So anyway, I do think one has to figure out how to make their way in the world financially. One has to figure out where to raise their kids. And from many perspectives, as much of a mismatch as Evergreen was for me in some ways, in some other ways, it was not a bad place to be parked. It gave me—I was anonymous from the point of the world and I could make progress on biology. So I have fewer regrets than I might.

(00:20:36)

Eric: Okay. This is so uncomfortable, but it is also the real substance of our relationship. I always resented the fact that you really excelled at, and enjoyed, teaching as much as you did, and you saw this in terms of a place to play with ideas, to teach students to have a pleasant and enjoyable life, healthy as it was in the great outdoors, et cetera, et cetera, blah, blah blah. And I still see these characteristics in you, and it drives me nuts because you're your own worst enemy in some ways, to me. What you really are, to me, is an unbelievable thinker and researcher, and beneath this kind of very nice, friendly pedagogue is a thinker that the world doesn't know. And I watched recently your interactions with Richard Dawkins, and it was absolutely infuriating. I mean, you know, he's very clear. It's like, "Well, Bret is a real hero, so far as free speech and standing up for free inquiry goes. But he's very confused." Well, no, I don't think that that's right. I think that you guys had a really substantive interaction about biology, which I wish he would spend more time on because he's phenomenal at it when he's focused on it, and *you're* phenomenal. And that was supposed to be a really different conversation. But because we got to know you the wrong way, in my opinion, you're always the guy who was strong enough to stand up to students at an obscure place, and this completely masks who you've always been, and you're not willing to take up the yoke, which is the more important role for you.

(00:22:21)

Bret: Well, I don't know that I'm not willing. I think you and I have a different approach to this and it may be, you know, birth order stuff or whatever, but, you know, and I also, I have the benefit of you in the world, doing what you do, which, I do wonder sometimes what would've happened to me at Evergreen had I only had my own tools at my disposal. It is quite possible I would have been effectively snuffed out in private and I don't know what I would be doing at the moment. As it happens, the Evergreen story turned into rocket fuel that propelled me into a strata where there's lots of interesting things to do, that may not be exactly what you're talking about, but they make sense.

(00:23:07)

Eric: Yeah, it's frustrating. I'm trying—I don't think you understand what it is that I'm trying to do here. I believe that you're miscategorized, and you're really not grasping that this is my opportunity—

Bret: No, I am. I am grasping it. What I think distinguishes us is that we have *very* different styles with respect to approaching things. I, for example, take a certain perverse pleasure in watching Dawkins slowly move in my direction, which I believe is happening.

Now. I would like him to move faster. He's not a young man and I think it's actually quite important that he recognize where the errors in his own thinking are. And to be honest, I believe I know where at least several major ones live, and I know what he would see if he could be brought to understand the nature of those errors and to confront the, frankly, the portal that opens if you walk through a slightly different door than he's been walking through. But you know, it didn't work in one evening—I always wondered if it would, but there is still the possibility that he will have the epiphany that I hope he will have.

(00:24:27)

Eric: I really don't understand even where we are in this conversation.

Bret: Okay.

Eric: Okay. You're not getting it. You were found at Evergreen State College. That is a communication to the world that you weren't very good.

Bret: Yep.

Eric: And every time I try to say this is completely wrong, you miss— you don't catch the ball that's being thrown to you, which is you're not understanding what you're up against. He doesn't take you seriously because you don't have a list of publications that speaks to who it is that you actually are, or what you've done, or where you've been, and as a result, you continue to be the good guy, who is very well spoken, very thoughtful, says very interesting things, and constantly gives away power to other people.

(00:25:14)

Bret: Mmm, I don't think so. There's a question about how to confront the opportunities that you've got, the hand you've been dealt, and I think you and I share a certain delight—when we do our homework and we discover something interesting and absolutely nobody else gets it?

Eric: Mm-hmm

Bret: That would feel bad to most people, because they would feel like, “What am I doing wrong? Why does nobody else understand this point?” To you and me, that feels good. It is to know that you have achieved something, you have discovered something, and that nobody else can even recognize it, gives you some sort of sense of how far ahead you might be. The question is what to do with those things, and there, I think the question is if I went through something with— I said something intemperate to the new atheists, and suddenly Steven Pinker, Jerry Coyne, Michael Shermer, Richard Dawkins, and Neil Shubin came at me all at once, not on the topic that I had caused offence— on a totally different topic. They had picked something off my YouTube channel. Jerry Coyne had claimed to have debunked it. He was wrong, but nonetheless it provided fodder for them to attack. Their point was that I didn't understand natural selection and that, to the extent I might believe I knew something that other people didn't know, the right thing to do was to submit it to a journal and go through peer review. I pointed out to them that peer review was not Richard Dawkins style, and that he in fact advanced the ball for the field, substantially, but has barely published a paper. That backed them off that course, and their tune changed to, “Well, how about a book then? That's what Dawkins did.” And to me that's a win. The idea— I'm not against peer review. I want peers to review my work, but I don't want it snuffed out in private. And so, to the extent that that little battle was the result of them underestimating me and not knowing that something was going to come back that was cogent and responsive to the world as it actually is, and having them back off their position and say, “Yes, actually a book would be a fine thing.” That was positive movement from my perspective. They underestimated me, and they had to back down. So I can't regret that too much. To me, on a different timescale, I believe I'm making progress toward a goal that you and I agree is the right one, but I'm not sure that coming at it, guns blazing is the way to go.

(00:28:16)

Eric: Well, I'm happy to stop the interview right here and right now, because that's adorable, and it's sweet, and it's incredibly patient, and it's it's a beautiful sentiment, but I also feel like I sat through all of the wars and battles to get your ideas into the world, and I'm not funding that program.

Bret: Does it sound to you like I'm surrendering?

Eric: No, it sounds to me like you're boring me. Like, this is really uninteresting.

Bret: *sigh*

Eric: If I think about what actually happened—

Bret: Yep.

Eric: This is a miss-telling. This is not even honest.

Bret: Okay. Floor is yours.

Eric: Okay. I want to talk about something I'm calling the DISC, the Distributed Idea Suppression Complex, and it has nothing to do with Richard Dawkins and peer review and Jerry Coyne and a bunch of other things that almost nobody cares about. It has to do with about a 50 year period in which great ideas got buried no matter where they occurred. Because great ideas were very likely to be highly disruptive to an institutional order. And between you and your wife, and me and my wife, three of our four theses ran into incredible problems, because they were trying to break really new ground. And the amount of delay that you suffered, I mean you're now 50 years old. This is a very late start in a career. You're coming from a very inauspicious place. You've been fitted with a story, which is "He's a sweet guy who stood up to a mob and that's his claim to fame" and you're not really understanding that you're not being taken fully seriously as a biologist. In part what Jerry Coyne is saying to you is, "Hey, you're really unknown to us. I'm at Chicago. Richard Dawkins was at Oxford." You know, he was the Simoni professor for the—

Bret: Public Understanding of Science.

Eric: Right. The point is you're not part of the Super Club. Don't get confused. You're just, some guys stood up.

Bret: Oh, I understand. That's what's being said.

Eric: Okay, so my point is I don't have time for your fairy tale about a healthy and kind and sweet

Bret: Who said anything about healthy? I'm, look, I'm interested in winning for a couple of reasons: One, the payload. Yeah, the insight that opens the portal to the part of biology we don't know because we've had bad Darwinian tools, and for those who heard that as an attack on Darwinism, it is not. Darwinism needs fixing, and there's nothing wrong with what Darwin contributed— it's what happened after.

Eric: Will you do me a favor?

Bret: Yeah.

Eric: I really, you've got your own podcast. It's called The Dark Horse, right? The Dark Horse podcast. I think this is a great place for you to explore, gradual change, incremental progression, turning minds around, opening hearts, all this stuff. This isn't your podcast.

Bret: Yep

Eric: This is my podcast.

Bret: Right. But we're talking about my life. Am I right?

Eric: We are talking about your life, but if that's what you want to do, I don't know that I'm that interested in doing what I was going to do, which was to try to get your ideas out into the world, curated by somebody who isn't you.

(00:31:43)

Ad

Eric: The portal is thrilled to welcome back, returning sponsor, WineAccess. Now in my family's own tradition, we are more or less mandated once a week to drink. And this gives me the confidence in the era of car service apps to ask the question, is it possible you're actually getting behind in your drinking? Are you having enough belly laughs? Are you breaking out the guitars, breaking into songs? Are you dancing with people you love or at least trading stories to bring you closer together. A great bottle of wine is a way to slow down and get off your phone. It marks time and lets you know something important is happening. Now our friends at WineAccess have an interesting philosophy. They take the most famous vintages and the most famous vineyards and they say, "Can we replace this at a fraction of the cost by sending out our team of geeks to scour the globe for offbeat opportunities?". They also send you information to let you know what kind of wine you're getting so you're better educated for the next time you want to repeat the event. With wine access dot com slash portal, you're going to get yourself one hell of a bottle, with wine access dot com slash portal. So why not order them bottles tonight. You get \$100 off and support the show by going to wineaccess.com/portal. You'll be glad you did.

(00:32:52)

Eric: You know one of the things, and by the way, I've had this issue with you—

Bret: Do I take it we are not in a podcast at the moment?

Eric: We are in a podcast. Oh, *believe* me, I'm going to put the hurt on you because you are backing out of your role in history, and I'm sick of it. Look, I love you like, like you were my own brother.

Bret: Amazing.

Eric: Okay. It is the case that you have always done this, and it means that you're not taking your place properly. And I had to go to the extraordinary length of tricking your advisor, Richard Alexander, one of the great evolutionary theorists of our times—

Bret: Absolutely.

Eric: Right? One of the absolute tops. Member the national Academy of Sciences, chaired professor at the University of Michigan. I had to trick him into writing a letter of recommendation for you so that we would have some record, as he was getting on in years, of who you actually were, because I knew that Evergreen was not going to be—it's not part of the game.

(00:33:59)

Bret: It's true.

Eric: Okay. Here's what he had to say about you. "Bret Weinstein may well be the brightest graduate student I have ever known. His thesis defense involved only one of his four thesis chapters, and it alone was far more than sufficient as a thesis. I don't know anyone who knows more than Bret about not only a wide variety of topics in biological evolution, but the problems and possibilities of cultural change and the means of bringing people together and solving difficult problems. For 40 years, I held frequent, sometimes almost daily seminars with my doctoral students in evolutionary biology. While he was a student, Bret was a major element in all of those seminars. When he spoke, there was almost always respectful silence, even when he was junior to most of the people involved. Bret's thesis topics are so significant and timely, and so well treated on the lifetime patterns of humans and other species, the function and importance of telomeres and explaining lifetimes as hedges against cancer and several other important topics such as species diversity and sexual selection, that he dramatically converted, on the spot, two reluctant—" And by the way, reluctant is British understatement here—"I will say mildly and skeptically evolutionist members of the committee. I think that, despite his youthfulness, in terms of the characteristics I listed earlier, Bret is the best candidate."

You were the number one student of Richard Alexander, who ended up at the Evergreen State College, which was a *giant* mistake. And it was always a mistake. You should never have been there. I was completely right. I'm sorry to be overbearing about it, but, like, how many years did I tell you, "You gotta get out of that place."

Bret: Well, look, first of all, Dick was very clear with me about the fact that, we're he trying to compete in the modern academy, he did not believe he would have succeeded. And he was clear about the fact that there was no good solution to the problem. So, you know. I can't say that I've ever heard that letter. I believe you have quoted parts of it to me.

(00:36:06)

Eric: Yeah, because you're going to do this thing where you downplay your gift, and I'm sick of it. I'm tired of it. I've just, I've had it. And part of it, what happened is that you are now distorting the history of science. You have a place in the history of science that you are not taking up, you are not advocating for, there's something that you don't like about this.

Bret: No, no, I don't think this is true. I just think I'm pursuing it—maybe I'm pursuing it in a way that it doesn't work out in the end, or maybe I'm pursuing it in a way that it would, maybe there's more than one path.

Eric: I've been through too much helping you, trying to make this happen, where people become aware of the complex of ideas that you've been pushing out, and my feeling about this is that you maintain this very beautiful, very calm position, and it's *enough* already. Like, you have a story and that story is an explosive story. I mean, I'm happy to bury this podcast so that nobody ever hears it, but I want to actually explore the truth, rather than this extremely good for you, high fiber, you know, low sugar, bowl of granola.

Bret: I just don't think that's where we are. I've been very clear and very public about the fact that I think my entire field is spinning its wheels, that they've gotten caught by a few bad assumptions and that they are spending decades in the weeds for no good reason, that there is a way out, that I didn't know what it was for a long time. I did figure out what it was, and getting their attention on the question of what they're doing wrong is a Herculean task. I've made that clear. The question is what is the best use of the opportunity that I've got, the cards that I hold, and we have a difference of opinion about what that might be. And you may be right. I'm not saying you're not right, but I am saying that there's at least a discussion to be had about what the best way to play the—

(00:38:01)

Eric: Why don't we have that on your podcast. I accept your invitation to come on. This is my podcast. We're going to do it my way.

Bret: Let's do it your way.

Eric: Alright. I'm the older brother.

Bret: I've noticed I have the ultimate Marcia Marcia market problem.

Eric: All right. Bret, this is not the story of your career and your life. What happened is that you got stuck at the university of Michigan for a very long period of time, because you made people very uncomfortable. What he's saying in that letter of recommendation is that you wrote four different theses, so far as I can remember, and they were on widely different topics. Furthermore, here's an interesting one: no one that I know of, despite the amount of discussion that's been spilled in ink over Evergreen has put you together with the hero of a book called *The Tapir's Morning Bath*, that appeared years earlier.

Bret: It's odd that it never shows up.

Eric: Right? It never shows up. And then you're also the recipient of the Golden Gazelle award, I think of the national organization of women, for standing up to ZBT at the University of Pennsylvania. And you got ejected, effectively, from an Ivy League school due to threats of physical violence for standing up for black women being exploited by white men. I mean, like, then you're like the, the field assistant and main student as an undergraduate of another legendary evolutionary theorist, Bob Trivers. And somehow, you know, Richard Dawkins is treating you as a guy who isn't really his equal. "You're not really a major theorist. You're very confused and you need to learn more about the extended phenotype" and all this kind of nonsense. And you're so polite that you're not even just, I dunno, I think you're out to lunch. No offense.

Bret: I get it. I get it. And you know, like I said, you may be right.

Eric: Okay. I want to talk about the subjects that you're most associated with starting with your thesis. And I want to get into the science of it using podcast. If people get left behind, they get left behind.

Bret: Okay.

Eric: Okay. Now Dick Alexander is a legend in evolutionary theory because it's very hard to use evolutionary theory to make predictions that can be verified in the world. It's sort of this loose amorphous collection of techniques and viewpoints. And people sometimes think it's not even a theory because it doesn't seem to be predictive.

(00:40:37)

Eric:

And then there are a few predictions. So, am I right? Darwin started this game off by predicting that there would be a moth with a really long tongue because there was a flower that had a really long distance to go before you could get the nectar out of it.

Bret: Yeah, he had been sent an orchid by Bateson, maybe, with a foot long Corolla tube. And he reasoned very straightforwardly that it would make no sense for this plant to have invested in this very long structure if there were not a tongue that could reach down to gather the nectar. And I believe he did not live to see the discovery of that animal.

Eric: I didn't know that.

Bret: But he was absolutely correct. There is a moth that has this beautifully long tongue. It's a Sphingid Hawkmoth one of these sort of hummingbird-esque moths, and anyway, yeah, it's one of the major predictions, demonstrations, that evolutionary theory actually can be used predict phenomena that you haven't been able to observe.

(00:41:34)

Eric: Okay. And you know, Darwin famously couldn't, for example, like, I don't know how much I've talked about this in the open, but my favorite Darwin book is the one he wrote after *Origin of Species*, which is on the various contrivances by which British and foreign orchids are fertilized by insects. It makes absolutely no sense as a title, I think that's what's so funny about it. But because orchids are so highly speciated, it turned out to be the perfect place to explore the consequences of evolution. And he couldn't figure out my favorite, I don't know whether it's clade or a group—

Bret: Clade is pretty safe.

Eric: Yeah, clade of orchids, the Ophrys system, which is just unbelievable because it mimics the pollinators, the female of the pollinator species using pheromones and some sort of replica good enough to fool males into copulating with the lower pedal of an orchid—

Bret: A 3D replica of the female that smells like her. And it just so happens that when the male lands on it to copulate, he gets these pollen packets glued to him, and then he screws up and makes the same mistake at another flower and delivers—

Eric: Well, he may, he may or may not

Bret: Put it this way—

Eric: Only the ones that screw up twice get to fertilize.

Bret: The reason that it gets glued to him is that it has worked enough times for this strategy to have been so beautifully refined.

Eric: Right. So Darwin saw that there was this mimicry going on, but he couldn't put it together. He spent pages and pages not getting it. So I think it's very funny. So he predicts some things, but he can't predict something else in a very closely related system. Okay. Fast forward, Dick Alexander comes out with a crazy prediction, which I still don't fully— I mean, it's just amazing that he made it— where he says, I bet that you will find the kind of behavior we associate with wasps and bees, which is in this class called hymenopteran ants of eusocial breeding patterns and organization, but in mammals that will live underground.

Bret: So, I *think*, the way this story actually worked, he didn't say you will find it—

Eric: Or, you *could* find it.

Bret: What he said is, in principle, there's no reason that a eusocial animal *has* to be an insect. That in fact, you could get such a thing in a mammal. And then he predicted—I forget how many characteristics there were—but he named some large—

Eric: So we should say that there's something funny about the system of ants, bees, wasps, which is that they've got this very strange haplodiploid chromosomal characteristic. Do you want to say a word about that? Cause that makes the prediction more—

Bret: Sure. So it has long been understood that the hymenoptera behave in this incredibly cooperative fashion, which effectively all of the workers of the colony forgo reproduction in order to advance the reproductive interests of the queen. And it was late discovered that actually their genetic system is unlike our genetic system, and that males have basically half a full complement of genes. They have enough genes to function, but they have half the female complement of genes. And, for reasons that are mathematically slightly complicated and require a chalkboard, the females are more closely related to the daughters produced by their mother than they would be to their own offspring, their three quarters relatives to her offspring. And there they would be 50% relatives to their own offspring.

Eric: Spot on.

Bret: So, they are actually evolutionarily favored by very standard mechanisms. Once you understand the crazy genetics underlying the thing, they are favored to engage in behavior where they forgot reproducing and fostered.

Eric: So, once you understand the chromosomal difference of the system, it is far less surprising that it would behave as a loosely coupled, in some way—don't overreact—unified organism, which is distributed. That there are ways in which the hive behaves as a superorganism, and there are ways in which it does not.

(00:45:40)

Bret: Yeah. Well, all I want to say is, I'm not sure how clear we have the story with respect to what precedes what— it's completely plausible that the behavior precedes the evolution of the genetic system.

Eric: Right.

Bret: And I actually, frankly just don't know where that research stands at the moment. We have found many other insect systems that have various versions of this. Interestingly, though, the termites are not hymenopterans.

Eric: Right.

Bret: And the termites engage in this behavior—

Eric: Termites are eusocial, but they're not haplodiploid.

Bret: They're eusocial, they behave very much like ants.

Eric: Okay.

Bret: But they don't have the strange genetic system, proving that the behavior can evolve even in the absence of this genetic system—

Eric: Well, the reason I bring this up is that if you look at, for example, Prince Peter Kropotkin, the great anarchists theorist, he was obsessed by finding analogs in nature of preferred human structures. And so it's very simple to say, why can't we work together the way an ant colony all works together? And then there's a counter to that, which is, well, they have different chromosomal structures, and then you say, well, but yes, but that's a kind of a cheap way of achieving eusociality. There are other ways of—so through this crazy kind of investigation, we get to Dick Alexander, who, and I think you're quite correct, says there is nothing prohibiting us from finding a mammalian species that exhibits ant- and wasp-like behavior. And it would be likely to have these characteristics, it would live underground, in a—

Bret: Yeah, underground, I believe eating tubers, was on the thing. It was a crazy list. And you know, my understanding from, from Dick—Dick is now unfortunately dead. He died a couple of years ago. But my understanding from him was that he didn't actually expect to find such an animal. He was speaking very abstractly, just completely theoretically. And at the point that he unleashed this idea, it may even have been in a talk, rather than a paper. The information made it back to him, actually—what about naked mole rats? They match your characteristics, and study reveals then that actually they are eusocial, they behave very much like ants, bees, wasps, termites, et cetera.

Eric: And this is like one of the great moments in modern science.

Bret: I really think it is. It's certainly the moment that people who know who Dick Alexander was, reference as sort of the high watermark because it's comprehensible. You know, Dick did a lot of things. He was very interested in people and other things, but this particular demonstration was so, it would be impossible to have predicted such a thing and have gotten lucky. He had to have understood some things that were extremely deep in order for that to have worked out. And so, yeah, it's really, I don't know of another example in evolutionary theory of a prediction that clean, of something that obscure.

Eric: I know one.

Bret: Oh yeah?

Eric: Yeah. I once heard a story about a graduate student who predicted that the breeding protocols of laboratory rodents would compromise the laboratory system in terms of its relationship to so called “wild type” versions of the same species. So you have the bred rodents and you have the wild rodents, and that they would be distinguished by virtue of the fact that the non-coding nucleotide sequence at the end of the chromosome, known as “telomeres”, would be wildly different in length if the prediction were true from pure evolutionary theory.

Bret: Wow.

Eric: Yeah.

Bret: Yeah. Yeah, that story that didn't happen *exactly* the way you said it, but you know, it's been a lot of years, and it takes a second to get back there.

Eric: Yeah. I mean it's, you, you did that.

Bret: Yeah, I did that.

Eric: And that story, unfortunately, has not really been told, and it is, in some sense, your central origin story as a biologist.

Bret: It's a pretty good one, and it definitely changed the way I saw myself in a way that has been very productive.

Eric: Okay. I want you to talk to me about that story, and because I lived with you, I know that it happened, and I know that it got buried, and I know that it's part of what I'm calling the Distributed Idea Suppression Complex because, quite frankly, you were not the only person who was a part of the story, and the story had to die because it said something, which is that the power of your theory was sufficient to predict, from first principles, a manifestly observed and surprising result, within molecular biology, from pure evolutionary principles.

(00:50:47)

Bret: Yup. All right. I'll try to do a short version of it.

Eric: You know, this is long form podcasting, and you tell—however long the story is, I guarantee you when people finally figure out that it may be that the rodents that we've used to test drugs on, let's say, might be compromised, and compromised in a way that would be potentially extra permitting of potential toxins in the form of pharmaceuticals. I think that it's going to be fascinating. And it's going to repay the study that it will take to understand the story. The floor is yours.

Bret: All right, so let me just set the stage a little bit. Evolutionary biology has—

Eric: But, do me a favor.

Bret: Yeah.

Eric: You can get into a very patient careful pedagogical mode. This is an exciting story. Tell it the way it actually occurred.

Bret: I'm going to tell it the way it actually occurred. And I'm going to be careful. I'm going to try not to be—there are parts of it that were for a very long time kind of emotionally fraught. But anyway, I think I remember it well enough to do a sparse but complete version.

Eric: Okay.

Bret: Evolutionary biology has long been biased in the direction of abstraction. Rather than thinking about mechanism, that is to say we deal in the phenomenology of things. We talk about gross patterns that we see in nature rather than talking about the fine detail of what drives them. That has been changing in recent decades, but it has a long history, and it comes from a very mundane place. That mundane place is that we just haven't had the tools to look, for example, inside of cells and we haven't been able to read genomes. You know, we could have been able to read a gene here and there at great expense, but the ability to peer into genomes is pretty new. The ability to peer into these molecular pathways is pretty new. So anyway, there's a historical bias in evolutionary biology against mechanism and in the direction of phenomenology. I have never been particularly fond of that bias. I have always been interested in mechanism. I'm interested in the phenomenology too, but I've always kept my foot in the door with respect to mechanism. And as an undergraduate, I took lots of mechanism classes. I took a development class at the time, developmental biology was in my opinion, a bit stuck. It is now unstuck in a very dramatic way. But anyway, I took a developmental biology class. I took some or immunobiology. And anyway, I was armed with these things in an environment in evolutionary biology where most people were not, most people were in the phenomenology. And one day I happened to be in a seminar. Dick Alexander was running a seminar for graduate students, and a student was there who was very out of place. He was studying cancer, and he, on a lark, decided to take an evolution seminar that looked good to him in the catalog, and it wasn't right for him. And he gave a talk at some point, and his talk was on his work with cancer and frankly, because all the other people in the room were evolutionarily oriented, nobody was really tracking what he was saying. But what he said struck me like a bolt of lightning. He said that in the realm of cancer research, people were looking at telomeres, which are these repetitive sequences at the ends of chromosomes. And they were toying with the possibility that the fact that these telomeres shorten every time a cell divides, that that is providing a resistance to tumor formation. Very straightforward—counter counts down and that would prevent.

Eric: So just for the audience that maybe needs a tiny refresher, we're taught in general that DNA is a string of letters called nucleotides, A, C, T and G, and that, in general, three of those that are adjacent to each other form words called codons. And for every word there is an amino acid or an instruction to stop coding for amino acids. So this is the instruction tape that tells us how to string together amino acids into proteins to make machines, molecular machines. This is some weird different thing, where the region of DNA could be interpreted as coding for a protein, but in fact might be instead just counting how many nucleotides are at the end. So it comes across as a counter.

(00:55:33)

Bret: It's a little better. It was known not to be a coding sequence. It wasn't a useful sequence. So what you had is a bunch of DNA at the ends of chromosomes that were just repetitive, and the length of the number of repeats varies. And the number of repeats correlates with basically how many times the cell can divide before it refuses. This being interpreted as a cancer prevention thing made perfect sense. But the reason it struck me like a bolt of lightning was that I was well aware of the existence of tumors and their implication in something entirely different. What they had been implicated in, as far as I was aware, was something called Hayflick limits, which were the tendency of perfectly healthy, happy cells to grow and grow and grow and grow in a Petri dish, until they hit some number of divisions and then to stop without apparent dysfunction. So—

Eric: So this was the theory of Leonard Hayflick?

Bret: Yup. It was the discovery of Leonard Hayflick, who basically overturned the prior wisdom about cells, which was that they would grow indefinitely as long as you kept feeding them and making an environment that was conducive to division. So I don't exactly know why that result had been misunderstood at first. Maybe somebody had a cancerous cell line and so they got the wrong idea and it just spread, but Hayflick checked it and it turned out to be false. It turned out there was a number of cell divisions that healthy cells would go through, and then they'd stop. The mechanism was not obvious to Hayflick, but later it became clearer and clearer that the mechanism was these sequences at the ends of chromosomes which shorten each time the cell divides. And the implication was that, potentially, this was a cause of what we call "senescence". What in common parlance would often be called "aging", the tendency to grow feeble and inefficient with age. If your cells are each in a cell line and that line has a fixed number of times that it can replace itself before it has to stop, then some point your repair program starts to fail. And that repair program, failing across the body, looks like what you would expect aging—aging follows the pattern you would expect if cell lines one by one stopped being able to replace themselves. So—

Eric: We know that there's a special sort of a, I don't want to call it cell line cause you keep correcting me for every tiny mistake I make in speech. But, if we divide our, our body into two kinds of cells, soma and germ, where germ lines are that which has a hope of immortality

through reproduction, then it's the somatic cells that have finite limits on their ability to undergo mitosis and cellular repair and whatnot.

(00:58:25)

Bret: And the germline can't because if it did, your lineage would go extinct as a result of small—

Eric: Small addendums.

Bret: So it's the soma, the parts of your body that don't go on to produce babies, that have this effect. The reason it struck me like a bolt of lightning was that I was aware of another very elegant piece of research done by a guy named George Williams. George Williams had finally—

Eric: One of the greatest of modern—

Bret: One of the greatest modern evolutionary biologists. I actually knew him a bit too. He is also now gone, unfortunately. But George Williams had laid out in a beautifully elegant paper, *the* evolutionary theory of senescence. It is an absolutely elegant argument that says that, in a lifetime there are, well, let's start somewhere else. A creature is built of parts and traits. It has a relatively small genome and a relatively high complexity. At the time it was thought there might be 100,000 genes or something and you have maybe 30 trillion cells with a ton of complexity. In order to get that small number of genes to dictate how to produce a creature that complex, the genes are doing multiple things.

William's point was when a gene has multiple effects, what we call a pleiotropy, those effects may be good or bad. If effects are good early in life—

Eric: By good we mean contributing to fitness—

Bret: Fitness enhancing traits at some costs late in life, then they will tend to be accumulated by selection. And the reason for that is because, well, there are two ways to think of it, really. If a negative trait occurs very late in life, then a large number of individuals who have the gene for that trait will not live long enough to experience the harm. So if it came bound to a positive thing early in life and you're dead before the late life harm accrues, you got away with it. Right? So William's point was, he was building on earlier work of MetaWare, but let's skip that for the moment.

His point was, because of tradeoffs, you will have lots of traits that are good early and bad late. Selection sees the early traits much more clearly than it sees the late traits, and it prioritizes them because of the discounting that arises because so many individuals aren't around to experience the late-life harm, and if they are around experienced the late-life harm, a lot of their reproduction is behind them anyway. So they count less. Selection counts more early in life. And

this timer starts at the moment of first reproduction, the usual moment of first reproduction for your species. So this was a beautiful hypothesis, and it was beautifully articulated with *many* predictions, which is the way really good work is done. And we knew, at the point that I was entering graduate school, we knew that the hypothesis was right. It was a theory.

(01:01:36)

Bret: And the reason that we knew it was real,

Eric: The hypothesis is the Antagonistic Pleiotropy Hypothesis.

Bret: The Antagonistic Pleiotropy Hypothesis for senescence. We knew that it was right because it predicted so many phenomenon in nature that we could readily go out and measure. And this is again where the phenomenology versus mechanism comes out.

Eric: Okay.

Bret: We know that creatures that are poisonous or have a shell that protects them or can fly away from danger, are disproportionately long-lived for their size. Small creatures tend to live shorter lives than large creatures. But if you can fly, then you're off the line of the other creatures of your size. So for example, their small bats who have been recovered after 30 years in the wild. So creatures that have special protections have disproportionate longevity. This matches William's hypothesis, because it is their ability to fly away from danger that makes the likelihood of their experiencing late-life costs go up.

Eric: Yep.

Bret: So selection sees their late life more easily than it sees a small Creek.

Eric: I just want to say something. This is a podcast. It's an unusual podcast and we can talk science and I'm thrilled, but we always have our colleagues in our minds when we're talking to a general audience and the colleagues are always in a "gotcha" mode. Well, you forgot about this. You didn't mention that. I'm even interjecting little bits because I want to make sure that you're immunized from all the bullshit that the academics, so I just want to make a general statement, which is we can come back and get into any level of specificity that somebody wants to, if they want to take you down, I don't care. What I'd love to do is to tell the story with enough punch that people understand what happens.

Bret: So we're about to jump into the meat of the matter. The theory of antagonistic pleiotropy was well established, but in four decades of research on the genome, nobody had found a gene that matched it, so that we knew that this explanation was right, but we couldn't find the genes that caused it. The mechanism was missing. So, anyway—

Eric: Does that mean, to be a gene, it has to be protein encoding?

Bret: Yeah. Anyway, I knew this assertively, I was well familiar with William's paper. At the point that I saw this talk on cancer and I knew already about the question of senescence, everything came together. This was obviously the answer, where the missing pleiotropy was. Well, the missing pleiotropy had to do with a telomere, which wasn't exactly a gene. It was genetic, it was DNA, but it wasn't a *gene*, but it was perfectly capable of producing exactly the effects that we see in senescence across the body, tissue—

Eric: So a counter, and not a protein, could be the answer.

Bret: Right. Now, I saw this *instantly* at the point I heard this talk, I raised my hand, and I tried to articulate what was so obvious in that moment, and I couldn't compel a single person in the room. They couldn't even understand what I was trying to say—

Eric: Which is bizarre.

Bret: It was bizarre. I mean Dick was in the room and you know, Dick was *very* broad-minded and I just couldn't make it clear.

Eric: Look, let me just interject something, and you can correct me if I'm wrong, but my impression of it is that it was a very simple idea attended to by an outrageous amount of irrelevant complexity that had to be very carefully pried off of the central idea.

(01:05:04)

Bret: Yeah, I think, I think that's well said. So anyway, I left the room feeling like I had just glimpsed something so important, kind of, you know, I wondered could it be right and I started to just do the first bit of library research to figure out whether somebody else knew what I knew or—

Eric: So I'm not even sure that you fully said it. I want to make sure that I'm even clear on it and I'm going to, I think I'm right, but correct me if I'm wrong. What you're saying is, "What if the Hayflick limit is a protection against dying from immortality at a cytological level", that some cell gets a dream of immortality that it shouldn't have because, let's say, it's a somatic cell, and it says, "Okay, I just want to keep dividing and dividing and dividing". Nature knows how to do this, and that immortality, which sounds good at first, is actually called cancer. And so in computer science we would say, okay, you've introduced a recursion limit into a while loop or a for loop to make sure that you don't have a resource leak, which is what a tumor is.

Bret: Yeah, so let me say it this way. If you have a damage to a tissue cut on your arm or something, the cells on both sides of that cut suddenly become aware that there is a problem, a gap, because they can't hear a neighbor on one side of them and their natural reaction is to start

growing into the gap until they can hear a neighbor which is the sign to stop. If you imagine that something like that is occurring in every tissue, or almost every tissue, the problem is that that means that every tissue in your body for which that story is about right, is in danger of having damage from radiation or whatever, turn it deaf to its neighbors. A single cell that has turned deaf to its neighbors will suddenly start replicating, and if it is deaf to its neighbors, then there's no message that it's going to hear that's going to tell it to stop. So that thing, imagine any cell in your body just taking off and growing and growing and growing—

Eric: Okay, this is terrifying. What you're saying to me is, is that if I'm comprised of let's say 30 trillion cells and I view them as each let's say subroutines, any subroutine that is not denucleated, right? Like this wouldn't happen in the in the lens of your eye because the nucleus has been removed, but any other reasonable cell is potentially your assassin, because it's mitosis process might completely go rogue.

Bret: It can run away.

Eric: Okay.

Bret: And so the rather elegant and very simple idea is that there would be a hard limit so that any cell that had become damaged, so it started down this path would just simply run into the number of cell divisions it was allowed in a lifetime and it would stop.

Eric: So like, the moles on my face that some of my less couth commenters loved to talk about—

Bret: Yep.

(01:08:01)

Eric: Are effectively attempts to kill me that may have stopped. And that the perimeter where they stop is where the Hayflick limit took over and said, "This cell line must die so that the patient will live"?

Bret: Yeah. The name I gave him was "prototumor" and the idea is a prototumor is a patch of cells arrested at their Hayflick limit. Because they had become unregulated. If you go to the dermatologist and you say, what do I look for? You know, they tell you certain things to look for. So a round patch of cells that suddenly becomes irregular in shape. Well that's what would happen if you took one of those cells and gave it a second mutation and it started growing again.

Eric: Got it.

Bret: Right. So anyway, the idea that a limit on cellular reproduction—

Eric: Yep.

Bret: Is adaptive to protect you from cancer—

Eric: K, so there's a little bit of a mind bender because what you're telling me is that I've got to avoid immortality, which can kill me, and that the solution to not dying is *death*.

Bret: Yes, *and* that what selection does is it balances these two competing forces to give you as much vigor and longevity as it can.

(01:09:00)

Eric: So all of the other diseases and insults and things that I can die from sort of start to fade away. And at the complete core of biology, in this theory, there are two things that I can't get away from, one of which is death by immortality, and the other one is death by recursion limit.

Bret: That's it.

Eric: It's a very elegant thing. And now the problem is, is that there's all this weird attended complexity that you had to deal with.

Bret: Right

Eric: So it was like stem cells versus germ versus ...

Bret: So when I went into the literature, what I found was that people had played around in the neighborhood, but that there was a particular fact which blocked every attempt to make sense of what was going on. And the fact was that rodents were understood to have ultra long, hypervariable telomeres. And I didn't know what that meant at first, but the more I looked into this possibility, the more I realized that dozens of longstanding problems would be solved if my hypothesis was true, but that my hypothesis couldn't be true because basically mice have long telomeres in short lives. Why is that? And I banged my head on the table for a couple of weeks trying to figure out what was going on.

Eric: Figuratively

Bret: Yes, maybe even literally on occasion. But the question was, I began to wonder if there was something wrong with the idea that mice had long telomeres. Sometimes, like in Hayflick's case it turned out that a bunch of people were copying some wrong result, so it seemed like a lot of people had seen it, but only one had. And I checked, was it true, that there was some, that everybody was parroting one study that said mice had long telomeres?

Eric: Right.

Bret: It turns out lots of people had tested it. Mice have long telomeres like 10 times the length of human telomeres. It just didn't fit. So finally, it occurred to me that it was possible that what was going on—I discovered something in trying to figure out what they meant by “mice”. Right? There's a lot of species of mice, but all the mice that we use in the lab, with rare exception, are from one genus, and often from a particular target species.

Eric: So you were focused, if I recall correctly, on *mus spretus*

Bret: *Mus musculus*, which is the common one. What shocked me was that it turned out all the *mus musculus* that were being used in labs across the country, and in many cases, farther afield than that were coming from one place, which I had no idea. There was one—

Eric: I remember getting a phone call when you said, what do you know about The Jax's lab?

Bret: The Jax lab in Bar Harbor Maine, right? They seemed to be the source of everybody's mice. And so it began to be—it was a possibility I could not shut down in my mind, that there was something about what was going on at the Jax lab that had resulted in the mice that were being sent out to all these other labs—

Eric: Is it that they were representative animals—

(01:12:04)

Bret: Right, these are a model organism. People were just using mice because mice were a convenient mammal, but they're all coming from one place, and it began to occur to me that that one place was not just a source of mice in the sense that we might think it, it was actually a selective environment that was impacting those mice. And when I dug deeper, it turned out that the mice had all, they were descendants of a long lineage that had lived in captivity under conditions at the Jax lab. And at some point I realized that the most likely thing going on was that there was something about this environment that had wildly elongated the telomeres of these mice. And that was simultaneously an unbelievable idea, but the only one I could think of that made sense of everything I had seen. And so—

Eric: Well, it's unbelievable because the consequences, I mean, look, I have not even heard whether anyone has said, “Yeah, we did that, we screwed that up.” But it is, like, your favorite model organism for mammalian trials being screwed up by a central facility. Because also there's this weird thing where medical people very often stop taking into account evolutionary theory because they treat that as “Well, that's that class I took in college or the beginning of graduate school.”

Bret: Right. So I began to focus on this question and I did something that was the right thing to do, but I did it in a way I will forever regret. I found somebody who was represented in the literature, who I regarded as very well versed. They made sense to me, their papers. Her name was Carol Greider. Carol Greider is now a Nobel Laureate. She was not at the time. She was the co-discoverer of the enzyme telomerase, which is the enzyme that elongates telomeres, when that occurs—

(01:14:01)

Eric: With the famous and co-Nobel recipient—she was the student of Elizabeth Blackburn.

Bret: Elizabeth Blackburn. Exactly. She was her student and they shared the Nobel prize with Szostak. In any case, her work seemed good to me. I called her up, cold, you know, I went into the insect division office and I sat down at the phone. I called her, I said, Carol, you don't know me. I'm a graduate student at Michigan. I'm an evolutionary biologist. I'm racking my brains trying to understand something. Can you tell me, is it possible that mice don't have ultra long telomeres? That it's only laboratory mice that do? And she said, huh, that's really interesting. I'm pretty sure that mice have long telomeres universally. But it is odd that if you order *mus spretus* instead of *mus musculus* and you order from European suppliers, the lengths are very different than what you get if you order *mus musculus* from the Jax club. I said, Whoa.

And she said, yeah, that's really interesting. And then she said, I can't remember if it was the same phone call or if we had a second phone call, but she said she was gonna put her student, her graduate student, Mike Hemann, who I think is now at MIT, on the project. And he was going to do a little work to figure out whether there was anything to this. And Mike did some work. They sourced some different strains of mice that were, they were actually not wild mice. Wild mice would have been the right test, but she couldn't get wild mice for obvious reasons.

Eric: You'd have to go out into the woods.

Bret: Right, exactly. And so she got several different strains of mice that had just been in captivity much less time. She actually got one strain of mice that was treated very differently in captivity. But nevermind. She put her graduate student on it, and he measured their telomere lengths. And I get this excited email. Mike Hemann sends me any email that says effectively, "Whoa! The hypothesis is true, mice have short telomeres!" Right? Now—

Eric: I'm sorry, this is like as close to a who'd done it Discovery J'accuse— the mice, you know, I remember, you were over the moon.

Bret: I still am! I still can look at this email and it is the moment at which I realized, A, there's no way I'm kidding myself about how well I understand this.

Eric: Right.

Bret: Right? That prediction was—

Eric: How old are you?

Bret: Now? Or then?

Eric: No, when you get this email.

Bret: When I got that email it was 1999? 98? Something like that.

Eric: Okay. So over 20 years ago.

Bret: Yeah. So I get this email, and—

Eric: By the way, that puts you at about 30. You're at the beginning of your career, and you—in this story, you've just predicted that—

Bret: It's a stunning coup for a graduate student. And, it wasn't in my advisor's wheelhouse, so it was clearly my own work. And, I mean, Dick was great about not blurring those things, but—

Eric: Okay, either you are a dirty dog liar—

Bret: Right

(01:17:10)

Eric: And I was there at the time—

Bret: Yeah.

Eric: Or, so we're both dirty dog liars about this particular story—

Bret: Or—

Eric: Or, one of the great moments in evolutionary theory, which is—and let me just curate this, because I'm not a biologist, but I think I can more or less get this—because it's a breeding protocol that is the alteration in the evolutionary landscape for these laboratory mice, and because it's acting on a non-protein coding region, the adaptation to a change in the breeding protocol can be *extremely* rapid. It doesn't have to undergo some sort of completely crazy typical Darwinian story about random mutation and some of them being retained and others being rejected.

Bret: It's even better than that. The creatures are presumably—so we haven't gotten to what the breeding protocol has to do with this—but the creatures are built in some sense to detect how dangerous their environment is, and to the extent that the level of extrinsic danger changes, their telomeres respond quickly so that they are better adapted to the environment. So, they're built to detect the environment and then what is actually a strict matter of market forces.

Eric: Okay, so there are no predators in this environment.

Bret: No predators in this environment.

Eric: And we're not killing them particularly early based on their skills. So environmental insult is sort of absent.

Bret: Environmental insult is more or less absent. What we are doing is imposing an economic rule on breeding so that we can maximize the rate at which we turn mouse chow into mice, which is obviously economically the right thing to do, if you're selling mice to all these labs, you want to produce as many mice as cheaply as possible. So producing as many mice as people—

Eric: The genius of the market!

Bret: It's the genius of the market.

(01:19:08)

Eric: There you go.

Bret: So in order to produce as many mice as cheaply as possible, what you do is you don't breed animals past eight months. They breed faster when they're younger because of senescence. And so you don't breed older mice. You throw them out and you replace them with younger mice who breed faster. What that effectively did was it eliminated the selection against cancer, and it turbocharged the selection in favor of youthful vigor

Eric: Well let me see if I get this—in general, almost all cancers, like, cancer of the germline happens early in life, but all the other cancer, in general, is much more common later in life.

Bret: I gotta pause. I realize I forgot to tell you one thing Carol told me in my first phone call with her that's vital.

Eric: Sure.

Bret: In addition to telling me that there was something funny about *mus spretus*, she told me that, consistent with the hypothesis that I was conveying to her, that all mice die of cancer. She said, "If you let them live long enough, and then you do the necropsy, you find cancer of one

kind or another”, and that was perfectly consistent because they had these wildly long telomeres and no cancer protection. That would be the prediction of the hypothesis—

Eric: That’s an extrapolation—it’s not really all mice. It’s all mice that we see in the lab, which happens to be the mice that are ordered.

Bret: Right. She was still speaking from the mindset of somebody who thought that the mice she was getting in the mail representative representative of mice in the wild.

Eric: Got it.

Bret: Okay, so let me clear up why the breeding protocol—and I should say, that it is the breeding protocol that is causing this? That part, I would say, is still a hypothesis. It has not been directly tested by anybody, but, what I would say is that many hypotheses were tested in the aftermath of the discovery, that lab mice have bizarrely long telomeres, and wild mice don’t, and no other hypothesis has stood up to scrutiny. So it is the last hypothesis standing and I’m all but certain that it will turn out to be true.

Eric: Yeah.

Bret: The reason that the breeding protocol has this weird effect, is that when you throw out the mice at eight months of age, you eliminate selection against cancer, you turbocharge selection in favor of—

Eric: Sorry, when you throw out the mice, for breeding purposes, at eight months of age.

Bret: Right,

Eric: Okay.

Bret: When you throw them out for breeding purposes at eight months of age, you are increasing the importance of their early life breeding, and you are discounting anything related to their ability to fend off cancer because they don’t live long enough in that period of time to get cancers that kill them. And so what has happened, according to this hypothesis, is that the mice that have longer telomeres have driven out the other animals from the colony. The trait of having long telomeres has swept through the colony and the telomeres have been elongated to an absurd degree, creating animals that *do* all die of cancer. And interestingly enough, another thing that’s evident from the literature is that if you look at their tissues, their tissues do not age in the way that a normal mammal’s tissues age, they remain young.

Eric: So there’s one aspect of aging, but that there’s a far darker interpretation of what you’ve just said. If I’m understanding you—correct me, I’ve never taken a class in biology, but I lived

this adventure with you—those tissues have, at a histological level, the level of how cells are organized, the possibility of radical histological repair.

Bret: Yes, radical effectively indefinite capacity to repair, which is going to come back in this story in the worst possible way. So—

Eric: This is like a—I mean, I just forget how great of a—

Bret: Me too, I go years sometimes without thinking deeply about it.

Eric: Without telling the story. Alright.

(01:23:06)

Bret: Yeah. Okay. So the story now gets kind of ugly. I recognize I've got all the pieces of the puzzle necessary to tell the story correctly. I have taken on a coauthor, we've found the literature necessary to do it in proper scientific form.

Eric: This came from you, but I want to mention your coauthor's name.

Bret: Yeah. Debbie Ciszek.

Eric: Okay.

Bret: And Debbie was an excellent coauthor, strong contributor to the paper. Anyway, we put together over the course of a year, I took a break from, effectively, my real dissertation work, and wrote a paper. Dick thought it was a fantastic paper. He was blown away by it—

Eric: Well I remember the revisions, and I remember this was like, I mean, if I think about what's on the line, like this combines one of these freak situations where you're using evolutionary theory to predict something, and in this case it's at the level of molecular biology, so with Darwin's orchid it's a tongue, and with Dick's thing, its behavior in naked mole rats. This thing is actually at a molecular level, *and*, it couldn't be more important if mice are going to be the major system in which we are going to test drugs, which are highly sensitive to what? Histological repair.

Bret: Yup. It's so profound on several different levels that I'm super energized about getting this into the world. It's transformative. Dick looks at the paper, he says, "This is fantastic". He puts me through the ringer to get it really tight. We get it tight. We send it to George Williams, the—

Eric: The number one guy in the world.

Bret: The number one senescence guy at the evolutionary level in the world, and he writes a beautiful recommendation letter for this piece. We're going to send it to Nature. George Williams tells Nature, you need to take this piece *very* seriously. We send it to Nature and they send it back with one of their absurd form letters that says that "The nature of the article is such that it's probably not—

Eric: Of limited interest—

Bret: To their readers. And we're, you know, I mean, we had a good laugh about that. You know, it's cancer, it's senescence—

(01:25:10)

Eric: Dude, it's *so bad*. Like, this is a response that indicates either malfeasance, or an Eliza program, or the janitor ended up responding who didn't know any bio—

Bret: It's the craziest thing, and you know, the cherry on top is that they're turning down George Williams recommendation? Like, how cra— do they know who he is? Like, what? Where?

Eric: On what planet?

Bret: On what planet do you turn down *his* recommendation to look at something about senescence? So, anyway, I get back this rejection, and I have purposefully not shown Carol Greider the paper in preparation, which I am afraid she might've read some way. The reason I didn't show it to her was because I wanted to preserve her independence as a reviewer for the paper. I was hoping, because I still thought she was an ally of mine, I was hoping that Nature would send it to her to review, and that she would look favorably on it, especially since it was, you know, very clear that she had done—

Eric: It was her lab that made the confirmation.

Bret: Yeah. And I, oh, another thing I forgot, I asked her at some point, something that now rings in my ears—I asked her, Carol, you've now got this result about, no, actually lab mice have long telomeres, but wild mice have *short* telomeres. That's a big result.

Eric: That's a hell of a Delta.

Bret: Where are you going to publish it so that I can cite it—

Eric: Right.

Bret: In my paper, which is the natural thing to do. And she says, “we're not going to publish it. We're going to keep the information “in house.” That was her phrase. I was too young to understand what the hell she was talking about.

Eric: I'll be honest, I'm 54 and I don't quite understand it myself.

Bret: Well, it's so heartbreaking. What she has effectively done is decided, “I could publish this result”

Eric: And then everyone would have it.

Bret: It would be huge, but then I'm on a level playing field with everybody else. If I don't publish this result—

(01:27:16)

Eric: I have a stream of papers I can get at.

Bret: Then I can start predicting other results. Nobody will know how I am doing that thing. I will look like a super genius. And so, holding it “in house” is a mechanism for a whole slew of papers.

Eric: to be, to be 100. You can afford to bend over backwards and not make inferences. Let's say the following, holding it in house is any seemingly inexplicable decision in science, but for the fact that it fits at least one story of this kind, which is that it is consistent with wishing to publish a stream, rather than the source of the information that would allow you—so you can either do one discovery or you can do a stream of predictions and that makes a certain amount of sense, given the ruthlessly competitive grant-winning environment. And we don't know exactly what happened, but there is no world that I know of in which you're allowed to hold back that kind of information, because, in part, of what's on the line.

Bret: Right. So—

Eric: I mean, this is not just a question of academic interest—

Bret: No.

Eric: Because these mice are used for medical testing.

Bret: Not even that. It's medical testing, but it's also all of the science relative, at least, to cancer, senescence, wound healing—all of the science that is stacked on these mice that is contingent on their function relative to their tiers is all compromised. You're letting year after year of this stuff accumulate. It's malpractice at an incredible level. So, I don't know that she has

turned on me, but I call her up, and I say, “Carol, we are stunned to find that our paper was turned away without review from Nature—”

Eric: *Without* review.

Bret: *Without* review. We need your help. Can I send you the paper and have you look at it? And she says yes. And I sent her the paper and she sends back the paper with an unbelievable number of intense criticisms that are not sensible. She pans the paper, does not believe it—

Eric: Do you still have that copy?

Bret: I have that paper, I have that paper with her handwriting. I believe I also have the FedEx envelope in which she sent it to me. But she hates the paper, and I have now forgotten a bit of the sequence. But as I am attempting to fix this up for another journal—oh, here's a, sorry, I hate to tangle this story, but it's important to get it right.

Eric: No but you haven't told this in enough—

Bret: I haven't told it in a very long time. After the rejection from nature, after Carol has seen the paper, and said it's cruddy, I get a letter I don't expect from a journal I don't—I know it exists, but I'm not super familiar with it, *Experimental Gerontology*. *Experimental Gerontology* says, “We are the editors of experimental gerontology. We have heard a rumor of your work. We're very interested. Would you be willing to submit a version to our journal?” and, oh, this is happening prior to Carol looking at my paper and panning it.

Eric: So the only way they would have known about this would have been from Nature or from Dick, or—

Bret: I'm pretty sure I know, based on what they, again, I was too young to sort out really what they were saying, but they indicate that they're fans of antagonistic pleiotropy, so what happened was George Williams, having heard that it got rejected, contacted some friends of his and was like, you should really take a look at this. So I begin the process of revising it. I've shown it to Carol, she's panned it. I send the revised version to *experimental gerontology*. They send it out for review. As you know, review is blind. You don't know who your reviewers are, but you can often tell who they are. It's not as obscure—

Eric: If it's a small field.

(01:32:00)

Bret: Yeah. So they read the acknowledgements of my paper, which are now on alert about Carol. I have to thank her in the paper for the work she did, but I'm now on alert that she's gone

strange on the subject matter of this paper, and so I've broken her out separately in the acknowledgements. I don't want to be as gracious to her, because she's being hostile to me.

Eric: Right.

Bret: But I don't want to not acknowledge her, so I acknowledge her separately. Experimental Gerontology then—I am 99% sure—sends the paper to her as the reviewer. She *pans* it. Absolutely *brutal* critiques, just pages and pages and pages of them. They are not high quality critiques. I could go through every single one.

Eric: Don't bother, this is a podcast, just—

Bret: No, I can't do it here, but I could have then—

Eric: No, okay?

Bret: But I didn't know what to do because she was in line for a Nobel Prize, that was well understood. I didn't want to accuse a leading light of the field of,

Eric: Okay, this is exactly why I got angry with the beginning of the podcast, you moron. No, no offense. You were in line for a Nobel Prize. You didn't. I mean, I'm sorry. There is an aspect of this about giving away your power, before you've even accumulated—you don't even have a PhD at this time.

Bret: I'm just saying, at the time, if you mentioned her name, people would say, "Oh yeah, her Nobel Prize is one of these years." Right? So my point was, I was in the awkward position—I didn't understand what I was supposed to do. I didn't want to send back a review that said, "I don't know who the person is who reviewed this, but they don't understand the material, and all of their critiques suck", because I didn't want to accuse somebody who was that powerful of not getting it.

Eric: I mean, here's the problem. What do you do? You don't actually have evidence in the hard form where like you have got videotape, but on the other hand, these are small worlds. This, all of this is preposterous.

Bret: Right. So I sit on the review for too long, not knowing what to—

Eric: Well you don't know how to play the game!

Bret: I don't know how to handle it.

Eric: I'm sorry, but, like, I had no advisor. Your advisor was not equipped for the modern era.

Bret: He wasn't equipped for the modern era. He wasn't equipped for molecular biology.

Eric: That's true.

Bret: I finally settle on a strategy that I can live with and I send back a note. I send back the review and my note says, "I don't know why, but this entire list of critiques is not high quality. If you would like to point me to any of the critiques in this list that you would like me to address, I am more than happy to do it, but I don't think it makes sense to address the entire list", and as I recall it, I hit send on the email, and within minutes, maybe it was an hour, I got back a response: "Your paper has been accepted for publication", which blew me away because I—

Eric: It makes no sense according to regular protocols.

Bret: Right. It makes no sense, because, clearly, they're supposed to send it out for review. The reviewer is supposed to say whether it's supposed to get published. The reviewer said it shouldn't be published. I said, "I refuse to address these critiques unless you ask me to." The editors have overridden the reviewer. *They* understood the reviews were cruddy. They needed me to say that in order to justify the move that they wanted to make. They knew the paper was good and the review was crap. So they effectively overrode normal peer review. Was my paper peer reviewed? Well, it was by the editors who were experts.

(01:35:28)

Eric: Let me jump in. Peer review is a cancer from outer space. It came from the biomedical community, it invaded science. The old system, because—I have to say this because many people who are now professional scientists have an idea that peer review has always been in our literature and it absolutely motherfucking has not.

Bret: Right.

Eric: Okay? It used to be that the editor of a journal took responsibility for the quality of the journal, which is why we had things like Nature crop up in the first place, because they had courageous, knowledgeable, forward thinking editors. And so I just want to be very clear, because there's a mind virus out there that says "peer review is the sine qua non of scientific excellence, yada, yada, yada, bullshit, bullshit, bullshit". And if you don't believe me, go back and learn that this is a recent invasive problem in the sciences.

Bret: Recent invasive problem that has no justification for existing in light of the fact that—

Eric: Well, no, not only does it have no justification for existing. When Watson and Crick did the double helix, and this is the cleanest example we have, the paper was agreed should not be sent out for review because anyone who is competent would understand immediately what its implications were. There are reasons that great work *cannot* be peer reviewed. Furthermore,

you have entire fields that are existing now with electronic archives that are not peer reviewed. Peer review is not peer review. It *sounds* like peer review. It is peer *injunction*. It is the ability for your peers to keep the world from learning about your work.

Bret: Keep the world from learning about your work—

Eric: Because peer review is what happens— *real* peer review is what happens after you've passed the bullshit thing called peer review.

(01:37:18)

Bret: Yes. Okay, so the paper was accepted by Experimental Gerontology. They went on to publish it.

Eric: This is called “Life’s Slow Fuse”?

Bret: No, “Life’s Slow Fuse” was the title as sent to Nature, and I changed the title because I did not want to compromise the story—I didn’t want to confuse the story.

Eric: The original submission was called “Life’s Slow Fuse”.

Bret: Right.

Eric: We probably have a copy of that somewhere?

Bret: Oh, of course.

Eric: All right. Then the Experimental Gerontology paper, what is it called?

Bret: The Reserve Capacity Hypothesis, which is a much less catchy title, but, nonetheless, the paper, I’m very proud of how it’s written. People read it who were not expert, could understand it. The abstract is *extremely* clear, and it ends with the clear point that, because we have unearthed, we have predicted, and Carol Greider has shown, that wild mice telomeres are short, and the telomeres had been elongated by captivity, that there is a clear danger that the mice we are using for drug safety testing are biased in an egregious way. And the bias would look like this: a mouse that has very long telomeres has an indefinitely large capacity to replace damaged tissue, *and*, it has a vulnerability to cancer that is preternaturally high. So, we may be overrating—if we use these mice, we may be overrating the danger of causing cancer, and *vastly* underrating the danger of toxicity. And, in fact, one of the things—so, the point was you give a mouse who’s got an effectively infinite capacity to replace its tissues, a toxin, and either the toxin is so deadly that it dies right away, but if it doesn’t die right away, it just eats up the insult. So those animals would lead us to release drugs—

Eric: By insult, what you mean is cellular necrosis?

Bret: Damage. Yeah. What this would cause us to do is release drugs onto the market for human use that are highly toxic across the body.

Eric: Wait a second—if the mouse standard was the *last* standard—

Bret: Well, no, even if it's not the last standard, because—

Eric: Well, it's important to say this—

Bret: The problem is, I mean, you can imagine how hard it is to test on large, slowly reproducing animals.

Eric: Well, and the ethics of testing on humans is very—

Bret: Absolutely.

Eric: —restricted, so mice is the last *cheap* place—

Bret: It's the last cheap place—

Eric: —to get large N data.

Bret: Not only large N, but it's the one place that you can make the following move. You can imagine that in many circumstances the accelerated lifespan, the accelerated life cycle of mice allows you to see long term damage as it would accrue in humans on a very short timescale. That doesn't work with monkeys. It doesn't work with human patients. It works with mice, maybe, but in the case of mice with ultra long telomeres, those insults will be invisible.

(01:40:36)

Eric: Let's just, I want to back up because I think this is a really important part of the story. What you're saying is if you take an organism that has an expected, let's say, 40 year lifetime, it's very expensive timewise to say, "We ran this experiment and found that there was no immediate damage that was visible, but towards the very end of their lives we saw a marked increase in morbidity" or—

Bret: Yeah, I mean if you took a drug and it knocked 15 years off your life on average, that might not show up in any notable way in a short term study.

Eric: If there was pressure to—

Bret: Right. And nobody is going to want to let drugs, you know, you don't want to wait 40, 50 years to find out what happens to these patients. So what we do is we make the assumption that if we give large amounts of a drug to an animal that lives a very short life, we will see those effects early. And if the animal happens to have ultra long telomeres, you won't see those effects early. So, it's a perfect storm for causing us to release drugs that should never have been released into public.

Eric: Can you think of one?

Bret: Oh, I sure can. Vioxx, for example. So Vioxx was discovered to do heart damage, right? Heart damage. How do you, why do we know that it's heart damage? Well, the thing about hearts, for reasons we can get into maybe another time, hearts have a very low capacity for self-repair, right? That's why they're vulnerable to heart attack.

Eric: Not much turnover.

Bret: Not much capacity for repair, and not much turnover. Now, there's an adaptive reason for that, but hearts don't repair themselves very well in a healthy person. And when they fail, it's hard to ignore, right? If somebody who's 30 has their heart fail, there's questions asked, right? So anyway, Vioxx was released into the public having passed drug safety testing.

Eric: This isn't the only system that doesn't have a lot of mitosis, like for example, neurons.

Bret: Neurons don't have a lot, cartilage doesn't have a lot.

Eric: Got it.

Bret: Your eye cells don't. Now note, all of the tissues I've just mentioned, when's the last time you heard about anybody having, you know, cancer of the cartilage, of their knee, cancer of the heart,

Eric: If they get brain cancer, it tends to be glial—

Bret: It's glial cells, exactly. So the tissues that have very low capacity for self repair do tend to wear out and they don't tend to get cancer, which is exactly one of the predictions of my paper.

Eric: Right.

Bret: Okay. So Vioxx is known to do heart damage. That created a big scandal because how the hell did it get through drug safety testing? It turns out a lot of drugs have done this. We've seen it with Gleevec, Fen Phin, Arithromycin. Your doctors probably still doesn't know that Arithromycin does heart damage—

Eric: Yikes

Bret: Right. There's all of these cases of drugs that were released and then later understood to do heart damage. Now my claim is they don't actually do heart damage. They do cellular damage and the heart is the most conspicuous.

Eric: Yeah, yeah, yeah, yeah. Geez. This is like another layer of this thing.

Bret: It's like a huge fucking nightmare, right? Because—

Eric: Well, but it's this thing about, like, perseverance and disagreeability. You've got all sorts of things that *sound* like something that invalidates the theory, and then it's sort of theories upon theories that allow you to see the original simplicity of the idea. I see the original idea is very simple—

Bret: Yep.

Eric: —but if you know a lot of weird facts about what you think are just mice, or something about hearts, you can't put together what is going on. The idea that ambient damages only manifest in the heart because that's the one system—you know, or the neural system—that, like, really doesn't have a lot of mitosis.

(01:44:17)

Bret: So, well, piece of advice to anybody who finds themselves in remotely similar waters. The signal that you are on the right track is that stuff starts canceling. Complexity in the story, which has accumulated because something was missing, starts disappearing in the story. You begin to take on a model. Anyway, so yes, we've got a situation where we've got a bunch of drugs mysteriously producing heart damage.

Eric: K, so now you've got a paper that's out. You've got a real world application. You've got a theory coming out of evolutionary theory. It's making a molecular prediction.

Bret: Yup. Successfully predicts mouse telomeres.

Eric: One of the world's leading labs has confirmed the prediction.

Bret: Yup.

Eric: Where are we now? What year is this?

Bret: God, well, let's see. The paper came out

Eric: And my recollection—and, just to be horrible about this—is that your fucking department at the university of Michigan, which has some great people, is also holding you back and enervating you year after year by not allowing—because this is *groundbreaking* stuff. This is Nobel quality work, at least one or two times over, in my opinion. I could be wrong. I'm biased because I'm your brother, but what concerns me here is that you are not comfortable with what this story really might be.

Bret: No I— Look, it's not mine to judge. I'm very proud of this work and the work—

Eric: But the problem, Bret, is that Jerry Coyne and Richard Dawkins did not know that Dick Alexander, Leonard Hayflick, and George Williams were all on this thing, because that community had broken down.

(01:45:59)

Bret: You know, the irony is, I sent a letter to Dawkins when this was going on, asking for his help, and he sent back a letter saying, “This is very interesting. It's not my area of specialty. You should talk to Bill Hamilton.” And I was in the process of writing a letter to Bill Hamilton on Dawkin’s suggestion, at the point that Bill Hamilton came back from Africa having—he was pursuing a remote hypothesis about humans having accidentally unleashed AIDS into the world with a polio vaccine. But anyway, so—

Eric: Bill Hamilton, I'm sorry, not everybody's going to know—this is the guy who came up with inclusive fitness?

Bret: Yes. He was one of the great geniuses of evolutionary biology in the late 20th century.

Eric: He was held back by John Maynard, right?

Bret: I don't know that story. I, you know—

Eric: I think Maynard is interviewed on Web of Stories where you—

Bret: Maynard Smith.

Eric: Yeah, sorry. Yeah. Maynard Smith. Right.

Bret: Yeah.

Eric: And Maynard Smith talks about like, you know, “It was very unfortunate. I didn't really understand who he was.” You should check it out. It's pretty amazing.

Bret: Well, as long as we're doing this, years after this story had cooled—

Eric: Yeah.

Bret: —I ran across a paper from John Maynard Smith that, I now don't remember exactly what its nature was, but it appeared to predict my whole story.

Eric: Uh-huh

Bret: Right? And John Maynard Smith was dead. I couldn't contact him. I really wanted to say, "Oh my God, you *nailed* it." Right. But anyway, so I was in the process of writing to Bill Hamilton to get his help. You know, he was sort of on a par with George Williams, and he went into a coma on his trip back from Africa having contracted malaria. And then there was, I think complication with the aspirin that he took or something. And he never woke from his coma and he died, tragically. So he never got the letter, and who knows what he would've done.

Eric: Okay, but look, that's a tragic and interesting story, but Hayflick was positive towards you. Williams was positive towards you and Dick Alexander. Those were the three that blew me away. That's a *huge* amount of firepower.

Bret: That's a lot of firepower, and it wasn't enough. But, here's the punchline to the story, effectively. At the point that my paper is out—

Eric: Right.

Bret: —and it *very* directly alleges the danger with these drugs being released when they're not safe, and the drugs have started emerging and turning out not to be safe, and the government is now really interested in what's going on, the government puts together a FDA commission to study the question of—the book that they put out, literally a *book* that they put out, at the end of their study is called The Future of Drug Safety

Eric: I hope it's a Blue Ribbon panel.

(01:48:53)

Bret: It's not exactly clear what it was. What *is* clear is that you can search the manuscript of this book. Nowhere does it mention "mouse"—

Eric: Antagonistic pleiotropy?

Bret: It doesn't mention antagonistic pleiotropy. It doesn't mention the genus "mus". It doesn't mention "telomeres". It's not *in* there. It's alleged in the literature in broad daylight that this is what is causing the problem, and—

Eric: Now you're—see, this is the Vampire Effect, where you don't exist if nobody reacts.

Bret: Right. And, so I start going to members of the press, I think, “This is a huge goddamn story. Somebody is going to make—

Eric: “Oh my god, you’re self promoting!”

Bret: —career on it”, and I call up members of the press, and it's always the same, right?

Eric: Always the same.

Bret: It's always the same. They're very excited about this story.

Eric: No, they're initially, the reporter—

Bret: The reporter is excited.

Eric: Yep

Bret: And then the reporter—

Eric: Talks to someone.

Bret: They talk to someone, and then either they stop returning your calls, or they say, “I'm sorry, the story doesn't hang together”. It's again and again and again.

(01:49:48)

Eric: Yeah.

Bret: And there's just nothing you can do.

Eric: Remember what I said about the Distributed Idea Suppression Complex

Bret: Yeah. And the people who man it don't even know what they are. For most of them, they don't know what role they're playing.

Eric: Look, you see the same thing with like string theory because none of the reporters are actually string theorists, so they're dependent upon this. You saw this with this woman alleging that she had the Epstein story three years earlier, but that the editors said, well, we might lose access to the baby pictures of the Royal grandchildren like, you know, you're seeing this with catch and kill. There's this, I mean, I want you to take this seriously. You're just showing a part of what I'm calling the DISC, the Distributed Idea Suppression Complex. We have 50 years of such

stories, and it happens that in our family, three out of four of us created such a story trying to get a PhD. And the idea for me is that every time you have to go into some closed system, like, there's a committee meeting or there's a blue ribbon commission or there's a peer review process, or there's a, what do they call them, the panels—study groups, for grants. That's where the DISC lives. We know that it's localized to the things that protect the integrity of science. It's an autoimmune disease, where what we have is an ability to stop highly disruptive ideas from getting a hearing in the general population of experts, by virtue of the fact that a carefully chosen group of experts can stop publication. Because look, if you're wrong about this stuff, there's a cost. It's not, it's not cheap.

Bret: No, I mean, in fact, it would have been career ending. I'm pretty sure, had I been—

Eric: I don't know that it would be career ending if it was done in good faith, but you know, this is my, my problem with this is that you're sitting on one of the great scientific stories—I would say that I've ever heard. But you know, I'm sort of, kind of saying, “Well, Bret, what happens next?” You know, obviously I know a lot of this stuff. I've forgotten it, but I lived this with you and this is, I can vouch that this is more or less the order of events as it was taking place, as we didn't understand what was happening.

Bret: So I have to go through the final Carol Greider chapter. In order for this story to fully make sense,

Eric: Where the Nobel Prize is given?

Bret: That's the very tail end.

Eric: Make sure you include that.

(01:52:46)

Bret: Okay. So at the point that my relationship with Carol is changing its tenor and she is becoming hostile and I'm not clear on what's going on, I contact her and I discover through talking to her that she and Mike are about to publish their paper on the long telomeres of laboratory mice.

Eric: So this is the Delta between a wild type and laboratory mice.

Bret: Yeah. And I'm shocked because she's told me they're keeping it in house and instead they've got a paper that there, she says in final revisions there that day submitting their final revisions to nucleic acid research with their paper. And I say, Carol, can I see the paper? And she says yes. And she sends me a manuscript, not the pre-print of the paper. She sends me a manuscript of the paper, no acknowledgements, no figures. And I contact her and I say, can I see the acknowledgements and the figures? She sends them to me, and I contact her and I say,

“Carol, I'm disturbed. This was my hypothesis that you were testing. I should probably be an author on this paper, but at the very least I need to be an acknowledgement in this paper so that I can go back and point to it and say that was”—

Eric: It changes everything. That it was a prediction. It wasn't just something that was stumbled upon.

Bret: Absolutely.

Eric: Yeah.

Bret: And her response is, “I have been through my email and I see no evidence of the communications you are talking about.” Now, when I said at the beginning that—

Eric: You had called her.

Bret: I had called her.

Eric: Holy Shit.

Bret: That was my, that was my error.

Eric: This is such fucked up. I mean, I don't swear a lot in this program.

Bret: Yeah.

Eric: But this is such fucking academic, petty, stupid ass bullshit. This is like one of the great stories of all times.

Bret: One of the great stories of all time, maybe, and human life hangs in the balance on this one.

Eric: No kidding.

Bret: Right. Okay. So Carol does get awarded the Nobel prize, Carol Greider, Elizabeth Blackburn and Szostak. Szostak, who mentions at the point that the Nobel Prize is awarded that he was shocked as all hell to get a Nobel Prize because his work was so deep in the history of telomeres that he just didn't expect it. And suddenly—

(01:55:14)

Eric: No, I should say, I want to be very clear, right. All of these people have made *fantastic* Nobel-worthy discoveries.

Bret: Totally.

Eric: There's zero allegation that these people—

Bret: Weren't deserving. No,

Eric: Absolutely.

Bret: No. And they, you know, Carol and Elizabeth got their Nobel prize for the discovery of telomerase, which is a huge, huge progress. So anyway, I don't deny that they were worthy of this prize. What Carol Greider does with her Nobel lecture, right. Nobel lecture being the biggest lecture a scientist will ever give, the lecture that—

Eric: And filmed.

Bret: And filmed—is she delivers a paper in which she very oddly has now embraced my entire set of hypotheses about the effect. She has come over from the comparison between the paper of mine that she panned and said didn't make any sense. She is now a total convert to the idea that senescence across the body is being caused by Hayflick limits that are telomere based.

Eric: Okay, and this is the first public incident that we know of in which the delta between the negative comments on your paper, which is not an anonymous peer review.

Bret: We have it in an envelope from her.

Eric: Got it. *And* it's immediately after the Nobel prize that the wisdom of that line of thinking is embraced.

(01:56:43)

Bret: Right. But there's more to the Nobel lecture. So she spends her Nobel lecture on what is admittedly a very beautiful presentation of the connection between telomeres and senescence. She goes through tissue after tissue, says cirrhosis of the liver is what happens when you have short telomeres and your liver, etc. She goes through tissue after tissue. She projects the data, the blot actually from the paper with Mike Hemann, the paper that I should have been a coauthor on, she projects it on the screen, but she does some weird freaking dance, where she, instead of describing the long telomeres of laboratory mice as a major bug in the system, she describes it as a happy accident, effectively, because it allows us to test certain things like, “Oh, isn't it delightful that they have long telomeres?” And it's like, what the hell are you doing? There is *so much* riding on correcting this and you're presenting it like it's just a bonus. And she, in her presentation, she's got several experiments that I did not know she had run that I had suggested to her and I said, you know, things like, “Carol, do you have any idea if a cell has many different

telomere lengths, is it the shortest telomere that controls how many reproductions a cell can do?" She's run that experiment. Interesting. Low and behold, it's the shortest telomere. It's a good guess. But anyway, so, she goes through this. There's no mention of me, there's no mention of the actual implications of the the long telomeres for things like science and safety testing and all of that. And I can't seem to raise the issue of the safety question with anybody. Right? At best, I get journalists who are interested until they call somebody, and the somebodies on the other end, I know what they say. They say "everybody that mice aren't great models". In fact, there's a paper out there that says something like the mice lie. It's not about this issue. It's just about the fact that mice aren't a perfect match. The issue in question could be solved. It could be addressed thoroughly. And, for all I know, once the Jax lab figured out what they were doing—

Eric: They could change the protocols.

Bret: For all I know they quietly have fixed this and there was a private, you know, I've heard that there was a private meeting in which they decided—

Eric: Look, this is the thing.

Bret: Yep.

Eric: You see something like this in statistics, everybody knows that most distributions that are bell-shaped are not normal. Right?

Bret: Yup.

Eric: And on the other hand, we all use normal distributions, and as a result, there are lots of things that at one level everybody knows—

Bret: Yep.

Eric: But don't percolate down to the important layers in which we test things. And I don't know where, like you and I have never been able to fully put together, cause we're not molecular researchers and I'm not even a biologist. How important are these results? How robust are they? Has there been a change? This is a quiet world at some level.

Bret: It's a quiet world. But I think what I have concluded, yeah, working backwards from the phenomenology of the field and how it reacts to this problem, is that there's a tremendous amount resting on failing to acknowledge the error. Even though the error was obviously an honest error to begin with, they would rather sweep it under the rug. I mean, imagine you've got all these knockout mice, right? These knockout mice, there's a major investment in them. It takes a lot of work to knock out a particular gene.

Eric: No, dude, you've got a central, you've got a single point of failure—

Bret: Right.

Eric: Whose projections are tendrils into everything.

Bret: Right? And you've got how many careers built on papers that are now suspect.

Eric: This is like an era. This is like a centralized irreproducibility crisis.

(02:00:37)

Bret: Yes, it's that bad or worse.

Eric: Okay.

Bret: And, and you know what happens if, let's say somebody hears this podcast and they check into it and they find out, lo and behold, this story is true.

Eric: Yeah.

Bret: Well now the FDA has a problem.

Eric: What would, wait, wait a second. I don't want to get too far out over our skis. We have enough listeners that people will get a chance to hear an unbelievable story. And if there are things in the story that are not true or misremembered or unkind or there've been changes or maybe we don't really fully understand how the drug testing works. I'm open and I, and I want to be very clear, and I want this in the podcast, I'm open to the idea that the most straightforward implications of the story are subject to adjustment. However, having lived the story, I can say that this was an egregious story at multiple points, with conflicts between the evolutionary community, the biomedical community, the professional publishing community. This is a terrible story, and it's also an amazing and beautiful and wonderful story. And you know, I felt really lousy at the beginning of this podcast goading you and prodding you. But I am so bored of you, no offense, as the guy who stood up to the funny kids at Evergreen, and you know, we know what's in the heads of these people. If you're at Evergreen, you're not that good.

Bret: Yep.

Eric: Right. And that was like, this is the, I just want to be open about it.

(02:02:17)

Bret: No, I look, I appreciate it, and I'm glad to have this story out. The story has many different layers of meanings. I know, I remember where I was when I finally sat down to watch Carol Greider's Nobel lecture and I had one of the oddest experiences of my life. I was actually in a hammock watching her lecture, watching her present my hypothesis without my name anywhere on it, and then she projects this image from her paper with Mike Hemann, and I was *flooded* with two simultaneous emotions that are just completely incompatible. Right? I've never felt anything like it. I was *absolutely* elated to see my work projected on a Nobel stage, right? That changed me.

Eric: You know I called the horse and rider problem?

Bret: No.

Eric: The point of the official complex of science is to knock the rider and take the horse, where the horse is the theory and the rider is the attribution.

Bret: Well, this was it. I was elated and livid simultaneously, and I can still almost feel what it was almost like my body was trying to figure out one half supposed to feel one thing and the other feels the other? But, this story has many levels of importance. Personally, it gave me the ability—I was already, as you are, very good at not being persuaded by the fact that everybody else disagrees with you, that that has an implication. Every great idea starts with a minority of one and you have to be able to endure being alone with a great idea in order to advance the ball significantly. This story was so extreme and so clear in the end that it just left no doubt. And I must say, I don't know how young students can arrange to confront materials so that if they're really good, they get a clear demonstration like this, that they're really good. So they know to keep going.

(02:04:40)

Eric: Bret, look, I think you're selfish, and I don't mean to be horrible about it. I think that the story is an inspiration. I've lived the story with you. I have my own version of the story where instead of it being the slide from the paper of Grider and Hemann, it's equations that are known as the Sieberg-Witten equations. And you see what you did, with somebody else putting, you know, putting it up on a board, it starts to change the field, and you suddenly say, you mean I'm not an idiot? Right? And what I'm claiming is that the next layer of this is, "Well, why don't you just submit a paper? If you have ideas, submit a paper, submit a paper, submit a paper." Who is this fucking suppose to fool?

Bret: Well, right, and this, this—

Eric: I mean, I just, I think the idea is that if you have a seat on the exchange, you know that by submitting a paper, your paper will get reviewed because you have, you present a credible

threat. It doesn't occur to you that what you're saying is effectively like "let them eat cake", to somebody whose paper is going to be reviewed by the person who's, like, holding them back.

Bret: No, this is exactly—when Jerry Coyne came at me with, you know, "Bret doesn't understand his, his explorer mode stuff is, is nonsense". And then Richard Dawkins echoed it "Bret doesn't understand natural selection. And, you know, if he did, he'd submit a paper." My feeling is, I lived this story, and you're going to pretend that there is even a mechanism to get a proper hearing?

Eric: Look, here's my proposal proposal. All right?

Bret: Yeah.

Eric: I think that you, Pia. and myself are indicative of an entire layer of GenX academicians, and now probably millennial academicians, whose work was suppressed, and we don't feel comfortable saying these words, which is that the purpose of the university system, in the time that we were there, was in large measure to make sure that big disruptive new ideas did not upset the apple cart because there was the ability to deny, I mean, this is what you guys call interference competition, which is that you keep people from sitting down in the chairs in a game of musical chairs. And then the idea is we have lovely parting gifts for our contestants. Doug Prasher, who did green fluorescent protein, ends up driving a shuttle bus in Huntsville, Alabama, features in the, you know, I don't know, was it the front page of the Science Times? A year later he's still driving a fucking shuttle bus in Huntsville, Alabama. Meanwhile, we're being told that Americans don't care about STEM. We're not really good at science, but thank God, thank God our friends in Asia are amazing at science, because, as bad as our children are thinking for themselves, we've got huge numbers of people who want to come from China, South Korea, India, and Taiwan in order to do the study in the labs, which is actually work, and I'm the guy who found the secret study in 1986 which says, "Hey, we're going to have to pay these American academicians over six figures very soon because of the supply demand relationships." And then they took away the demand curves and they only showed the supply curves. They said this was a demographic rather than an economic analysis, so price and wage certainly didn't enter into it. Like, our problem is that the American scientific enterprise, headquartered in the National Science Foundation, National Academy of Sciences, and our university systems is fraudulent, and it serves to suppress radical new ideas. And I'm not saying that everything is guaranteed to be right about your story, but this is a story that you and Carol should have warred out, in public, without your submitting into a system where you don't know who reviewed this, you don't know how to respond to the comments. You can't measure the delta where somebody in one year says, "this is crap" and the next year they say, "this is my theory". Right? And what I want. I would love to invite Carol Greider onto this program, because I think she deserves the right to rebut what you're saying.

(02:08:58)

Bret: Yup. That'd be cool.

Eric: And Elizabeth Blackburn is fantastic. I'd love to have—and these are great scientists.

Bret: Frankly, you're going to say, this is me being too nice.

Eric: Yeah.

Bret: I'd even like Carol to come clean and just put this behind us. I'm not, you know, at this point—

Eric: No, it's not a question of that Bret, there is—you have the right to offer somebody a hand up.

Bret: Yup.

Eric: But you're skipping the step of—let me be blunt—how many universities offered you a position after you were run out of this crappy Evergreen State College by a weak president who refused to stand up for academic freedom, freedom of speech, and anti-racism, which you exemplified.

Bret: Professorship? Zero.

Eric: How many biology lectures were you invited to give at top tier AAU universities? American Association of Universities? Or, Association of American Universities.

Bret: None

Eric: Okay. What the fuck is that? I mean, let's, let's just say the word “fuck” a lot, cause I had Andrew Yang in that chair. I don't say fuck a lot.

Bret: Yeah.

Eric: Okay. So the idea is you have a Maoist insurgency against a student of Dick Alexander, who is supported by George Williams, with support from Leonard Hayflick. He's predicting something from evolutionary theory, registers in molecular biology. It may have drug testing implications, and, like, *nothing*, silence. And you're terrified to talk about this.

Bret: I don't think I'm terrified to talk about it.

Eric: Well, I'm sorry. Can you tell me something? Where have you told— you have a podcast?

Bret: Yeah.

Eric: Where is the story written up? Where is the story lodged? You and I have the ability to lodge it. I'm forcing you to do this on my podcast. I haven't heard you do a podcast about this. I hear you talking about free speech. I hear you doing things with the heterodox Academy. I hear you doing things in the intellectual dark web, something with Andy know something with Antifa. Okay. The whole purpose of the Intellectual Dark Web is to keep the channel open based on merit, because if we do something like the diversity of ideas, you know, for all I know, the people who are suppressing you are more diverse than you are, you know? Okay. These are ideas that needed to come out. There are health implications potentially of these ideas. This is not ethical to suppress. In effect, it's not ethical for you not to talk about, not to be rude.

Bret: No, no. Look, I get this. I tried for a decade to get this story to come out. Now, I'm sure I would have been less aggressive on the social front. I would have let Carol go in order to get the story out and get the drug safety issue addressed. I don't know what you regard that as. Maybe that's—

Eric: It's not a question of this. Look, there is a Carol Greider and Elizabeth Blackburn and everybody else in like senescence land, Judith Campisi, who knows. Everybody's got a problem, which is there's way too much transparency, and there's too little funding, and there's not enough autonomy, and there's too much peer review, and for whatever reason, a new game has cropped up where everybody says we need more transparency, more diversity. We need to make sure that we're not wasting taxpayer dollars. We have, you know, ever more oversight. All of this is denaturing our society. We have to compete with China now. We are going to have issues with Iran and Russia, and we are losing our minds because we are serving a baby boom group. Almost, like, you pick a leading university. It is headed currently by a Baby Boomer. That's almost true without even telling—if I ask you, “Hey Bret, pick a university. Don't tell me which one it is.” I will tell you that the number of administrators that that university has soared above the levels of admissions, the tuition has soared above medical inflation, which is above regular inflation. If I ask you about the grant structure, older professors that are winning more grants and younger people are winning fewer grants. This is a giant complex. I am going to have somebody from Sugar Baby University, which is a subset of Seeking Arrangement, because the Baby Boomers made student debt non-dischargeable in bankruptcy. And now this group is offering older men the ability to date younger women with an allowance, right? So we're starting to get into gray area sex work, where the Baby Boomers to keep this lifestyle to which they've become accustomed are effectively enslaving—

(02:13:44)

Bret: Well, they're hoarding wellbeing on every front, including the sexual, which is no surprise at all.

Eric: But here's my claim: we are in a holding pattern. I'm in my 50s. You're in your 50s. I've done work that has never seen the light of day. You've done work that's never seen the light of

day. Pia has done work that's never seen the light of day. I don't know about Heather. My claim is: it's time to crash land the planes into the control tower. It's enough.

Bret: Wholeheartedly agree.

Eric: Okay. Bret, it's been an absolute pleasure having you on . Come back anytime. I want to say that anybody who is misportrayed by this podcast is welcome. We are not claiming to have absolute and universal knowledge. You are more than welcome to correct the story if you have knowledge about this that checks out. But the problem is that this is a story that needs to be told. It's like the story of Margot O'Toole and David Baltimore that played out at MIT, when, I believe that she found that she couldn't reproduce the work of Dr. Imanishi-Kari. And of course the system turned on the person who was trying to say, "Hey, I'm seeing irregularities. I'm seeing problems."

We have a biomedical complex that needs whistleblowers. It needs iconoclasts. It needs challengers. The food pyramid has been off for years. Our health recommendations are completely off. I think that this is an essential story. You need to move out of Intellectual Dark Web stuff, which was about keeping the pipe open. Let somebody else do that. And it is time to hire you as a professor at a top tier university. And I'll be happy to talk to you about what happened when you and Richard Dawkins encountered each other on stage in Chicago, because I think in terms of pure evolutionary theory, it is time to boost a young Richard Dawkins who contributed two of the most important ideas in the form of extended phenotype in the mean, which largely dislodges the old Richard Dawkins and his hatred of religion, which has appeared to take over his thinking as regards his own contributions to biology. We got a lot of work to do.

Bret: No question.

Eric: All right, my friend.

Bret: Well, thanks for having me.

Eric: Thanks for coming. You've been through with Dr. Bret Weinstein, professor and exile from the Evergreen State College. Please subscribe on Apple or on Stitcher or on Spotify, wherever you listen to podcasts, navigate over to our YouTube channel and not only subscribe, but remember to click the bell icon to be notified with our next episode drops. And hope to see you back on the next episode of The Portal.