This picture was taken six years ago. This is me and Dennis Noble from Oxford University, and I was invited to the Royal Society where that picture was taken, and we announced a $10,000,000 prize for the origin of the genetic code. And we get submissions about every week and nobody's solved it yet. But it's a very interesting question and Dennis is holding my book Evolution 2.0. And after we announced that, I hired a publicist.

And as you would all know, about nine months later, we got hit with this thing called the COVID pandemic. And I I got a call from my publicist, and she said, hey, Perry. The press is not interested in anything unless it's vital(?). So what do you got? Like, nothing.

She goes, can you try? I said, okay. I'll try. I will spend one day researching this new coronavirus thing and see if there's some tie in to Evolution 2.0 and my prize in this previous work. Turns out there was a connection.

And so one of the first things I found was what's on the screen is a genome sequence of a COVID 19 on the top, a SARS in the middle, and a MERS virus in the bottom. And you'll see that they're very similar, except that some of the parts are shuffled around, and some parts are present in one or two, and not all three, and some not. And if you pay any attention to standard explanations about virus evolution, what you'll usually hear is that it's driven by random mutations and genetic recombination. And I looked at that and I said, no. That's not what I see happening here.

That is not random. That is systematic and reminds me of several other things that I've seen before. If you go back quite a few years before that, I had made another connection. In 2002, I wrote a book called Industrial Ethernet for the leading trade organization in the process industry. And because I understand how Ethernet packets and how the data goes back and forth across the wire, when I started studying genetics, I saw a striking similarity.

It was that an Ethernet packet in DNA transcription and translation are almost identical. The math is identical. These are packets of data. They have segments. They have headers and footers, and I made that connection.

And back then, I immediately understood because I understood Internet data that there was no way that the mutations that drive evolution are random copying errors. There had to be a much more rich explanation than that. And so I wrote the book Evolution 2.0, which explains mechanisms like

symbiogenesis and horizontal gene transfer. And this started to become a fascinating rabbit hole. I'm like, so how do these viruses evolve?

I knew that the viruses did not have editing machinery of their own. I knew these sections of DNA were being moved around and I said, I think the host must be doing this. It's the only available explanation. And so I started making phone calls and sending emails and this presentation is an MD, three virologists, a geneticist, a cell biologist, and a cognitive psychologist walk into a bar. These are the people that helped me with this project.

And so here's what we found. So in my book, Evolution 2.0, I describe what I call the evolutionary Swiss army knife that genetically there are five major mechanisms of evolution that cells use to alter their own genetic destiny and ward off threats and adapt in the ways that they need to. Transposition, moving genes around, horizontal gene transfer, which is shuttling genes from one organism to another, epigenetics, which is switching genes on and off, and symbiotic mergers. There are several kinds of those, and they're on the screen. And my reasoning was that the systems that do this stuff are also editing the virus.

So what I'm going to advocate here is a view that virus evolution is mediated mostly by the host. It's not random copying errors. Viruses lack the machinery to do this, and code is exchanged across life forms and it's not at random. And the paper that we published two years ago is at the bottom there. It's called Cellular and Natural Viral Engineering in Cognition Based Evolution.

So first of all, when we wrote this paper, we didn't say a word about COVID. It was too hot of a potato. We made our case using entirely other viruses. And you can go read the paper if you wanna see that. Now that it's 2025 and I'm probably not gonna get decapitated for having an opinion about this, I'm gonna talk to you about COVID as well.

So, there are papers that show that if you analyze a virus before the host got it and after the host got it, there are edits made in the signature that they are signatures of APOBEC editor enzymes that are that humans have and ADAR enzymes. And the study showed that host driven edits dominate, and they are not random polymerase errors. And so these added enzymes are part of the human immune defense, and they are the tools and the enzymes that enable natural genetic engineering, which is what allows corn plants to, and bacteria, and really everything to modify their own DNA.

So the enzymes are actively editing the viral RNA inside the host cells. The edits are not random. They are targeted at certain areas and certain motifs, and they often occur in functionally relevant areas such as the spike protein. And so what is happening is our cells are trying to re-engineer the virus to make it less

lethal. And it's it turns out to be an advantage if the virus is more virulent because a less lethal virus inside your body will out-compete a more lethal virus, which is better for you. And so the virus is actually engineered by the host. Here's another slide showing that SARS CoV2, the virus is using APOBEC mediated mutations for its own fitness and evolution.

And this is a quote from the paper. It says, unlike the unpredictability of random mutations, this study has significant implications in predicting the mutations. Thus offering a basis for guiding future antiviral therapy in vaccines against the escape mutants. 96% of edits occur in the exon coding regions. So this is very functionally significant.

Now, I think this is medically very important because if you believe that evolution is random, you're at a dead end. And it means there's no pattern. If there's a pattern, there's something that can be done about. So the virus responds by engineering its own changes in our own defense mechanism, helps the virus evolve. And so now we're in a game of guessing your opponent's next move.

And so, when the pandemic was going on, we all heard about the different strains, Omicron, Delta, etc. And Omicron in particular suddenly emerged showing dozens of coordinated mutations. And it generated hypotheses that said cryptic circulation, chronic infection, animal reservoirs, and directed editing were involved in producing that mutant. So what I'm saying is that as the virus spread through the human population and as hundreds of millions of people got COVID, the change in all of those variants and the changes were generated by the human hosts themselves. And that's why the COVID virus went from being highly lethal and less transmissible, to less lethal and more transmissible, which is the general direction of virus evolution.

There's another version of this story, which is the placenta. The human genome contains about 8% endogenous retroviruses, which are viruses that have been forcibly inserted into the human genome and become part of our germ line. And research has shown that the human placenta was built from code that was originally inserted by an endogenous retrovirus. So when retroviruses do those insertions, it's usually very disruptive and it causes the host to become very ill. And now they have a bunch of code they don't need.

And what the body does is say, what could this be good for? And there is a protein called Syncytin that is expressed in trophoblast, and what this this slide shows is that the Syncytin protein became an absolutely critical part of the placenta layer. And so this is a layer of tissue that allows nutrients and oxygen to pass from the mother to the fetus and for waste to come back the other way. And it's the glue that allows this to happen. And this is a slide where where it shows the layers of the mouse placenta and the way that is constructed from the various proteins.

And I don't think I have time to go into full detail on this. I'm gonna keep going. There's another function of viruses which is a symbiotic relationship between - so panic grass is a grass that grows in Yellowstone in extremely hot soil temperatures. It can go up to 65 degrees C, and it's possible only because there is a three way relationship between the grass, a fungus, and a virus. And if you take out the virus or the fungus, the grass cannot survive the high temperature.

But when things are working together, it has temperature resistance. Viruses function as gene vehicles across species and across ecosystems, and we know that they can pass from bats to humans and they can go to plants and all kinds of animals and microorganisms. Viruses are often a huge factor in certain kinds of cancers, and viruses accelerate evolution. We believe that viruses are the open source repository for code to be exchanged in evolution. Viruses are the GitHub of evolution.

It's open-source biology where genes are molecular code, hosts are developers and testers, and viral platforms are the distribution channels of that code. One of the phrases that we use in our paper is “viruses alive or not?”.

So people often ask the question, is a virus alive? Here's an analogy. If you're driving in your car and you put a CD in the CD player and it starts playing a song you love and you start dancing. Here's my question. Is the CD alive?

No. Is the sound waves from the radio alive? Is the song is the sheet music alive? No. Is it alive once it's inside your head and you're dancing?

I say, yes.

Not only that, you might change the song and go to a campfire and sing a different version, and now you modified the virus. This is exactly how it works. A virus sitting on your kitchen table is not alive, but a virus in your body is just as alive as any organelle that's in any of your cells. And so what we say is viruses, alive or not, are liminal life forms capable of co engineering with host cells.

And yes, of course, they cause disease. They also have a broader role, which is driving adaptation, enabling innovation, supporting resilience in ecosystems. They are related to transposons, which are absolutely critical elements in the ability of all of our cells to adapt to threats. And so it reframes evolution. It's not just random mutation selection, but it is a cognition based information exchange.

One of the assumptions that all of the people in our research group have is that all living cells are cognitive. There is a great paper by James Shapiro from, I think, 2021 by that title. All living cells are cognitive - and he's a very thorough researcher. And he makes a robust case that all of the signatures of cognition are present all the way down to the cell level.

My work in cancer evolution shows that when a cancer cell goes rogue and decides to become a terrorist, it is a cognitive entity that is now functioning as a separate organism. It is no longer you. Cancer is a disease of identity, and cancer is the cells of your own body evolving out of control.

And so intelligent cells and viruses solve problems collaboratively. And so if you draw a tree of life, it's not a complete tree unless you consider the fact that between all the branches of the trees are exchanges of virus code going from anywhere to anywhere else. So natural viral engineering says that viruses are not just pathogens that make us sick.

They are also tools that help life evolve. And just as people share and share ideas in open source software, viruses do the same thing. And evolution is also teamwork and cooperation. And thanks to my collaborators, Bill Miller, Arthur Reber, Frantesek Baluska, James Shapiro, and Luis Villas, and a couple others.

Unintelligible...

I do not know the answer to that specific question.

I can give you a little bonus piece of information, which is that Mike Levin at Tufts University has done really interesting experiments where he takes a cell out of an organism, out of a frog. He calls those xenobots - out of a human and he calls those anthrobots. And one of the things he discovered so if you take a human cell out of a human and you put it in a petri dish, the age clock resets. And those cells do not necessarily think that they are 60 year old man anymore. That has really interesting implications and we're, like, only scratching the scratch on the surface of what that means.

I do not know the specific answer to your virus question though. Who else?

Mark: Thank you for giving viruses that they deserve reputations.

Perry: Viruses get a bad rap, everybody. Give them a break.

Unintelligible...

So in general, in medicine, there is almost no medicine that actually heals any disease. Mostly, they just deal with symptoms and receptors and things like that. And most medicines are very mechanical. And Mike Levin has a saying. He says, I believe the medicine of the future is something like physiological psychiatry.

And what he means by that is talking to tissues or listening to them in or asking them, what is the problem here? All cells communicate. There's an immense - one of my favorite TED talks is by Bonnie Basselor at Princeton. And it shows that even bacteria have linguistic structures. They build these molecules.

They send them back and forth. And it's as linguistic as human language. There's a paper called, I think it, Sungchul Ji has a paper about the linguistics of DNA. It's like it has 10 out of 13 characteristics of human language. And so I think the next evolution in medicine is getting cells to talk to each other rather than for this the thing that's fighting the disease to evolve the way that the disease does.

And, of course, that's what our immune systems do. And so I'm not a virologist. I think you guys can all tell. But I do think that this would completely change the way that you approach a virus. So talk more after the break.

Yes, sir.

Attendee: The elephant in the room, back to the first or second slide. Yeah. Did the COVID come from bats or from the lab?

Perry: If you want my personal opinion, my opinion, I think the Wuhan Institute of Virology lived up to its name. And all it would take - so there's a paper from 2013 where a bunch of miners went into a cave and got a horrible respiratory disease and died, and it looks an awful lot like COVID.

We know that people from the Wuhan Institute went to that cave and they brought viruses back. We know that the US, Canada, and China were doing gain of function research at the Wuhan lab, and so I don't know. All it has to do is get on somebody's shoe. That's my opinion. I don't know if it's right or wrong.

Seems like where there's smoke, there's fire. Alright. I got karma.

Unintelligible...

Yeah. I think the answer would be a very small number.

James Shapiro has a very interesting line in one of his books. There are no hard limits on horizontal gene transfer. In other words, however improbable it may be worm to horse, and I think it's also important to be clear that the different kinds of evolution mechanisms have different ability to work at different levels. So you're not gonna get major evolutionary events from transposition, but you can from hybridization. Igor's talk was about thirty minutes ago, and you can get major evolutionary events through symbiotic mergers.

I think symbiotic mergers are the real heavy weight lifters. So thank you everybody. Appreciate you.