(00:00):

With Geert Vanden Bosch. Hello, Geert. Um, first of all, I want to thank you for taking the time today to, you know, really get into the details of your theories and issues with this vaccination program, with this pandemic and, uh, the SARS coronavirus. So thank you for taking the time. You're very welcome. Thanks for having me and for our audience. That's watching. And as you know, you are in Brussels Belgium. I am in Austin, Texas, but through the magic of filmmaking, we're sort of sitting in the same room virtually because I wanted to put out a video that really feels more intimate because we're going to get very intimate on this conversation. So to begin with, I have done several shows now on the different interviews you have done over the last almost half a year, last several months, you put out a video very early on from your own social media page, a warning, the who, that there was a real problem with the approach towards the SARS coronavirus for SARS cov two,

(01:09):

The aquatics at the who, my name is here, felon Bush. My background is veterinary medicine. I'm a certified expert in microbiology and infectious diseases. I have a PhD in virology and I have a long standing careers in human vaccinology. I'm urging you to immediately open the scientific debate on how human interventions in the COVID-19 pandemic are currently driving viral immune escape. I'm urging you to invite me for a scientific hearing, open to the public and to scientists all over the world on this very topic, ignoring or denying the impact of stringent infection prevention measures combined with mass vaccination using prophylactic vaccines is a colossal Belinda. Please do listen to my cry of distress and let's first and foremost, deliberate on a scientifically justified strategy to mitigate this tsunami of morbidity and mortality that is now threatening us. And let's meanwhile, devise a strategy to eradicate the steadily emerging, highly infectious variants on behalf of humanity.

(03:02):

I sincerely thank

(03:04):

You for considering my call

(03:07):

To begin with this time. Uh, I want to start out with sort of your background. What is it specifically about your experiences that is giving you a different perspective than we're hearing from other a world renowned scientists?

(03:23):

Well, I think it has in fact more to do with what I decided back 10 years ago, 10 or 12 years ago, uh, where I decided I would take a completely different approach to science, but it started all with my intention that I would start all of a sudden to look at the science, not as, you know, an objective in its own, right, but as a tool to solve problems, uh, because I was really very much bothered having worked in both academia and in industry, I started thinking more and more about these two different worlds. That boat start with P the worth of the publications. That's what counts in academia and the world of products. That's what counts in industry and in between everybody talks about translational medicine, but it's very, very rare. In fact, that we find solutions that we use the science to really solve problems.

(04:31):

And of course my background is veterinary medicine. That's very broad, that's very diversified. And, um, I had the chance during my career to touch upon several different fields. I specialized in virology, uh, in vaccinology. I learned my vaccinology. In fact, in industry, that's also the place where I learned, uh, uh, immunology. I was teaching zoonosis. So infectious diseases that can be transferred from animals to humans at the, uh, university I had, uh, I have a longstanding interest in evolutionary biology. And all this is, is, uh, has to do with interactions between the pathogen and the host. So the host immune system and understanding these interactions is very, very important. And then what's also very important is to understand the evolutionary capacity that a pathogen has when it is put under widespread immune pressure. That is something you don't have in clinical studies, for example, right?

(05:41):

This is the population effect. And so they simply illustrates that you have to, to be able to draw from all these several different fields. I was so fed up that we put many products in the pipeline in industry without even understanding how they interact, how they work. Exactly. And I was so fed up in academia that all, what counts is, is publications. We need to connect both because otherwise we are in a situation right now where we put out products without understanding what's going on without understanding the pandemic. And, and, you know, my point I'm 200% convinced that this is going to lead to a catastrophe, a catastrophe to, to a disaster

(06:23):

Gear. You worked for Gavi on the Ebola vaccine program. You've worked with the bill and Melinda gates foundation. Now, obviously you didn't start out by deciding to put your career in jeopardy as a world renowned vaccine maker, as a scientist, as a, as a professor, uh, you must have reached out to your, your peers at the who, who I know you've worked with before at bill and Melinda gates foundation at, you know, Gavi. Um, what did, how did you reach out and what was the response?

(06:57):

Well, they're alive reached out, uh, at least three times two, I would say all the global health, uh, authorities, a number of public health authorities, a CDC, a NIH, the bill and Melinda gates foundation, uh, who of course. And, um, I got maybe one or two anonymous responses. Maybe I said this in an earlier interview, and I'm not going to disclose his name, but it is certainly one, if not the most famous vaccinologist on this globe who told me gives you a right. These vaccines are basically just going to breed variants, but nobody is going to listen to you because you go against the main street. Right? I mean, imagine, imagine it is just, I have no words for this. I have no words for this.

(07:52):

Are you surprised when you got no responses? It's the first time you've reached?

(07:57):

No, I was not surprised. No, he Ebola story. I mean, it was the same, but it was a small scale that it was small. It was just, just the countries in west Africa. What was

(08:08):

Your job on the Ebola vaccine?

(08:11):

I was coordinating, uh, the Ebola program. And part of this was the vaccines. Uh, it was not just the vaccines was also about a number of measures that, uh, needed to be taken, uh, you know, to control moralists the, uh, Ebola rises in collaboration, of course, and met with who and, and UNICEF and, uh, CDC and, uh, uh, strengthening, uh, health measures, et cetera. Uh, so it was a collaboration between all these, uh, international, uh, health authorities. But of course, since I, uh, came from the vaccine field, I was very much interested in, in the vaccines would, uh, uh, be deployed and that would be used.

(08:59):

And what was that issue? Uh, when it came to evaluating the effectiveness of the vaccine and the safety of the vaccine,

(09:06):

Basically it came down to, uh, the naive interpretation of those who conducted the study that the incubation time of, uh, Ebola was well, either don't remember exactly 10 or 12 days, and that therefore they would not need to start the clinical observations before. So many days after the vaccination, after having identified the index case. And then you have the contacts that were all vaccinated. The endpoint was vaccine efficacy after, you know, so many days after the vaccination, but the vaccine that was used was alive, uh, vectors that everybody knows. And I come from the adjuvant fields as well is very, very strong in inducing, inflammatory cytokines. If you study the pathogenesis of a baller, you will find out that people die of a cytokine storm of a huge inflammatory cytokine storm. So what do you think happens if people who are incubating this because you were, you identified the index case, and then you were immediately vaccinating the contacts who potentially were already in the incubation phase and would have been the ideal controls. So these people are already incubating a disease that leads to inflammatory cytokine storm. And then you come with a vaccine that is well known to stimulate some pro-inflammatory cytokines. So these people, those who were infected ever vaccinated, they simply didn't make it till they 10 before they started the observation.

(10:53):

So let me get this great. They basically said, we know the incubation to be, if he bowls to be about 10 days, therefore when we find the index case, the person we know that's infected, they went and vaccinated. Everybody that had come in contact with them, right? Ring vaccination, ring around the index case, but they said, let's not collect any data for the first 10 days because we know that abandon is going to happen inside the 10 days. It's, you know, it shouldn't count. And what you're saying is you gave an inflammatory vaccine to people that were potentially incubating a highly inflammatory disease. That's what we know, the hemorrhaging, all the issues you are then creating a perfect storm. These two things coming together, where obviously, if someone has already had a hemorrhagic disease of huge site of climb, and it was also driven by the vaccine, those people are going to die. So did you ask to see any data of those that obviously there were potentially deaths happen there that weren't making it into the evaluation of the safety and efficacy of this product? Did you reach out to the who to say, can I see the data before the

(12:06):

10 days? The question was very, very simple. We just wanted to have the case fatality rate over the whole period as of day zero from vaccination compared to in the vaccine is versus the placebo just from the very beginning and just compare the case fatality rates. So what is the case fatality rate in the vaccines versus the placebo? Right from start, I was working with Garvey and ask that question to who

we never got an answer because it was confidential. And then, uh, I remember I was on vacation and my boss send me this paper in the lens set, and which revealed that it was 100% efficacy. I immediately knew that there was something wrong, but of course this was so to say small scale was only west Africa. But just to, to tell you that this is not the first time that I'm taking a deep dive in such things, because I cannot stand it. That first of all, the science is violated and that people who cannot, who, how can layman, how can layman understand the science? It's very, very complex, right? For me, it's not even about, you know, uh, freedom or, uh, about, um, uh, conspiracy theories or, or even about side effects. Okay. The side effects are important, but this is about a global health drama that affects every single individual.

(13:40):

Let me ask you a question that I'm not sure I've seen you answer, but you've done a lot of different interviews explained to me in, in your understanding what would have happened. Had we basically really just done nothing, had we, no one ever like screamed, there's a deadly brand new, you know, pathogen, a sweeping the planet, no vaccine product, uh, is raised on the market. What would have happened if we just sort of let things go naturally? Well,

(14:12):

We would have had a pandemic, a natural pandemic and a natural pandemic that would have primarily, or almost exclusively affected. I call them vulnerable people. You know, these people, these are typically elderly people, people who have an immunosuppressant immune system and an aging immune system, or people with underlying diseases or people who are, uh, otherwise, uh, immune suppressed, the only way to calm down a pandemic is to cut a transmission. And that is only possible through herd immunity. So we would have had a couple waves. Some people indeed would have died, uh, depending on what would have been the possibility is for treatment of this, uh, elderly, uh, people and also people, uh, uh, who, who are immune suppressed. And then of course you have the young people, typically I'm saying, this is not a childhood disease who have a very strong innate immunity and basically, uh, or not affected at all.

(15:18):

They at most develop asymptomatic infection, right? Asymptomatic infection. And then of course you have in-between people who would not have sufficient innate immunity to be protected against the disease. Those people would have gotten the disease would then recover and would then have built a long lift, uh, acquired immunity. So that is typically how pandemics curved and within one year the virus would not have been eradicated, but it would have been under control under control due to herd immunity, to a large extent due to the innate immunity that all youngsters and people in good health have.

(16:03):

Okay. Now I hear two terms that I think I, that many of us have been interchanging, perhaps inappropriately. I hear you saying naturally acquired immunity and innate immunity. And, um, I think many of us are confused that there's a difference between the two. So what is, first of all, is there a difference between innate immunity and natural acquired immune?

(16:28):

Yes. Yes. And, uh, what we call innate immunity very often. And I'm very often talking about innate antibodies, uh, in contrast to the naturally acquired antibodies, the innate antibodies, these are pre-existing antibodies, antibodies that, uh, or already, so to say, pre-primed that you have at birth already, not as a result of antigen experience, right? So a new born, all of a sudden gets confronted with a number of pathogens. Yeah. He or she, you know, the newborn can not have, uh, all these antibodies because has never seen these pathogens. So there is that this is providing them with a good start. The innate antibodies were found in every vertebrate species also to tell you how important they are from an evolutionary viewpoint that they are in all that you find them in all vertebrate species. So this is something that, um, we have been completely, uh, neglecting.

(17:31):

And these are basically the antibodies that protect all these young children and people in good health from a disease that is therefore, that is there for not a childhood disease, right? [inaudible] so the cells that generate those innate antibodies, we call them B one cells. It is a particular type of B cells, uh, which is different from the B cells that generate the naturally acquired antibodies, the naturally acquired antibodies or antigen specific. First of all, they do have, they do have a high affinity for that particular antigen and they are long lift and they are typically acquired when we talk for example, uh, about SARS and, uh, yes, and, and, and other infectious diseases, they are typically acquired. Once you have, uh, gotten the disease and you recovered from the disease and they have a high longevity longevity. So they are, uh, almost, and even, even if they disappear, they generate immunological memory. And because they generate immunological memory, they can be recalled very, very rapidly. So that is also some misunderstanding. Many people are worried because their antibodies have disappeared in their blood. They should know that as soon as their body sees the antigen again, or is re-exposed to the virus that within no time, because there are memory cells, these memory cells will, again, start to produce these naturally acquired antibodies that then will protect them.

(19:09):

So just prior to explaining the difference between the innate immunity and the naturally acquired immunity, you were talking about in a natural infection situation where we didn't have a vaccine, had we just let this thing run its course. Yeah. I want to just sort of summarize now, make sure I understand this correctly, that you would've had those people that would get the infection. Usually the elderly that have comorbidities, they have other health issues that are bringing down their immune system, their ability to really fight properly. They're going to be at a high risk situation. Some of them, uh, could, will potentially die, uh, because they just won't handle the virus. Well, then you have this other group. Middle-aged people, you know, they can be different ages, but also that also represents sort of a different immune system, strengthened and ability. Now they're going to get the infection becomes symptomatic.

(20:04):

And in that process, their body will create these, these antibodies, the naturally acquired antibodies that have long-term memory. And that's really the immune. That's the only immunity we all really have ever talked about. For the most part. That's what we understand. My body sees this virus or a bacteria. It creates antibodies that are, suppose if I have a natural immune system, it lasts forever. If they're induced by vaccines, maybe not quite Rever, but for a long period of time as the goal of the vaccine. And then you referenced the children as though they have a little bit of a different system, they have an innate immune system, as I understand it. And when they come in contact with the virus, they may not actually ever get to that place where their body is making these naturally acquired, uh, antibodies

because their innate immune antibodies are so strong and capable that they end up just attacking the virus very well and killing it before there's a need to go. And, and I'm guessing all of that talk of asymptomatic, um, that we've heard about those asymptomatic cases would have been those where their innate immune system reaction was so strong. They never went into a symptomatic reaction, which would have driven more of the production of those naturally acquired memory antibodies. Is that, am I, am I getting that right? Yeah.

(21:29):

That is why we call it the first line of immune defense. Right? The innate.