Elite Learning

Podcast Transcript

Be COVID Correct: Vaccine Facts vs. Fiction

Episode 1 – COVID-19 Vaccine Development

The following transcript has been lightly edited for clarity.

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Transcript

DR. DANIEL GRIFFIN, GUEST: ... we're able to leverage about 20 years of mRNA vaccine research to generate a product that we thought was going to work. We were lucky it did. We were able to do phase 1 and 2 compressed. Normally, the FDA would say do one, take your time. What's the rush? Well, there was a rush.

(SOUNDBITE OF MUSIC)

DR. DEBORAH MARTIN, HOST: Hello and welcome. I'm Dr. Deborah Martin for Elite Learning.

When it comes to vaccines developed in response to the COVID-19 pandemic, there has been no shortage of discussion – among healthcare professionals and the public alike. Some hail the vaccines as a medical miracle; others have been skeptical of the science underlying the vaccines, particularly since their development appears to have occurred at breakneck speed.

The skeptics are not small in number. According to the CDC, about 15% of adults in the U.S. remained unvaccinated in early 2022; that's roughly 38 million people. Among healthcare workers, the ranks of skeptics was also high. The latest review, conducted by the CDC in September 2021, showed 30% of people employed in healthcare had not been vaccinated at that time.

As our guest noted at the top of this episode, the COVID-19 vaccines were developed far faster than previous vaccines. The path from viral sequencing to an FDA-authorized vaccine took just 11 months as the scientific community pushed to develop tools to help reduce the illness, suffering, and death caused by something we hadn't seen before, a novel virus. And one of the tools developed, the mRNA vaccines, was novel, too. Although tested for years, mRNA technology hadn't been previously rolled out for clinical use. That's a lot of new for people to absorb.

We don't usually see pharmaceutical feats happen in real-time. Saad Omer, a vaccine researcher at Yale University, pointed this out when he tweeted, "This is evolving science. You are seeing sausages being made — in front of the world's eyes."

To help us make sense of the sausage making, and how the COVID-19 vaccines work, we were joined by Dr. Daniel Griffin, a physician scientist who is board-certified in internal medicine and infectious disease with expertise in global health, tropical medicine, parasitology, and virology, including COVID-19. You can learn more about Dr. Griffin's background in the show notes that accompany this podcast.

Dr. Griffin, some people have hesitancy about COVID vaccines, due to the quickness in which they came to market. It would be great if you could explain how that happened. And I don't know if you want to start with that, if you want to lead with that, or if there is something else you want to start with first. Because that could be a heavy topic, just in and of itself.

(LAUGHTER)

GRIFFIN: No, I agree. Well, welcome, everyone. Glad you're here. And what I am hoping is that by the end of our discussion, everyone feels really confident, they feel educated about vaccines. And as a consequence, they're able to make a decision that they feel is not frightening, that they feel is an educated decision. So I have a number of objectives. I always like to lay these out

(CROSSTALK, LAUGHTER)

MARTIN: That's a great educator in you. Yes, thank you.

(LAUGHTER)

GRIFFIN: OK. So one is I want people to list. It's not a long list – but list the different types of vaccines that are available for COVID-19. Fortunately or unfortunately, how you look at it here in the US, it's not a big, long list. It's OK. The other is I want people to actually understand. What are the potential risks of vaccines? But what are the actual vaccines? What are we seeing? And what are those risks for you as an individual?

And then what I'm hoping people can then do is apply this knowledge. So either they can be recommending, or they can be selecting the best vaccine for themselves, the best vaccines for their patients. Because different vaccines may be a different choice for different individuals.

The other – and I think this is really important – long COVID. We're going to discuss the impact of COVID-19 vaccines if you take these before a potential exposure. But what if you get exposed? What if you get infected? Should you take a vaccine afterwards? Is that going to help with long COVID? Do people get reinfected? Getting vaccinated – can that help you prevent that second infection, that third infection? Which I think we're up to four or five now in some people.

MARTIN: Wow.

GRIFFIN: And this is really important because I think this is confusing a lot of people. What's the difference between vaccine efficacy against infection and the vaccine's ability to prevent you from getting infected, versus vaccine efficacy against disease?

MARTIN: Good point.

GRIFFIN: So we've got a lot to talk about.

MARTIN: We do.

GRIFFIN: And I have my case study that I start with. So let's think of a real person. We were all probably thinking about ourselves. But here is a 70-year-old man with hypertension, diabetes, a little bit of a weight problem. And he is considering vaccination options.

He's already taken the plunge. He already got his first dose of the Pfizer-BioNTech vaccine. But he had a reaction, went ahead, was seen by an allergist, and was actually diagnosed with a PEG allergy. We'll talk a little bit about what that is. And he's here to discuss his options. And he just really wants everyone here to really understand vaccines. He really wants us to lay it out.

So what are we going to be talking about when it comes to the vaccines? He wants to know about the different studies. How do we get the information? What are the different types of vaccines? And how do you make these decisions when some people feel like we don't have as much data as we would like?

MARTIN: And when you say he had a reaction, some people say they have a reaction when they have a sore arm. And then other people have reactions that are much more impactful than a sore arm. So did this gentleman have a big reaction or was it an irritating reaction?

(LAUGHTER)

GRIFFIN: So that is excellent. That's one of the first questions we asked when someone says, I had an issue. I had a reaction to the vaccine. Is it expected reaction? Is it reactogenicity? Is it, you basically had an immune response? You saw what we were hoping the vaccine would induce by turning on your immune system.

Did you get a little bit of a fever? Were there some swellings of the lymph nodes? Did you feel a little crummy, all that immune reactogenicity? Did you maybe get a little redness? Or maybe it hurt where you got that vaccine. Or did you have trouble breathing? Did you feel like your throat was closing?

That's really the distinction. We're talking about the difference between normal, expected reactogenicity, evidence that the vaccine is working, and an allergic reaction where I am having trouble breathing. I'm covered in this horrible rash.

And I think that is really important as that guides us. Because we hear a lot of people got that vaccine and took a day off from work, feeling a little bit – that's expected. That's reactogenicity. We're OK with that. This gentleman actually had trouble breathing. He's one of those rare – but individuals who had that.

MARTIN: Thank you for that clarification.

GRIFFIN: No, thank you. So let's go through. I mean, I think your question like, how do we find out that these vaccines are safe? What studies, what science goes into it? Because a lot of people have raised this concern, right? I mean, early on, how quickly can we get vaccines? And initially, we thought it would be many years. And when we got them in a year, they said, ooh, that was too fast.

MARTIN: They can't be good if you did it that fast.

(LAUGHTER)

GRIFFIN: Exactly. You go back out there, and you work some more. You come back in a year. Well, how do we do this? And part of it was financial, part was compressing what we normally do. And how did we speed things up during the pandemic? Normally, we go through a number of study phases.

We start off with a phase 1. This is just a pure safety trial. We give the vaccine to a small number of individuals, and we do a few things to see if it's actually stimulating the immune system. Then we move up to a phase 3, where we expand this a little bit more.

Now, we're starting to give it to hundreds of people. We're starting to give it – people in a broader range of age. We're looking and seeing. How well do they tolerate this? So this is safety. And we're also starting to get a sense of, what degree of immune stimulation are we getting? We're hoping in these first two trials, we're finding not only, is it safe or not? But also, are we seeing those immune stimulation blood levels, things like antibodies? Maybe looking at T cells, things we'll talk more about.

Now, what we did in the time of the pandemic is that phase 1 and phase 2 are pushed together. Instead of it just being 10 or 20 people, we say, no, let's go ahead. Let's give this to 100 people. Let's accelerate and make what we call a phase 1 and 2 at the same time. So we sped that up a little.

The next thing – and this is the phase 3. We call them phase 3 efficacy trials. These are those big placebo-controlled trials where we actually look at efficacy. And we start to look at less common side effects. So these are the trials that we're going to talk about 30,000, 40,000 people, half or a third getting placebo, the rest actually getting the vaccine, and then watching and seeing during a pandemic.

How many infections are we seeing? How many people are getting severe disease? Now, these are expensive to do. If you're not during a pandemic, how often is someone going to get an infection? How often is someone going to get in the hospital? So a couple things we were able to do.

So we're able to leverage about 20 years of mRNA vaccine research to generate a product that we thought was going to work. We were lucky it did. We were able to do phase 1 and 2 compressed. Normally, the FDA would say do one, take your time. What's the rush? Well, there was a rush.

MARTIN: There was a rush as millions of people were succumbing to the virus.

GRIFFIN: Yes. Yeah. And then the big thing with phase 3: Phase 3 trials are really expensive – tens, hundreds of millions. It can really cost a tremendous amount. And so who's going to foot that bill? Who's going to pay for that trial? What if it doesn't give us the answers we want?

So that was Operation Warp Speed. That was companies like Pfizer just entering, anteing up, and taking the risk, and saying, you know what? We are going to go ahead, and we're going to throw money at this problem. And, boy, throwing money at problems can speed things up, as we saw.

So just how did it happen so fast? There was nothing magical going on. It was just really all these things being compressed. And then during a pandemic if you say, well, we're going to watch and wait for the first 100 people to end up sick or 100 people end up in hospital, well, during a pandemic, you don't have to wait very long.

MARTIN: They're showing up at your doorstep in no time.

GRIFFIN: Yeah. And I think we also have to thank the public, right? A lot of people signed up to be citizen scientists. So we were involved with some of the J&J early investigation. And what a lot of people did – and by a lot, I'm talking about a million people volunteered. And they said if you want, I will be in a trial to test the J&J vaccine.

We created readiness cohorts. So the same day that J&J got the go-ahead to do their phase 3 efficacy trials, we had a million volunteers scattered around the country. We could look and see. Where are we seeing the most COVID cases? Where are we expecting to see them in the next two or three months after we vaccinate? And then we had 30,000, 40,000 people enrolled within a week or two.

MARTIN: That's pretty amazing.

GRIFFIN: Fastest enrolled vaccine trial in history. And that was because people – citizen scientists were jumping in. I want to volunteer. I want to be part of the solution.

MARTIN: I like that, citizen scientist. And the contribution that they made was incredible. Were there other companies that also donated funds? Did the government throw money at this as well or was it just the big pharmaceuticals?

GRIFFIN: So Moderna, the mRNA vaccine. Moderna, the Spikevax. That was actually the US government, saying, we are going to give you the money. We're going to help you ramp up. We're going to give you the money to do these trials to produce this vaccine. Pfizer did it on their own. J&J got support from UnitedHealth Group, some other companies.

So a lot of people were coming. My colleague, Peter Hotez, who's worked on that vaccine down at Baylor that they're going to be distributing in India – Tito's Vodka. Everyone was stepping up. This was all-hands on deck. It was really a special time. This is not something that happens in a vacuum. This is what happens when everyone gets together and works together.

MARTIN: It is amazing what can be accomplished when the focus is laser-focused on a disease such as this.

GRIFFIN: It really makes such a difference.

MARTIN: And it really wasn't developed in a year. We could almost say it was developed in 21 years or 22 years. Correct? With a background.

GRIFFIN: Yeah. No, I think that is a great way to think about – this did not just happen in 6 months, in a year. At least for the mRNA vaccines, it was 20 years in development. For the adenovector vaccines like J&J, AstraZeneca, again, decades of people working on these problems. We're, in many ways, lucky, the timing of when this happened. If this had happened too many years in the past, we wouldn't have been ready. We wouldn't have been able to launch these vaccines so quickly.

MARTIN: Very impressed.

GRIFFIN: So let's talk a little bit about the vaccine types and what we're trying to do with these vaccine types. And a little bit of this is going to be that history lesson. What vaccines have we used over the years? And what are we doing now?

So some of the first vaccines that we're used to are these whole-virus vaccines.

And we're going to use polio as an example here. Let's think about an individual who has polio, and they get sick, and potentially have paralysis. Well, one of the first ideas – this was the injectable Salk vaccine – is let's grow up a whole bunch of that virus. Let's use formaldehyde. Let's inactivate it. Let's make it so that virus can't make people sick. And then let's let the immune system see it that way.

So that was our whole virus-inactivated vaccines. Some people went down that road. But what are the challenges? Right? Got to grow all this virus – huge, huge vats of virus. You've got to inactivate it, make sure it's not going to make anyone sick.

The early days of polio, there were some issues. People didn't do such a great job. Then there was the Sabin Sundays. I don't know if any of our listeners are old enough to remember Sabin Sundays, everyone going to the gym, getting their pink sugar cube.

And that actually had a virus which had been made weakened, attenuated. People took that sugar cube, were exposed, made a response so that should they actually encounter the wild-type polio virus, they would have that protection.

But now, we move into the modern era, protein-based vaccines. Think of that shingles shot. When we all hit 50, everyone should be thinking about that shingles shot. No one wants shingles.

MARTIN: No one.

GRIFFIN: Those of us that had chickenpox as kids, that virus is sitting there. It's waiting for an opportunity. And this was really just taking the protein. And a protein is something that's maybe on the outside of the virus, showing your immune system, letting it learn from this. So if that chickenpox virus tries to come out, our immune system damps it down. This is that Novavax vaccine we're waiting for in the US. We'll talk more about this, letting your immune system see that protein and then being ready to protect you.

Then we move into the mRNA. We call it the nucleic acid vaccines. And we'll go through a little bit of the immunology on how this works. But we're trying to let our immune system see the protein before we see it as an infection. So we're ready to go.

And here, basically, we call it the sticky note. We're giving our body the instruction manual, so our body can make that protein, makes it for about 48 hours. The immune system sees it, we learn. And then the J&J, the AstraZeneca style, the viral vector vaccines. You actually go ahead, and you take another milder virus, and you have that actually present to the immune system this spike protein that we'll talk a little bit about.

So, all right. Are we ready for some basic immunology?

MARTIN: I think so. And I will tell our listeners that you have provided some really good graphics. So if you're a visual learner, they will be available in our show notes for this episode. So be sure to take a look at the show notes. Because there are some really cool diagrams that will be in there for them as well.

GRIFFIN: OK, thank you for bringing that up. I am a visual learner myself. So all the visual learners out there, I'm going to be actually talking about basic immunology. And this will be up for you to look at.

So what is going on when we say teaching the immune system? Who is this? Who's this immune system? What players are we trying to teach?

Well, the big thing – that we're trying to teach are our B cells and our T cells. What is that about? And what is the difference?

Well, the B cell is that antibody-producing cell, right? We hear, I want to get my blood work. I want to get my serology. I want to get my antibodies checked.

Who's making those antibodies? It's the B cells. And the B cells are a go-it-alone, right? They can actually interact directly with the virus and start making those antibodies. They can go to our lymph nodes, work a little bit with others, get even better at this. But it's our B cells that produce a lot of those antibodies.

But I will tell you – and we'll get to this. Once those B cells start producing those antibodies, some of them keep producing the antibodies, some take a little step aside, but they remember. They're the elephants of the immune system. They're going to remember so that should you see this again, they're ready to jump into action.

The other side – and I'm going to say T cells will save the day. I feel like the poor T cells are not getting enough attention. The T cells, they need a little bit of help. Once the virus gets into a cell, there's a processing. There's actually special antigen-presenting cells that are going to show parts of that spike protein and other viral proteins to the T cells. And then the T cells are going to learn. They're going to expand. And again, they're going to remember as well.

This is that, we think, major protection against severe disease, so B cells and T cells. And we'll be talking a little bit about how the different vaccines – so just to run through those different vaccines one more time that we talk about. How do they connect with the B and the T cells?

Well, whether we're dealing with an attenuated, so a weakened form of the virus or an inactivated form, those viruses are going to end up in cells and professionally be presented to the T cells. Or our B cells are going to see that spike protein ahead of time. And now, they're going to be ready to go.

And we have some examples of vaccines that have worked that way. As I mentioned, the oral polio Salk, the oral polio Sabin vaccine, so the Sabin Sunday vaccine. And we have the injected Salk vaccine. So this has worked. And some people are trying those. Now, I will say, why aren't those prime time here in the US? Have not worked quite as well. We have better vaccines that we've been able to develop. So let's talk a little bit about those better vaccines.

So we're going to talk about the viral vector vaccine. So what do we do here? This is a little bit of cooking in the lab. We're taking a virus that is not that worrisome for us. And we're actually giving it a payload. We're having it present that spike protein to our immune system.

Our B cells are seeing this. Our B cells are starting to produce all those antibodies. Over time, those antibodies are getting better and better at binding that virus and neutralizing it.

We're also getting our T cells. Those proteins are getting into our cells. They're being presented, same thing is happening.

Now, prior to this, we had not used a lot of these viral vector vaccines. We had used them in the Ebola outbreak. At that point, we had given it to 10,000, 20,000, 30,000, 100,000 individuals. So with the rollout here of the COVID vaccines, millions now. And we're actually learning a lot about this technology.

Originally, the idea – we'll get into this with the J&J – was that one and done. Because people wanted that option. Can I just get one shot? I really don't want to. Well, pretty good with one, but not at the level where we want to get. So we'll be talking a little bit about that as a choice.

And then we move to the darling of the day, our mRNA vaccines, the fight between Moderna and Pfizer. Who's better? How close to 100% can we get? I was joking. My middle daughter, Eloise. Eloise, you already have a 95. What are you going for?

(LAUGHTER)

MARTIN: 100, right?

GRIFFIN: Yeah, 100. We are going for 100. And I have to say, going into this, we had two decades of work on these mRNA vaccines, trying to get them ready. But we had not brought any licensed vaccines. What are we doing here? How does this work? Well, remember, we're trying to show our B and T cells that spike protein.

We can inject it in the virus itself. But it's been weakened, or it's been inactivated. We can just present that protein in [sic] mass. We talked about the protein-based vaccines. Or we can do the sticky note. We can give the instruction manual to ourselves. We'd say, you know what? Take a bunch of this spike protein, show it to your B and T cells.

And that mRNA in our cells – everything is full of mRNA. We put that little bit of sticky note in there. And for about 48 hours, our cells are producing that spike protein. And then they crumple up the sticky note. They throw it out, but there's a memory that we saw that.

One of the things we have seen with the mRNA vaccines – I think this is important – is it really takes a second time. You see that sticky note, you make a little, not that impressed. You see it a second time. You're like, I saw this. I really should take this seriously. I should make a lot of this protein. I should remember. And that's what we're seeing. And even in some cases, seeing it a third time is even giving us a little bit of, dare I call it, a boost.

(LAUGTHER)

MARTIN: A boost. Those of us that are all boosted out here. Yes.

GRIFFIN: And the protein-based vaccines, which I touched on a little bit there with the Shingrix, with that vaccine that my buddy down at Baylor is working on, this is, you just cut to the end. You say, you know what? Let's just produce a whole bunch of that protein. Let's just show that to the immune system and see how we do.

So far, the studies have been pretty good. We'll get back to this. But it is challenging. It's not that easy to make these vaccines. It's not that easy to make them pure so that they can be safe. I think we're all anxiously waiting. I keep getting that question. When, doctor, will Novavax be a choice for me?

MARTIN: When it's safe is the question, or for the answer, right?

GRIFFIN: So that's exactly the answer. And that's what the FDA is waiting on. They want to see that this can be produced in quantity and that it can be safe, that there aren't any issues with impurities. Because you're going to be putting this into your body, right? And everyone is asking the question, is this safe? Well, the FDA wants to make sure the answer is a very resounding yes.

MARTIN: And that brings up a question that I have heard. And maybe this isn't the right time to ask it. And you can say I'll get to that later and that's fine. If a patient is immunocompromised, will vaccine efficacy be reduced or will put them at a greater risk?

GRIFFIN: So I think that's a great question. So if a person does not have a great immune system, then we're probably not going to be thinking about using one of these attenuated vaccines. Right? We don't want to potentially – and we do worry about this. If a person doesn't have a good immune system, what might be a weakened virus in a normal person could get them into trouble.

MARTIN: Yeah.

GRIFFIN: So here, we want to do the education without any virus that can replicate. So we start thinking more of the mRNA vaccines. We start thinking more of maybe the J&J-type vaccine where that's not going to be an issue. These are not able to keep replicating. Maybe the protein-based vaccines when they're available.

With the currently available vaccines, the only issue is really, how much of a response will I get? Depending on what the immune issue is, if the T cells are not working that well, you're just not going to get a great T cell response. If the B cells aren't working well or they're deficient for some reason, you're not going to get a great antibody response.

And that, at some point, might bring up passive vaccination, which let's throw that in right now. Some people can't make their own antibodies. And actually, just recently, the approval was for a product where you can actually pre-exposure. Before someone even gets exposed, you can passively give them antibodies so that they have that antibody shield.

(SOUNDBITE OF MUSIC)

MARTIN: We've been talking with Dr. Daniel Griffin, an internal medicine and infectious disease physician with additional expertise in virology, about vaccines for COVID-19. It's vital that we as healthcare professionals understand how the vaccines were developed and how they work so that we may address and alleviate the concerns not only of our patients, but also our colleagues — and even of those of us who have remained reluctant to be vaccinated.

In our next episode, Dr. Griffin sets out the facts concerning adverse events; vaccine efficacy against infection; supplements we've all heard about, such as ivermectin and zinc; the role healthcare professional play in supporting vaccination; and more. We hope you'll join us.

This is Deborah Martin for Elite Learning.

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