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Tricia Johnson:

It's Aspen Ideas to Go from the Aspen Institute. I'm Tricia Johnson. The race to develop a vaccine for COVID-19 is on, but ensuring it's safe and effective is easier said than done. A few vaccines show early promise, but questions remain about whether they're truly effective. Judith Aberg is Chief of Infectious Diseases for Mt. Sinai Health System. She said it's unwise to speed up the process with an emergency use authorization from the Food and Drug Administration.

Judith Aberg:

What we really want to know is that it truly is benefit, right? It's truly going to form antibodies, going to have a response and you're protected from getting COVID and that's going to take much longer.

Tricia Johnson:

Today, the science behind what it will take to develop effective protection from COVID-19. Aspen Ideas to Go brings you compelling conversations from the Aspen Institute. Today's discussion is from Aspen Ideas Health.

Tricia Johnson:

Antibodies, convalescent plasma, gene-based vaccines. You may have heard these terms on the evening news, but what do they mean? How might they help fight the battle against COVID-19? For the pharmaceutical companies developing vaccines, are they including a variety of ages and ethnicities in their tests? What if multiple vaccines arrive on the market at the same time? Will one be better than another? And perhaps most importantly, when will a safe and effective vaccine be available?

Tricia Johnson:

Judith Aberg is leading Mt. Sinai's COVID-19 treatment guidelines and the hospital's COVID clinical trial unit. She sits down with Florian Krammer. He's a professor of vaccinology at the Icon School of Medicine at Mt. Sinai. Ken Davis leads the conversation. He's President and CEO of Mt. Sinai Health Systems in New York City. Here's Davis.

Kenneth L. Davis:

Let's begin with a little history. I want people to have some appreciation of what the beginning of this pandemic was like at Mt. Sinai and then I'm going to transition to first talk about convalescent plasma and then we'll go to vaccines, which I know everybody is very interested in.

Kenneth L. Davis:

We were overwhelmed in March with how many patients came with COVID to Mt. Sinai. We increased the number of our beds by 50%. We were on the phone almost daily with our governor. We didn't know how bad it would be. We added staff everywhere. We put on beds everywhere. The worst day we had,

there were in our eight hospitals over 2000 COVID patients and on our worst day of mortality, we had 80 deaths. In the middle of that chaos, because we have a great strength in immunobiology, in virology, in creating vaccines, the two people that you're going to meet today were instrumental in thinking about new therapies and ways to measure antibodies.

Kenneth L. Davis:

I want to start with Judy and Florian and ask them this question. How, in the middle of that chaos, did you guys think about convalescent plasma? How did you decide to use it? And what have we learned from that convalescent plasma experience that was so nascent in March and then gets emergency approval months later? Judy, do you want to start?

Judith Aberg:

Absolutely, and thank you again for inviting me to be here. Convalescent plasma is something that has been used for decades, actually a century for different infectious diseases and I was involved with the NIH back in 2009 for the H1N1 influenza outbreak and at that time, we had collected convalescent plasma, then it was distributed and given throughout the country at those centers that were doing that study. When COVID hit, it was actually a similar group of individuals all thinking at the same time maybe convalescent plasma would be the right thing to do here. So there was a group of colleagues across the country that we all were emailing and chatting, how should we do this? A group started working on the protocols that we initially had hoped we would do, randomized control trials, so that we could really look at efficacy and the FDA released that emergency IND which allowed us then to go ahead and jump start and be able to offer that to individuals that were really ill.

Judith Aberg:

Mt. Sinai and then Methodist Hospital in Houston both gave the first doses or infusions, transfusions of convalescent plasma in March and I think it was a tremendous effort by many to do this. But Mt. Sinai really then, we went all in to develop this convalescent plasma, be able to offer it to the patients and we initially were able to transfuse 39 individuals. And looking at our preliminary results, we noticed that the oxygen status of our patients improved much more than the patients who had not yet been given it. Now that's the caveat. This was not a randomized trial so we just have match controls from that time point, but it suggested very promising therapies.

Kenneth L. Davis:

Right. And we knew that it had a biological plausibility from so many other diseases. But it was facilitated by being able to measure antibodies because you've got to take that plasma from people who are recovering. Florian, tell us about antibodies, how you develop the assay, why it was available so quickly? We were the first people to do it and it was essential. Give us a history of antibodies and how it fits in with the convalescent plasma story.

Florian Krammer:

Basically, we know that for many virus infections, antibodies are something that your body develops in order to fight a virus. Antibodies are very often able to neutralize virus, to basically get rid of them and that is certainly also the case for coronaviruses and that's well-known. So if you use plasma, plasma contains antibodies and if you have SARS coronavirus-2 infection, many cases, you make a lot of antibodies that are present in your plasma that can neutralize the virus. And so we started pretty early to develop tests to measure these types of antibodies. So the sequence of the virus was released on

January 10th and at that point, it became clear that we were dealing with [inaudible 00:07:33] coronavirus that looked very much like the SARS virus of 2003 and it probably behaves very similar.

Florian Krammer:

So for me, it was clear that this could be a problem and so we started very quickly to produce from the sequenced reagents to build tests that allow us to measure antibodies. And these tests were basically ready in February, so it took us approximately a month, one and a half months from knowing the sequence to establishing the assays. And so my laboratory is a research laboratory and so we transferred the assay then into our clinical laboratory and that allowed it to be used to measure antibody responses in patients and to collect convalescent plasma. And that was in March.

Kenneth L. Davis:

So how high? Is everybody, anybody who's had COVID get enough antibodies that we could have used them? Or how do we choose where to get that plasma from and how did your assay help us with that?

Florian Krammer:

The vast majority of individuals that get infected with SARS COVID-2 make antibodies.

Kenneth L. Davis:

High enough levels, though, for us to use as a therapeutic?

Florian Krammer:

Exactly. And that's the important point. You want to have plasma that has very high amounts of these antibodies and the assay allows you to look for that. It's a quantitative assay so you can measure how much antibody is there. And you can actually then select the donors based on how much antibody they have. And that's very important to select the right donors.

Kenneth L. Davis:

Right. So you're being a little modest because nobody else has a quantitative antibody assay. Yours was the first. Yours is the only quantitative antibody assay and there are a lot of other antibody assays that aren't nearly as specific or can't be used for this purpose. But at any rate, thanks to you, we're able to then measure the right amount, get the right plasmas from people who have the high levels of antibodies and Judy is able to infuse them. But Judy, how did you decide when in the course of illness to infuse, who to infuse and were you really convinced? How long did it take you to be convinced you were doing the right thing, giving it at the right time to the right people?

Judith Aberg:

Those are great questions. I think historically when you look and it makes sense that you want to transfuse people with convalescent plasma containing those antibodies before individuals make their own antibodies. Just a little bit more, I think, almost common sense, right? If somebody's already developed their own antibodies, probably giving more antibodies is not going to change their course. So when we initially started this program, we offered it to individuals that were within the first seven days of their hospitalization.

Judith Aberg:

So we wanted people early in their course. And I think as time went on, we did learn a little bit more about timing was really important. After more like 10-14 days, individuals really are starting to produce their own antibodies and there is less likely to see any benefit. The other is that now that we've transfused over 500 individuals and starting to look at that data, individuals who have less comorbidities, individuals that are younger, people that get transfused before they're on mechanical ventilation seem to do better. So our own impression without having those, like I said, randomized control trials is that the earlier in the course of the disease that you give them, it's most likely to be beneficial.

Kenneth L. Davis:

Okay. So let's move on to vaccines. Everybody wants to know when there's going to be a vaccine. But I don't have to tell you, but I want to tell everybody who's listening, the vast number of vaccines that get into clinical trials fail. I've seen a number that suggests that 94% of vaccines that enter clinical trials never make it into people, either for lack of efficacy or safety, yet we're all waiting with baited breath and the expectation that something's around the corner. Give us, each of you, your best thoughts on where do we really stand with vaccines today and when do you think we're going to have something that could be safe and effective? Judy, do you want to start?

Judith Aberg:

Sure. I'll be happy to start. There's several vaccine candidates out there that may be beneficial. When you think about vaccines I'm more simplistic. I think about vaccines that are either protein-based, which are the common ones that we give now like flu vaccine, where we're giving an attenuated flu or recombinant protein.

Kenneth L. Davis:

To help people so we don't get them in jargon, protein, you mean the virus, a piece of the virus.

Judith Aberg:

The virus, yeah, pieces of the virus.

Kenneth L. Davis:

You're vaccinating people with a piece of the virus.

Judith Aberg:

Right. Correct. And then you can use gene-based vaccines. And that's what has a lot of enthusiasm right now. By gene-based vaccines, what I mean is it has pieces of that genetic material of the spike protein. The spike protein on this virus is what enters the cell, so you want to make antibodies against that spike protein. So what they're doing is they take little what we call nucleic acids, the beginning of the building blocks for proteins, either DNA, something called messenger RNA or you can take another type of virus, make a little cut in it and insert some of the COVID protein in it, so sequences in it.

Judith Aberg:

These gene-based vaccines, what they do is that it goes into your cells and instructs your cells how to make spike protein, right? And when your body sees spike protein, then it starts making antibodies. So that's the thought behind the different types of vaccines that we're looking at. So right now, there are a

couple of vaccines that are in what we call the phase 3, where we're starting to look, we've got preliminary evidence on safety, a hint of maybe there's some efficacy. But I want to point out, when people talk about efficacy, they're just talking about that the body had made antibody responses. Nobody has shown that if you give vaccine to one person and placebo to another, that you actually have protected them yet.

Judith Aberg:

So we don't have true efficacy data. What we have is that we know some of these vaccines seem to produce a pretty prominent antibody response in the individual. We don't know how long they're going to last. We don't really know if they're protective. So I personally think it's unwise to use what we call this emergency use authorization where the FDA can do that because it may be of benefit, right? What we really want to know is that it truly is benefit, right?

Kenneth L. Davis:

Right.

Judith Aberg:

That it's truly going to form antibodies, going to have a response and you're protected from getting COVID. And that's going to take much longer. I really don't see that happening in 2020.

Kenneth L. Davis:

Okay, but let's summarize what you said because it's really important. There are different ways to make vaccines for the body to make a response. So we're using some new techniques here and some old techniques here. And what we've seen so far is that these vaccines get us antibody responses, which is good, but we don't know whether it's working yet to prevent disease because we haven't shown yet that these vaccines are significantly diminishing the likelihood that people are going to get COVID compared to people on placebo.

Kenneth L. Davis:

Let's step back a second because I want to ask Florian and Judy may want to chime in after Florian on this. No one's ever made a genetic vaccine yet. What Moderna is trying to do and what Pfizer is trying to do has so far, not been done. How likely is it and why are we starting with a completely new way? And do you think it's safer to do what some of the other companies are doing, which is taking just a piece of the virus and trying to inject that? How excited or how realistic is it to have started with a completely new technology? After all, Moderna doesn't have a single successful vaccine for all the work they've been doing. Florian.

Florian Krammer:

Yeah. That's certainly correct. There are no genetic vaccines that are on the market for humans and Moderna has not licensed a single vaccine. There's no Moderna vaccine on the market right now. However, these technologies are exciting because they're a new way, a very fast way to make vaccines and if we go back a little bit into pre-clinical experience, meaning testing in animals and there are very good animal models for SARS coronavirus-2, specifically in primates, in monkeys, we see that these vaccines work really well in protecting these monkeys.

Florian Krammer:

But I completely agree that it is a very good strategy to have many different candidates, some of them built on new platforms like RNA vaccines, like these genetic vaccines, but also, platforms that are based on more traditional approaches. And I think that's the safety net that we have. As we said, a lot of vaccine trials fail, but if you go with many different and very diverse approaches, it is very likely that one of these approaches or even more succeeds.

Florian Krammer:

And we already see that the more classical approaches of using bits and pieces of the virus as vaccine antigen are also working very well. There is data from Novavax, for example, that are really promising. They're not as far as, for example, Moderna in their evaluation in humans, but these are promising approaches, too, although they are more classic or you could even say old fashioned. So I think it's good to test these new approaches, but it's also good to have backup approaches that are based on classic technology.

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Kenneth L. Davis:

So, these big, phase 3 vaccine trials will enter 30,000 patients, 15,000 get placebo, 15,000 get vaccines. But we are concerned that there are various subgroups of population that differ from each other. So how do we know, for instance, when this vaccine might be approved, that'll get emergency approval, that 75-year-olds who have less of ability to mount immune response should be taking this vaccine? How do we know, for instance, that enough minority groups have been included so that the group that we know is getting most affected is also going to have a good response? Tell us what you know about the inclusion of enough groups so that we can make the right decisions about subgroups of patients. Judy, what do you think?

Judith Aberg:

Right now, I have to say that Moderna and Pfizer both have been in the news, talking about the challenges of really recruiting minority populations. As we know, blacks and Hispanics were two and a half times more likely to suffer consequences from COVID, yet this is a population that we have challenges enrolling. So I think we need to do a better job as a country educating and really going out to the communities that are most at need. And I think we failed a little bit on that overall, as far as our recruitment strategies. At Sinai, we're taking much more action to go out to the communities, having focus groups, trying to engage the community that's most at-risk. That includes minorities.

Judith Aberg:

It also includes older patients, as well as patients with comorbidities. I think when you look at who died from COVID, it was more so older individuals with multiple comorbidities. Typically, those are not the individuals that are coming to be recruited into ... normally, you think about healthy volunteer studies.

So doing those types of targeting recruitment strategy is going to be very important for this. Both Moderna and Pfizer, the minority enrollment has been less than 15% overall and that's just not acceptable.

Judith Aberg:

And more so to your question, Ken, how will we know with older individuals that it works? So some of the trials are actually stratifying by age. Johnson & Johnson, the Janssen vaccine which is what we call an adenovirus type, where you take a common respiratory virus, adenovirus, and you insert part of the COVID sequences in there. They actually are stratifying at entry for individuals 18-60 and then the next group being 60 and older so that they can actually have the number that they need to evaluate that. And their sample size is 60,000 compared to Moderna and Pfizer that's 30,000.

Judith Aberg:

So I think that's what we really need to do is we need to stratify up front and then again, afterward they can look at subpopulations, as well, to see what the response is.

Kenneth L. Davis:

Right. So we're going to have to look for that data when it comes out and hopefully, it will be readily available so every subgroup of patients is going to be able to know is this going to work for me? Help us with ... both of you, help is with this question. There may very well be within six or eight months more than one vaccine. How will you choose and could you take a second vaccine if you've already gotten the first vaccine? What are your thoughts? Florian, do you want to start?

Florian Krammer:

Yes. Sure. Actually, I think it would be good to have more than one vaccine, specifically one of the first vaccines that gets licensed is one that is based on these more modern technologies. We know already from phase 1 and 2 trials that RNA, the genetic vaccines, but also, the adenovirus vaccines cause more [inaudible 00:24:29]. And what I mean by [inaudible 00:24:30] is that you get a sore arm, that you might get a slightly higher temperature that might feel flu-like symptoms from the vaccine. And if you have one of those vaccines that are already tested in adults, it might do that at a much higher rate in children. So it might be good to have at least a second vaccine candidate or a second vaccine on the market that is based on classic technologies that does not do that. So I think that's a really important point.

Florian Krammer:

The question about combining vaccines is something that fully needs to be studied in the future. We have seen for some experimental influenza vaccines that actually vaccine A followed by vaccine B can sometimes give you a superior immune response and superior protection, but it could also be the other way around. So these mix-and-match strategies need to be tested and it's clear that this will happen once people get vaccinated.

Kenneth L. Davis:

So let's assume just hypothetically that we get vaccine 1 which is said to be 55% effective, which is they say we have to be more than 50% effective to get approved, but we all want a vaccine, so 55 gets approved. And the second one is 60%. So we don't have any single vaccine that's going to do 95 or 100%. Do you start to tell people you'd better get both of them?

Judith Aberg:

Obviously, as Florian was saying, there's insufficient knowledge right now about combining vaccines. One thing that sometimes we do with vaccines is you give a vaccine and then you test a couple of months later to see if you've had an immune response. So you could see if they, in fact, did have an antibody response and then in those that are the nonresponders, consider giving a second vaccine. And we do that for hepatitis B now.

Judith Aberg:

The other is that we do have some knowledge of giving different types of vaccine. We do this for pneumococcus, which is a type of bacteria that commonly causes lung infections or in kids, ear infections. And we have one type of vaccine that we were giving and then another one got developed. So now we do give them in a sequence, eight weeks apart. So we do have some experience with vaccines, giving one and then almost doing a booster or covering different aspects of whatever we're trying to build immunity against.

Kenneth L. Davis:

How concerned are you that there will be many people who are afraid of vaccines or who won't trust this vaccine and if we get a low turnout of people being vaccinated, what do you think are the consequences of that for the population? Judy, do you want to start, then Florian?

Judith Aberg:

Sure. No, I think there are going to be individuals who are concerned about getting vaccine. We already have that with influenza itself. Only about 50% of whites in America get vaccinated and it's much lower in minority groups, even as low as 20, 30%. So we already know that we're going to have individuals that are reluctant to take the vaccine for many different reasons. And this is where I also think that we need to keep developing these other types of therapies for prophylaxis, for prevention, so monoclonal antibodies, where we make laboratory-based antibodies just like your bloodstream does that we can give to individuals to prevent them from getting COVID.

Judith Aberg:

The convalescent plasma program at Mt. Sinai, what we're doing is we have a partnership with Emergent Biosolutions who is making a product called hyperimmune globulin, which is purified antibodies from the plasma that then doesn't have to be blood type-specific. Individuals that don't want to be vaccinated or don't respond to a vaccine could be given this hyperimmune globulin to prevent getting COVID, as well.

Judith Aberg:

But I think it really comes back to, Ken, really the wearing of the masks, good hand hygiene, the social distancing. That's still going to be very important over the course of this next year.

Kenneth L. Davis:

Florian, what are your concerns about people who don't get vaccinated and what this means for ultimately get herd immunity?

Florian Krammer:

Well there's two groups of people that might not get vaccinated. The [inaudible 00:29:14] antivax individuals that just refuse to get vaccinated and they don't think they are the problem. In this case, I think what we need to make sure is that people who are skeptical because these are [inaudible 00:29:29] vaccines and they have been developed very quickly, they need to be convinced that these vaccines are safe and effective. And I think to make sure that people understand that and people trust the vaccines, we need a very transparent process of getting these vaccines licensed and everything needs to be done by the book. I think that's the most important message to people who might be skeptical about vaccines.

Florian Krammer:

We need to do a very thorough evaluation of efficacy of these vaccines and of safety. And I think if that is done and if it's done in a transparent way and if it's communicated well, I think then a lot of people will actually trust the vaccines and will get vaccinated.

Kenneth L. Davis:

Okay. So I've been looking at some questions and let me give you some of the more provocative ones that I think will give us some very important information. Some people are pointing out that they've heard that this virus mutates. Will that affect our vaccines? Should we be worried about mutations? Are the mutations like the flu mutations which cause us to need vaccines, new ones every year? So tell us about COVID mutations, COVID-19 mutations and whether it's going to impact subsequent vaccine policy. Go ahead Florian.

Florian Krammer:

Yeah. I think I can take that question. We have to be careful. We shouldn't look too much at flu. Flu is a very different beast. We know that all RNA viruses mutate. That doesn't mean that they change in a way that they would require a new vaccine. Measles, for example, is an RNA virus that mutates and we are still using vaccine [inaudible 00:31:17] from the 1960s and they work very well. And we also experience from human coronaviruses, even the population that cause common colds and those viruses don't change much.

Florian Krammer:

In addition to that evidence, we also know that coronaviruses compared to other RNA viruses have the [inaudible 00:31:39] activity. So they actually mutate less. So there's many factors that come together that suggest that this virus will not readily mutate and that we will not need a new vaccine every year like for influenza.

Kenneth L. Davis:

Okay. So here's another important question. We've heard a lot about comorbidities and people who are immunosuppressed. Given the rapidity with which this vaccine is being developed, they're sure that there won't be a lot of people who might be on immunosuppressants or have important comorbidities. So should people with comorbidities and immunosuppressants get this vaccine? Judy, what do you think? What should we tell those people?

Judith Aberg:

In the clinical trials now, again some of the companies are, in fact, limiting the amount of comorbidities or the type of comorbidities, whereas other companies are more wide open and they, in fact, are, they're including individuals who have HIV, hepatitis B, hepatitis C and there on immunosuppressive agents. So I'm hoping that more companies will follow suit with the ones that are having a broader availability because you're absolutely right. At the end of the day, it is the people that are most at risk that you would want to offer vaccine to.

Kenneth L. Davis:

Right. Assume that we don't have that kind of data early on. What would you tell someone who's on immunosuppressants? Should they get the vaccine?

Judith Aberg:

I would do it. I absolutely would encourage doing it. I've been taking care of people with HIV for a long time and we had the same issue with vaccines. They weren't tried in the HIV population, so they were excluded from the trials. But we've learned and we've given in and they worked. So I think that we should absolutely do that and again, we could test the antibodies to see if they have a durable response.

Kenneth L. Davis:

Okay. Here's a very interesting question. Why is the number of new COVID patients so low in New York now and in the neighboring states? Could it possibly be that this area already has herd immunity?

Florian, you want to take it?

Florian Krammer:

Yeah. I can take that question. We have to be careful when you talk about herd immunity. It's not a black-and-white thing. Of course, if 20% or 25% of the population already have some type of immune response to the virus, which is the case in New York City, of course the reproductive number of the virus or how it spreads is a little bit lower than if everybody would be completely naïve. But we also have to keep in mind that behavior in New York City changed a lot. People are sticking to wearing masks and that's really important. We don't have indoor dining. A lot of activities are carried out outside, where the risk of getting infected is much lower.

Florian Krammer:

So I think right now, it has to do a lot with behavior. And there is, of course, the risk that we'll have another wave in New York City. This is a possibility and we hope that with the behavior that people here show right now that we can suppress a second wave. But at this point, I would not bank on herd immunity in most areas on this planet.

Kenneth L. Davis:

So you would suggest that the reason New York is so low right now is because people have been using masks and are socially distant and it has nothing to do with how many people have already been infected.

Florian Krammer:

I think that's the main reason why we have very few cases right now. There is an influence. If you have 20% immune or individuals that have antibodies, of course that lowers the reproductive number in how fast the virus can spread. But I don't think that's the main driving factor right now.

Kenneth L. Davis:

Okay. Here's a question for Judy. Are there plans to pool data from the small randomized control trials for plasma treatment and have the randomized control trials been hampered by the emergency use authorization? What do you think?

Judith Aberg:

There is a group that is trying to pull the data together. The caution with that is the eligibility criteria differs. The testing that was done differs. So pooling data like that may or may not really give us the answers that we want. I do think the emergency use authorization, again because it's wide open. It says if there's any chance somebody may benefit, it's okay to give. And I think that has diluted down potentially the benefit that we may be seeing, as people have been transfusing convalescent plasma much later individuals.

Judith Aberg:

I will say that there was just recently a study. It's in pre-print right now from India, where they did a randomized control trial, although it's open-label, so you know who got plasma and who was given other standard of care. It was interesting. They did not see a benefit. The difference in mortality between those that received convalescent plasma versus those that didn't, they didn't see any difference.

Judith Aberg:

But I'm going to say the caveat being that over 80% of the people receiving plasma already had antibodies, going back to my point that if we really want to be able to show that convalescent plasma or any type of antibody therapy is beneficial, it needs to be given before an individual makes their own antibodies.

Kenneth L. Davis:

Good. So since COVID-19 is one of many coronaviruses, do you think there's a potential for a universal coronavirus vaccine at some point in the future?

Florian Krammer:

I'll answer with cautious yes. We are doing something similar for influenza right now. There are several universal influenza virus vaccines in clinical trials and so far, there is very promising data. So if you can do that for influenza viruses which are very diverse, it might also be possible to do that for coronaviruses. Getting to that point will probably take a lot of time and it's probably not going to be easy to show that you have universal protection, but from the basis of how antibodies recognize diverse coronaviruses and from all the knowledge that we have gathered in the last couple of months, it might be possible to design a vaccine that broadly protects against zoonotic coronaviruses, meaning coronaviruses that spill over from animals into humans.

Kenneth L. Davis:

Okay. So here's a question that I think you're going to find easy to answer, but I think we should address it. If someone in your family or a close associate has already had COVID, you know you're exposed to it so you still get the vaccine.

Judith Aberg:

I think that if you yourself do not have antibodies, I absolutely think you should be vaccinated. I think it's less known if you have your own antibodies, I don't think at this point I would promote doing vaccines, although I will tell you again, in the trials we are not checking antibodies first because when you think about when you're going to roll this out to millions if not billions of people globally, you're not going to do any testing before you would give it. So you would want to just give vaccines to everybody without knowing what the antibody status was.

Judith Aberg:

If I never had COVID, but somebody in my family did and I was exposed and I know as a healthcare worker I've been exposed a lot, I would still get the vaccine.

Kenneth L. Davis:

Right. But if you know you have antibodies, then you need to get the vaccine?

Judith Aberg:

I think no, probably not. It would depend, too, what is your antibody response? What we still don't know is how durable that response is. Most individuals, as Florian mentioned, had very high antibody levels that we checked. There are some individuals that despite having COVID, don't have antibodies and those individuals that would or if they were low or if they wane over time, right? And that's all the uncertainty that we have at this point.

Kenneth L. Davis:

Right. But can we get people to feel a little bit more relieved around the question of how long will the vaccine work? There's some people who talk about it may only last for a month or two. So what do you think we should tell them Florian?

Florian Krammer:

[crosstalk 00:41:05]. That is relatively easy to answer. There are no vaccines that only work for two months. That's just not how the immune system works. And it might be that you need a booster vaccination of let's say three years or four years. That could be possible if the antibody response wanes, but to be honest, that's not really a disaster. There are vaccines that are given in those intervals. So if, for example, you need to get a tetanus shot every now and then to boost up your antibody titers and if you end up with a scenario like that where you need to get a booster dose every few years, that would not be a big problem. But it's very unlikely that a vaccine would not induce an antibody response or an immune response that would not at least be durable for a number of years.

Florian Krammer:

And we see now more and more and know that there were media reports initially that the antibody responses of the natural infections go away very quickly. There is now a lot of very solid data that says that this is a very normal immune response to respiratory virus. So what we see is right after infection,

you get a lot of antibodies that stays very high for a few months and then it goes down a little bit and then it stabilizes over time. So it looks very normal and there is no reason to believe that a vaccine-induced antibody response would look different.

Kenneth L. Davis:

Are you concerned that political pressure to the FDA will result in emergency authorization before we really have convincing proof of safety and efficacy? Both of you quickly.

Judith Aberg:

I've been very concerned about that. I think it will be a mistake if we release a vaccine before we have more data. I think an emergency use authorization, as I mentioned before, just says that there may be benefit, but there may not. And I think the ramifications to the public will be great if we release a vaccine before we actually know it's safe and that it works because the worst thing you want to do is vaccinate, again, millions of people and then it doesn't work because then there's just additional mistrust and distrust in what we're doing and it'll affect future vaccine efforts. So I think it's really important we follow this through.

Kenneth L. Davis:

Florian, any last thoughts on that question?

Florian Krammer:

I'm worried, too. I know that there is a lot of very good scientists at the FDA and I trust them. We have also seen that vaccine produces pushback on that, but I can only echo what Judy said. I think it's very important to evaluate these vaccines very thoroughly and once we know they are effective and safe, then they should be licensed, not before then.

Kenneth L. Davis:

All right. Well thank you both for a very informative 45 minutes.

Tricia Johnson:

Judith Aberg is a nationally renowned researcher in the field of HIV and AIDS. She was instrumental in developing one of the country's first convalescent plasma programs to treat COVID-19. Florian Krammer is the principal investigator of a collaborative influenza vaccine innovation center at Mt. Sinai. Ken Davis is President and CEO of Mt. Sinai Health Systems in New York City. He's a trustee of the Aspen Institute. Their conversation was held September 11th. It was part of the 2020 series from Aspen Ideas Health.

Tricia Johnson:

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