

Childs, Richard 2020

Dr. Richard Childs Oral History 2020

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"Fostamatinib Inhibits Neutrophils Extracellular Traps Induced by COVID-19 Patient Plasma: A Potential Therapeutic," *J Infect Dis*, 2021 Mar 29; 223(6): 981-984. doi: 10.1093/infdis/jiaa789.

"Opposing activities of IFITM proteins in SARS-CoV-2 infection," *EMBO J*, 2021 Feb 1; 40(3): e106501. doi: 10.15252/embj.2020106501. Epub 2020 Dec 21.

Dr. Richard Childs

Behind the Mask

October 20-21, 2020

Barr: I'm Gabrielle Barr of the Office of NIH History and Stetten Museum. Today we have the pleasure of talking to Dr. Richard Childs for our Behind the Mask Project. Dr. Childs is the Clinical Director of the National Heart, Lung, and Blood Institute's (NHLBI) Division of Intramural Research. Thank you very much for talking to us today. Our first question is, so you've been involved in a lot of different COVID-19 initiatives, one of them even before the virus reached American shores. Can you shed light on your experience when you were administering remdesivir to the passengers of the Diamond Princess cruise ship in Japan?

Childs: I am an officer in the Commission Corps of the United States Public Health Service and I had the honor of leading a team of 25 officers that were deployed to Yokohama, Japan, early during the COVID-19 outbreak just when it was starting to come outside of the borders of China. Our team was deployed to assist in the disembarking of numerous American passengers on that ship, including some passengers that were infected with COVID-19. That was my first experience with dealing directly with COVID-19. We were deployed very quickly, within less than 48 hours from the time that I was notified about the mission to actually having boots on the ground in Japan.

Barr: That's quick. How did you prepare so fast?

Childs: The Assistant Secretary of Health had prepared all the officers in the Commission Corps for the possibility that we may need to be rapidly deployed both nationally and internationally to respond to this health crisis. We were all in the mode that we could literally be out the door at any moment. Our uniform changed from our typical operational uniform where we wear khakis or a more dressy service dress blue to our, what we call, our ODUs or operational dress uniform where we wear boots. It looks more like what you would typically see with officers in DOD that are involved in military engagement. In this case, we wear a uniform that prepares us for deployments in the field. We were all wearing those uniforms knowing that we could be called upon at any moment, so we were in the mindset that we could be out the door and be needed. That was the case and in a very short amount of time I found myself in Japan with my fellow officers.

We were engaged in the initial mission of the disembarking and then that mission morphed very quickly into the mission to try to identify how many Americans were infected. Then we had to figure out where they were because on the ship manifest—we realized that there were numerous Americans that weren't on the ship anymore that had been hospitalized throughout Japan. In the end we found out there were over 60 Americans that were hospitalized in 25 different hospitals. Fifty-two of them had confirmed COVID-19. The number of them was a high percentage.

Overall, on that ship it looked like somewhere between 15 to 20 percent of the folks that were on board got infected. We identified where they were. We identified that there were a number that were critically ill. Thirteen ultimately were identified to require intensive care support. Ten of them were on ventilators or they were on extracorporeal membrane oxygenation or ECMO, sort of a heart-lung bypass. The most critically ill patients get that kind of treatment.

Then our mission changed to getting off the boat to support the Americans that were hospitalized throughout Japan. We served as their advocates. The Japanese provided outstanding care, but at the time there was nothing that was known to actively alter the course of the viral infection. We based on some preclinical data showing that remdesivir was active in the laboratory against the virus. So, we went on the hunch that of all the drugs that were what we call "shovel ready", meaning that they're available for use in patients, either as an FDA approved drug for another use or as an investigational agent that is available because it's going to be studied in a clinical trial or has recently been studied from desperation in Ebola, we contacted the company and said, "We've got these really sick Americans. Would it be possible for us to get a compassionate use authorization to use this medication in those patients?" We were fortunate to have established very, very important critical contacts with leadership in Gilead Sciences, that makes the drug, as well as with the Japanese Ministry of Health. [Robert] Walker, from BARDA [Biomedical Advanced Research and Development Authority], assisted us in making all these critical contacts that were needed to get authorization for the drug to be shipped to Japan.

Barr: How quickly did it happen?

Childs: It happened really quickly. It was within three days the first patient was being treated with remdesivir from when the ideas came along that maybe we can get this drug, to the drug being available. You know it was a lot of work by everybody that was on the team. Our team was reduced from the initial group to a smaller group of eight officers that were focused on the remdesivir, which we called our team. We were burning midnight oil just trying to get everything lined up from the consent form to the caring physician at the hospital buying in on the use of the drug for compassionate use. Each hospital had to have their own IRB [Internal Review Board] approve the drug for that use. I mean literally, if you were to line up all the obstacles to prevent us from doing this, it would seem impossible that we could have done what we ultimately achieved. I think probably if someone had written down everything at one time that we needed to do, I would have said this is not gonna happen. But we just said, "We can do that, we can do that." We worked our way through it.

The next thing you know the patients are getting infused with the medication and it was a tremendous effort on numerous folks' part and at the time we didn't know if the drug even worked. There was a real possibility that we would have gone through this tremendous exercise for nothing if the randomized study had shown that the drug wasn't active. There are other drugs that seem to have activity in the lab, in vitro, that now clinically may prove to be an active hydroxychloroquine. There's some data now in the context of active infection that that drug doesn't do anything compared to placebo in terms of improving outcome. That very well could have been the case for remdesivir, but we had nothing else. We had high hopes. We had looked at the in vitro data very carefully and it was an educated guess and we said, "We're going to be able to pick one thing; let's go for that one."

So, we ultimately treated all these patients and everybody that was critically ill got better. What role remdesivir played in this we don't know. I can tell you it was a couple months later when the randomized trial came out and showed that the drug significantly shortened the time till people recovered and we were really excited about that because we said maybe we did play a role in helping our sick citizens to get better. That was a very exciting day for everybody that was on that team. You know we picked the right drug.

Barr: Were you and your team monitoring the patients in the hospitals or were you more afar?

Childs: We had a command center that was set up in two different places. Initially we were in the embassy and then in a hotel and then we transitioned completely over to a command center in a hotel. I would go out and I would visit hospitals and there were some circumstances where family members requested that I came out or the attending physician requested that I would come out. So I made a number of those visits where I could actually see the patients. But the Japanese were the ones that were delivering the care. They did an absolutely outstanding stellar job. I think that we were really fortunate to have the highly qualified and motivated healthcare providers that took care of our citizens there. It seemed like they were energized by the fact that they were taking care of Americans that were sick and needed them. It was really an incredibly rewarding experience to be part of that team that was involved in caring for those folks.

Barr: I understand that you were in Liberia with Ebola. How did this situation differ for you? Both are very infectious diseases. Very different experiences or what were the similarities?

Childs: There were differences and similarities. I mean similarities in the sense that both were emerging infectious pathogens that were potentially life-threatening. Ebola was sort of an order of magnitude of greater risk because with Ebola, at the time when I went over to West Africa, most people that got infected died. There were some fatalities associated with disease, that were 100% at the time. We had heard that anyone that got a needle stick got infected through a needle stick. That the mortality was 100%. That was very scary because when you're taking care of these patients and the teams that I was involved with, I was a Chief Medical Officer, so I was involved in direct patient care. I would go back and see patients every day. You had to be very careful because if somebody left a needle on the field and you stuck yourself with it, that could be game over. So the level of caution with both viruses in terms of PPE [personal protective equipment] was great, but with Ebola you realize that if you made a mistake, you know that you could potentially put yourself at extreme risk or someone else at extreme risk if you weren't following the proper safety procedures.

Everything was a little bit slower with Ebola. The PPE was much more extensive. We had an expression that there were no emergencies with Ebola. If you had to do something quick with Ebola, usually you weren't going to help the patient and you were just going to put yourself at risk. You know we were not doing CPR on those patients because of the concern that you could breach the PPE and get infected during CPR. Knowing that CPR would unlikely be successful for the patient anyway, I think the fear factor with Ebola initially was higher.

Now once we had been trained, we were all very confident that the PPE was going to protect us and that we were not going to get infected as long as we followed the protocols. But everybody watched everyone, and everybody served as a safety officer because if somebody was breaching protocol, they could put your life at risk or put the whole team's life at risk. If one officer got infected, you could potentially lose the whole team. So, there was extreme caution there. With COVID-19, knowing that it was predominantly spread by droplets and that a significant proportion of the patients that were getting infected were surviving, and even some evidence that people could be asymptomatic even early on in that process, we had no idea that the percentage could be so high of folks that were infected and asymptomatic. You know that concern about yourself personally was less than with Ebola because nobody wanted to get infected. Everybody followed the PPE matrix to protect themselves and it was less extensive than with Ebola, where you could not have any bare skin even being shown. You knew that if something happened and you did have a breach and if you got infected, you were unlikely to die from it—in contrast to Ebola.

END OF FIRST INTERVIEW

Barr: Today is October 21, 2020 and I have the pleasure of talking to Dr. Childs. Thank you for coming back for part two. We spoke earlier about your experiences in Japan with remdesivir. But you've also been doing a lot at NIH and currently you are involved in a study that is sponsored by the National Heart, Lung, and Blood Institute (NHLBI). It's looking at how to evaluate the safety of fostamatinib in comparison to a placebo and how to assess disease progression in COVID-19 patients. First, can you tell us a little bit about what fostamatinib is and why it is crucial in treating and preventing conditions in COVID-19 patients?

Childs: Fostamatinib is an FDA approved drug used for the treatment of chronic ITP, idiopathic thrombocytopenic purpura. It's an autoimmune disease where you destroy your own platelets, you have a low platelet count, and you're at risk for bleeding. The drug was approved a couple of years ago for patients that were either refractory to conventional therapies or were receiving high dose steroids that were debilitating. It is something that is called a spleen tyrosine kinase (Syk) inhibitor. It inhibits a particular pathway that cells use to destroy targets that are coated with antibodies and the cells that then come and bind those antibodies are no longer able to destroy the platelets because the pathway that they use which is kinase is inhibited by the drug.

We got very interested in this drug because there was some work being done to show that it not only inhibited that particular pathway but that that pathway in COVID-19 was utilized by the immune system to try to eradicate the virus in patients that had been infected with Sars-CoV-2. That, when their bodies were making antibodies against the virus, would lead to the immune cells becoming overactive binding the antibodies and in the process of being overactive they would release chemicals, cytokines or very sticky material called "NETs", or Neutrophil Extracellular Traps. This substance could clog up blood vessels, could activate platelets and make platelets form a platelet clot. These are all now believed to be part of the pathology that occurs in patients with COVID-19 that leads to them becoming very sick, that makes their pneumonia worse, that may make them go into very high concentrations of oxygen or even go onto a ventilator. Inhibiting that netosis or that part of the overactive immune system may be very important in preventing people from dying from COVID-19 that have very severe disease.

We think that that window, when the immune system is kicking in, is a double-edged sword: very important to eradicate the virus, but the immune cells that are recognizing those antibodies are in some cases a little bit out of control and they end up doing more damage than good. We're very excited about this drug because it is the only Syk kinase inhibitor that's currently FDA approved. We're using it as a shovel-ready, off-the-shelf drug that already is known to be safe in the context outside of COVID-19. It has a known and very good safety profile and it's easy to produce and it's readily available so, if the trial shows that this has a signal for being not only safe but being effective, it could be given to a large number of patients that get infected with the disease. Now this study is a phase 2 study, so it's intended to first use the drug in the population for the first time ever.

This study is now giving COVID patients this medication. We know they have never received it before. We need to first make sure that we finally know it's safe for people that have chronic ITP. That doesn't necessarily mean it's going to be safe in people that have COVID-19 so we need to establish that first. The second part of the study is to look to see, compared to placebo, not only what is its safety profile, but are we seeing a signal that it actually may be helping patients that are infected? The way we can pick up that signal clinically is by looking and seeing how patients do when they started the medication. Are they less likely compared to placebo patients to end up on a ventilator? Are they more likely to recover quicker than patients that are receiving placebo? Do they come off oxygen quicker? Do they get out of the hospital quicker? Is their survival better? It's not a big study to be able to say with confidence, even if there is a beneficial effect, that that effect is absolutely related to the drug. You need to have studies that have hundreds and hundreds of patients to be able to say things definitively regarding survival now.

Barr: How many people are a part of this study?

Childs: This study has 60 people, so 30 will get the drug and 30 will get placebo. It's a one-to-one randomization. The expectation is we won't be able to say anything about survival because we don't expect there to be a high enough mortality in the placebo group and such a massive reduction in the drug group as to be able to achieve statistical confidence. Even if we saw a reduction that was definitively related to the medication, we'd like to see trends. That's where if something looks like it's occurring less often, like death or people look like they're getting out of the hospital quicker, those kinds of trends, even though we may not be able to say with high levels of statistical confidence that it's related to the drug, we can say it might be, it looks like it could be. That would lead to the bigger, more definitive study that we would call a phase 3 study that would have potentially 500 to 1000 patients that would be able to say yes, this drug does improve the odds of survival, this drug does prevent people from going on ventilators, that are on oxygen when they started. Those are some of the questions that we're hoping that a phase 3 study will [answer]. We'll definitely be able to answer.

We're very excited about the drug because it inhibits this newly recognized pathway that plays a major role in making folks critically ill, and probably in contributing to their mortality. We know from autopsies on COVID-19 patients that their lungs are full of these nets. We know in the laboratory that you can take COVID plasma that makes nets and you can put a little bit of this drug mixed in with that plasma at a concentration that people would get in their blood streams if they took a pill, and we can see that it inhibits the formation of those nets. So, we have some preclinical data that is very exciting that makes us think that this really could interrupt this very dangerous pathway that makes people critically ill and contributes to their demise.

Barr: Have you ever seen any other disease that has done something like this or is COVID-19 very unique in this way?

Childs: We learned a lot about COVID-19 very quickly. There's a lot of things that COVID-19 does, that SARS-Cov-2 does, that are similar to other viruses and there are some parts that are quite unique. That may have to do with just unique features of the virus and specifically where that virus binds. Expression of the ACE-2 receptor is on certain tissues and those tissues are targeted by the virus and can be damaged selectively. So those parts are unique. Take the NETs, for instance, how unique that is to this particular virus versus other viruses? That part is just being looked into now because NETs were really not appreciated to be factors contributing to mortality and morbidity in many diseases until recently. So, I think you will see a lot of research aimed at trying to define: Does this problem of clogged up blood vessels and activated platelets occur in other viral illnesses or bacterial disease processes that are associated with people critically ill or dying?

You'll see a lot of research in the next five to ten years on the role that NETs play in common viral illnesses. Maybe, you know, illnesses like CMV [cytomegalovirus], RSV [respiratory syncytial virus], and with bacterial infections and sepsis, there are really many reasons to suspect that NETs are not limited to this virus, but that this may be sort of a common pathway that contributes to patients becoming sick with so many disease types. I think that COVID-19 will be the place where this medication is tested, but I would not be surprised if in the future we see there are many diseases where inhibiting this kinase is beneficial to treating other diseases.

Barr: What point are you and your team at with conducting this study?

Childs: I have to tell you the study got off the ground at lightning speed. I've been here for 25 years and a trial like this, if someone told me they were designing it, I would say, you know, you're probably looking at 12 to 16 months to get the study up and running. It required a placebo arm. It required FDA oversight. It required rigorous scientific review including IRB [Internal Review Board] review and a data safety monitoring board, setting up all kinds of infrastructure, case report forms, and things like that. This study went from the first discussions to IRB approval in three months. I mean, just amazingly quick. Everybody came together as a group knowing what was at stake here and looking at the numbers and seeing that COVID-19, unfortunately, did not do what we were hoping it would do. Back in March we talked about bending the curve and hopefully going to zero and then worrying about COVID-19 coming back in the winter. We never even got a chance to worry about it coming back in the winter because it never went away. It stayed, unfortunately, at very high numbers and continues at very high numbers. We are really, really hoping that these vaccines are going to be effective because, if they're not effective, we're going to be dealing with this for a very, very long time. When we started seeing the numbers in June, July, and August, it became very clear that we need to continue these lines of therapeutics because any kind of magical thinking that this was just going to disappear was gone, looking at the numbers.

Barr: What challenges have you had with the study so far and has there been anything that has also surprised you, that has gone better than you could never have expected?

Childs: The challenges are just administrative and logistical hurdles. The part that surprised me is that everybody that potentially represented an area of review or a potential bottleneck came together and realized the importance of trying to get the study up and running as quickly as possible. So, for instance, the scientific review folks that typically take two weeks to do the review, reviewed it in about four hours. It was great seeing people, knowing what was on the line and coming together, doing things on weekends. Scientific review occurred at multiple levels on a Friday evening. That was incredible. The IRB typically would not meet to review a protocol until it had been reviewed by the FDA, met in advance of the FDA which helped grease the wheels for changes to the study the FDA wanted when they reviewed it. They were agreeable to make those changes and still review the study just a few days later as opposed to saying start over and we'll look at it again in a month. Everybody came together and that part was incredibly rewarding—to see the NIH teams represent the National Heart, Lung, and Blood Institute in a very close collaboration with members of the Clinical Center. The Critical Care Medicine Department and members of NIAID have worked together very closely to get the study up and running, bringing patients into these types of clinical [studies is] difficult as there are issues related to trust, and there is concern as to the type of research you're doing and the risks that may be imposed with that research. It's important to effectively communicate why you're studying this drug and what your hopes are for the drug and what the risks are associated with the drug.

There was a lot of thought put into how we would do messaging to the community and recruitment. Working with our partners at Inova Fairfax [Hospital], has been really just an amazingly rewarding experience. Inova Fairfax has a large number of COVID-19 patients and they are collaborators. So, this has been conducted at NIH and Inova Fairfax and they've put their heart into the study as well and they have been enrolling patients very quickly. We've only been really officially sort of up and running for about the last two weeks and we have now, I believe, just enrolled our eighth patient on the study. The goal is to get up to 60 but sometimes, when you are just starting, things go a little bit slow until the word gets out. That our colleagues and collaborators have embraced the trial, put their heart into enrolling subjects on the study. The hope is that we'll be helpful and we'll be safe and that we'll get the answers quickly. If it does do what we hope it does, then the subsequent trial, the phase 3 study, will roll out quicker and we may very well end up establishing a new standard of care for the disease.

Barr: That's really great. Have you noticed any side effects to the drug at all or it's too soon to say?

Childs: Too soon to say. We are blinded as to who is getting the placebo and who is getting the medication. We have a couple of people that are unblinded that are looking at the data. As subjects enroll, they'll look after the first 10 patients are enrolled and they'll be able to compare between the two groups, placebo versus the drug group, to make sure there's no unexpected side effects in the fostamatinib arm group. The safety profile of the drug is well established so we know that it can cause a little, a small percentage of people, diarrhea, some elevation in liver enzymes, some reduction in their white cells or high blood pressure. Those kind of things we haven't seen so far from the reports that have been coming in.

Barr: So, what has been your role in this study?

Childs: My role has been, basically, I was contacted by the company because they were interested in exploring the medication and they were looking for ways to have the drug explored in the extramural community. They sent me a slide deck and I looked at it. I got very interested in the drug. We've wanted to utilize the Clinical Center and the Intramural Program for cutting-edge first-in-human research because the Clinical Center is ideally suited to do that. I'd seen many, many drugs that people were interested in. When I saw this drug and I saw the fact that it was inhibiting these NETs and that it was really unique compared to the other therapeutics that maybe were aimed at inhibiting the virus or killing the virus or just turning the whole immune system off, that's what really got me interested. Once I got that information, I said we could do this study here. Then I reached out to my colleagues that had expertise in the management of critically ill patients and infectious diseases. That's where we connected with the Critical Care Medicine Department and Dr. Strich who is the PI on the study. He saw the data and he was even more excited than me. That's where we said, okay, let's create a partnership here and let's look at the possibility of writing a trial. We also had a relationship established with Fairfax [Inova Hospital] to get samples from patients that had COVID-19 over to the NIH, so our investigators and the NHLBI could do research in the lab. We had had multiple calls that had been going on for a while. When Dr. Strich started writing the trial, I talked to Dr. [Steven] Nathan over at Inova and I said, "We're writing this trial. Would you possibly be interested in being a second site?" I think he sort of had the same reaction that I had when I first heard about the drug: "Well, there's a lot of drugs, there's a lot of companies that are coming here. We'll take a look at it."

When he saw the information that I saw and the unique pathway the drug inhibited, he also got very excited about the drug and said this would be great to be able to bring something like this, as novel as this, to his patients over at Inova Fairfax. That really built the partnership. Then we just took it from there. We all worked together on the protocol, worked on putting the infrastructure together necessary to get the trial up and running. Voila! We've got a study—we've got a patient here at NIH, multiple patients over at Inova, and a referral base. We're getting calls now literally everyday with several patients that we're hearing about. We're actually enrolling so quickly that we're now focusing on making sure that we can manage the data as quickly as it's coming in. There's a lot of data and there's a lot of stuff that will be done behind the scenes in the laboratories to see if this medication inhibits the formation of NETs. Not knowing when these experiments are done if they are getting placebo or not, but once all that data is put together, if it turns out that in this group we saw an inhibition of NETs and in this group we didn't, if it matches to whether receiving active drug versus placebo, then we'll have very clear laboratory data to support the drug doing what we hope it does in the person's body.

Barr: Very, very exciting. In addition to this study and through some of the earlier work you did with remdesivir, have you been a part of other COVID-19 projects in programming?

Childs: As Clinical Director, I've been involved in trying to support the research of numerous investigators in the NHLBI that are looking at ways to target the virus and develop new therapeutics and to develop ways to predict who is going to be sick when they're infected and who won't be sick. In order to do those experiments, our investigators need to have access to samples. I have been involved in striking partnerships up with collaborators outside the NIH, in particular Inova Fairfax, to do collaborative work using samples from their patients coming into the hospital and getting those samples out to our investigators that have interest in particular areas that are related to their expertise. We have nine of our investigators who got grants, an award called ITAC [Intramural Targeted Anti-COVID-19]. We were the institute that had the most ITAC grant funding. We're very proud of that and I'm very proud, as Clinical Director, to be able to help our PIs [Principal Investigators] get access to these samples, so they can do the important research that needs to be done.

Barr: That's really wonderful. I also read somewhere that you were involved in helping set up the Javits Center when all the patients in New York needed somewhere to go. Can you talk a little bit about that experience?

Childs: When I came back from Japan, I was deployed to the Commission Corps Headquarters where I was the Chief Medical Officer for Safety. I had safety oversight for the accessory hospitals that were being stood up in multiple different cities as overflow hospitals when the COVID-19 numbers were exploding, particularly in New York City and Detroit.

My job was to make sure the facilities were set up appropriately to maximize safety. We looked at workflow; we looked at all aspects of safety from donning and doffing PPE [personal protective equipment] to the patient areas, to how patients would be resuscitated if a code were to occur. If there was a breach in their PPE or if any kind of an exposure, I was involved in making sure that we assessed those officers and got them appropriate medical intervention if necessary, or isolation or quarantine or testing. I did that for about 2 months. Javits saw quite a few patients, close to 1100 COVID-19 patients. That operation went really, really smoothly, so I was very proud of all of our officers that participated in that deployment and took care of those patients and I'm happy to report not a single one of our officers got infected in the field. That was a great accomplishment and a great testament to the science related to the safety interventions and procedures that we follow being effective.

Barr: That's really interesting to hear. I'm going to move on to some more personal questions about your experience with COVID-19. Personally, what has been the most challenging part of dealing with COVID-19 for you? And have there been any positive aspects?

Childs: The challenging part is we want to get our scientists and our researchers back in the labs and in the hospital doing what they do best. But we have to do it safely so that has been a challenge to figure out how to do that. Knowing that we had to start at low numbers and gradually ramp up and also have everyone buy into the safety processes that we were engaging in. It's a slow process. We're not able to run at the level that we would if we were back to normal without COVID-19. We're running roughly at about 50 percent. I'd love us to be running at 75% but I'm not really sure that we can get much better than 50 because we have some barriers that are difficult to overcome. We need to avoid density of our staff because that increases risk for transmitting infection. We need to wear PPE in the work environment. We need to limit the number of patients that we can bring in. We have to clean after every patient. There's lots of hurdles to keep us from operating at a level that even comes close to the way we were operating before. It is frustrating to know that we have very important science that's being slowed down by this whole process.

It is rewarding to have gone from zero to 50% and we are seeing that our safety maneuvers are working. We're not seeing staff, for the most part, getting infected in the work environment. There have been a few cases here and there but, in general, the safety maneuvers we're instituting are working. I think there is some frustration with some of the investigators that they want to do more, and they want to do it quicker. It does require that we stay vigilant and that we inform everybody of the need to continue to do what we're doing to assure that we don't have a big cluster or outbreak that would set the whole thing back and lead us to the possibility of having to slow things down or even close again. That's what we don't want to happen and that was a big concern in the beginning. If our safety maneuvers failed that could lead to an all-stop and, fortunately, you have not seen that happen.

Barr: That's really good. What is something that you think—COVID-19 has been in the news and science has been more in the forefront, but there's still a lot of misperceptions—what is it that you think that the public doesn't realize about the scientific process and how do you think that NIH and other institutes like it can kind of change those perceptions for the public?

Childs: We just have to communicate the facts. You know the bottom line is, in Maryland every day we're seeing 500-800 cases and in Virginia a little higher, 700-1000. The question is what is happening? Those are people that are infected now. If someone's infected today, they didn't get infected the last six months, so what happened that led them to get infected now? Was it that they were having mass fatigue? Did they run out of hand sanitizer? Did they just have to go out and eat in that favorite restaurant because they hadn't been there in six months? I think we have to keep reminding ourselves to stay vigilant as this will pass eventually. We're hopeful the vaccines will work, and the vaccines may be available in the next couple of months. You just have to stay focused and committed to engaging in safe practices. If you let your defenses down—you can have your defenses 99% of the time; that one percent of the time you're gonna get infected. If you tell me I wore my mask 99 out of 100 times, I will tell you if you got infected, it was the one percent of the time you did not wear it. It does incur fatigue having to do this day after day after day. The way to get past that fatigue is to appreciate the light at the end of the tunnel and that light is getting brighter and brighter. Our hopes for these vaccines are becoming more and more close to reality. This is when we tell you to remind folks: don't stop now.

Barr: Good advice. Is there something about the way we're living during COVID-19 or do you think our thinking [about sanitary practices] should stay around after COVID-19 has finally ceased?

Childs: That's a good question. I think a lot of our practices are going to change permanently because of COVID-19. I think that hand hygiene is very important for the spread of infection in hospitals and we've seen hand hygiene rates in compliance at record highs. I would hope that we don't go back down to our 60% or 70% compliance rates, but we stay much higher because people understand that once COVID-19 is gone, there are other viruses, there is influenza, there are bacterial infections, things like that, that will greatly benefit in terms of reducing the risk if you have proper hand hygiene. I would see that as something that you know.

One thing that's kind of interesting we talk about is telework. It wasn't clear that you could do a lot of these jobs via teleworking. Now it's clear that a significant portion of the work that's being done at the NIH can be done remotely. We want folks on campus, but we've always had problems with having enough space for folks particularly when it comes to offices. Knowing now, from having basically been forced to telework, that we can do some of our jobs remotely gives us greater opportunities to hire more people and not have space being the major limitation for onboarding staff that may be critical to supporting various aspects of science.

Barr: That's definitely true. This is a fun question. Where in the world would you want to visit once COVID-19 is over?

Childs: For me it's less of the visiting and more of the congregating. I just want to be around people again. I want to go to family reunions. I want to be able to hang out with my friends anywhere. We can go to a place that may not be the most beautiful environment, but just being able to be together with friends and family and not having to worry about getting infected or infecting someone, that's very dear. I'm looking forward to it the most. Those weekend calls I keep getting every once in a while, it's like, "Hey, you want to go up to the vineyard this weekend?" It's like I really want to go, but I am not going because that sounds like a great opportunity to put the defenses down and get infected. I'd love to go out and do wine sampling with my friends again. So that's the real thing I am looking forward to the most.

Barr: That sounds really good. Is there anything that you would want to share with the American people as an NIH scientist and also as a person who's living through this pandemic like all of us?

Childs: The main thing is that we are looking past this virus. There will be more viruses to come. I think the most important thing is that we really analyze the job that we do when this is all said and done, so that the next virus that comes, we don't make the mistakes that we made this time and the things that worked well this time we capitalize on. We say, yes, we did that, we've seen this before and we effectively dealt with it using the following interventions or approaches or strategies to test new therapeutics. I think this is in some ways, as terrible as COVID-19 is, it could have been much worse. The case fatality rate doesn't even remotely approach something like Ebola, for instance. The next virus that comes could have case fatality rates of 20, 30, 50 percent or higher. We have to learn from this, and we have to do better the next time so that we can get therapeutics out there quicker. We can do the science quicker. We can get vaccines quicker. I think the vaccine development has been quick, but it needs to be quicker next time.

We need to be able to develop vaccines where we're able to give them to people in the order of four to six months as opposed to 10 months to a year, something like that. We've done well in some regards, but we certainly have room for improvement and that's what we really need to do is to say, okay, you know COVID-19 did damage but the next one could do a lot more damage. How are we going to be a lot more effective and smarter to deal with the next threat when it comes, having learned from COVID-19?

Barr: Thank you very much for talking with me today. I wish you all the best in your study and all the other things that you're doing at NIH. Thank you.

Childs: Thank you very much for interviewing me a second time.