Dr. Jennifer Petersen

Behind the Mask

June 8, 2022

Barr: Good morning. Today is June 8, 2022. My name is Gabrielle Barr, and I'm the archivist at the Office of NIH History and Stetten Museum. Today, I have the pleasure of speaking with Dr. Jennifer Petersen. Dr. Petersen is a research scientist in the Section of Integrative Biophysics and the Division of Basic and Translational Biophysics in the National Institute of Child Health and Human Development (NICHD). Today, she's going to be speaking about some of her COVID-19 research and experiences. Thank you very much for being with me.

Petersen: Thank you so much. It's an honor to be here. I'm happy to participate in your project.

Barr: I'm excited to hear about all the different things you've been working on these past two years. To begin with, how have you applied your training and experiences with other diseases to your approach to COVID-19? You look at the dynamic of disease, which is a different way of looking at it than others at NIH.

Petersen: Yes. My background is actually in neuroscience—I got my Ph.D. in neuroscience. I use microscopy to look at individual cells but also to look at the insides of cells—what we call the "ultrastructure"—using light microscopy. I do mainly electron microscopy so I can look at the actual organelles that make up cells and are responsible for the jobs that cells do. I applied electron microscopy to the COVID work in the lab.
In Josh Zimmerberg's lab, we also work with some other viruses and what we call "nanoparticles." We have the training to work with not biosafety level three, which is COVID, but biosafety level two, which are safer, easier to handle viruses. When the pandemic started, we wondered how we could apply what we're doing in our other projects to work with this new emerging problem.

Barr: That brings us to your COVID research. What are pseudotyped viral particles, and how are they used to study the variants of SARS-CoV-2?

Petersen: Yes. As I was just saying, SARS-CoV-2 needs to be handled in a biosafety level three lab, which means you would have to be wearing spacesuits. It's a lot of work to go in the lab and it's a lot of work to leave the lab, and all the handling is much more difficult. The field uses these little mimics—they're like little fake viruses—called pseudotyped viral particles. Those are basically just engineered little fake viruses that don't carry any viral genome. They're not infectious, but you can use a producer cell line to just basically release these little fake viruses into the culture medium. You can engineer it so that these little fake viruses that get released have, in this case, the spike protein of SARS-CoV-2 on their surface, so they look just like a little virus but they're not infectious. It has a corona of spike proteins. They have these spike proteins on their surface just like a real coronavirus would. They are used to study the function of the spike protein for coronavirus—and any other virus you're interested in studying. It's that spike protein that's responsible for binding to the cell that it wants to infect, mediating that fusion of that viral protein with the host cell to deliver the viral genome, but since these

don't have a genome, it's just a way to study the spike binding and entry. They're also used in high-throughput screening assays to search for drugs that may block or impair that spike-mediated entry or screen neutralizing antibodies that may also inhibit entry of these pseudotyped viral particles to target cells. That's how they study these aspects of the spike protein in a safe environment and a non-infectious particle.

Barr: What made you and your team consider that the structure of the Delta variant could be the reason that it has such a higher infectivity rate?

Petersen: Yes. You've probably heard a lot of discussions about spike protein because it's the target of all the vaccines that we have. Since it is the prominent structure presented on the outside of the virus, it's also the first thing that the immune system sees. It is the target of a lot of vaccines. Also, it's the target of the immune system initially, to try to tamp down infection. What were our clues that the spike protein might make it more infectious? As we all live through this pandemic, I'm sure you've heard about variant after variant evolving.

Barr: Some of them didn't even last very long until another one appeared.

Petersen: Yes, they immediately get outcompeted by the next one. It's really been a roller coaster. Since the spike protein is the main mediator of infection and it gets the process started, whenever there's a variant, there's a new collection of mutations on that spike protein. There can be mutations in other proteins of the virus, but it's the ones on the spike protein that scientists look at the most because that spike protein is so important. It can be the source of vaccine resistance and neutralizing antibody evasion. If a new variant displays this increased infectivity, it can quite likely be because whatever mutations occurred on the spike of the new variant could, in different ways, promote that spike being able to increase the binding of the spike to the ACE2 receptor on the target cell. It could increase the ability of that spike protein to drive fusion and entry of the particle. Or it could have made the spike more invisible to the immune system so that it was less reduced by neutralizing antibodies or interferon response of a cell. Whenever there's an increase in infectivity with these variants, there's a good chance that it's going to be due to some changes in the spike protein.

Barr: Can you discuss your methodology and analysis of the variants—particularly the Delta one?

Petersen: Absolutely, yes. As it went, when the pandemic started, like I said, we wondered how we could redirect our research to help with this disaster. Dr. Zimmerberg formed some collaborations with a group in NCATS, the National Center for Advancing Translational Science—Catherine Chen and Miao Xu—who were working with these pseudotyped viral particles in high throughput screening assays to screen possible drug blockers of spike mediated entry. They were working with a local biotech company called Codex BioSolutions, which is run by Jimmy Lu. We met those two teams early in the pandemic. Since we did electron microscopy, we got involved in looking at the pseudotyped viral particles as they were developing them to produce them. They're really pure, had good spike proteins on their surface, and worked well in their entry assays in their high throughput screening. As we would look at this particle, each time a new variant came, I would look at them by a process called negative stain electron microscopy, where I can just directly look at them and see them. They look like they're structurally intact—they have lots of spikes on their surface. Every time a new variant came

along, or they were testing a new type of pseudotyped viral particle, I would take a look. Actually, it was just serendipity that when the Delta variant emerged in the [United] States in mid-2021—before that the Alpha variant had been prominent, and then it got replaced by Delta—we had looked at Alpha and we had looked at several other variants before that. When I look at them by negative stain, I see the pseudotyped viruses, and they look like little viruses. They would just be sprinkled around on the surface that I had attached them to. I was looking at them on an electron microscope.

Barr: Is it the same process for every single one?

Petersen: We try to keep it the same for any differences. I can see the spikes. But by negative staining them, you can't see, for instance, individual amino acids, and you don't get to atomic level resolution. But when I got to the Delta, they also were nice little round spheres with spikes on them, about 100 nanometers in diameter. Instead of being sprinkled around on the substrate, they were often in clusters—or what we called aggregates—of 10, 20, or 30 of the pseudotype. It's like little viruses, but they were in a clump. I hadn't seen this with any of the other pseudotyped viral particles. Did something go wrong with this sample? Proteins can aggregate if they get denatured or damaged in some way—if you change the temperature or the pH or different things. Or is this something unique about the Delta spike? The particles are identical except for the spike on their surface. If the others are present as single pseudotyped viruses, but the Delta end up in little aggregates, the only thing different between them is the spike protein. Is this aggregation due to the spike protein? I'm not a virologist by training, but at that point all of us became pretty knowledgeable in viruses just because of the news and everything that I'm hearing.

I wondered if there's anything about viruses aggregating. Does that happen in any kind of physiological context that would have relevance or do viruses ever want to aggregate? Is this good for a virus under some circumstances to be in a little group? Or do they always want to be individuals? You go and you search papers online and look on PubMed, and it looks like there is this emerging evidence more and more—that in some cases viruses like to travel in groups, because in some cases one virus has the potential to infect a cell. But if anything happens to go wrong while that virus is trying to infect the cell, it doesn't succeed—like maybe it doesn't bind well to the surface of the cell, and it never gets in. Or maybe it's trying to get in the cell, but in the meantime the immune system comes and eliminates that virus, so it doesn't get in. Or maybe it attaches well, but something goes wrong with its ability to pierce the membrane of the cell and introduce its genome. There are reasons why a single virus on its own might fail to enter a cell and infect the cell. But if they were in a little group and they could bind to the surface of the cell in maybe a group of ten, then that might increase the chances of one of them getting in before the immune system was to eliminate it. It might increase the number of spike proteins that can look for their ACE2 receptor to bind. There are other contexts with other virus types they found, that in different ways viruses can travel in groups, and that increases their infectivity. But we needed to show that this was not what we call in science an “artifact”—meaning some kind of result of how we're handling them that has nothing to do meaningfully with the behavior of the virus.

We went to Jimmy and said, “Hey, Jimmy…” In his company, he makes these pseudotyped viral particles. We saw that the Delta particles are in aggregates unlike any of the other pseudotyped viral particles shared with us.

We wanted to test this and see if this is unique to Delta. He designed some experiments where he produced four different kinds of PV [pseudotyped viral particles] spikes—PVs with no spike at all, so just a bare vesicle, and the Delta spike containing PVs. He produced them all in parallel, meaning produced them at the same time—different dishes, but all in the same production run. Then they get released into the culture medium—so they don't really get manipulated too much. They don't get spun at a really high speed or anything to concentrate them. They just get released by the cells, these little virus-like particles. You just collect the culture medium. Then I would drive to North Bethesda, pick them up right after he collected them, bring them back to the lab, and immediately get them onto the substrates that I used to look at them on the electron microscope. And, importantly, I blinded them, so I didn't know which was which so I would have no bias about looking for the aggregates for the Delta. When we did that, we repeated that three times. In each case, after we unveiled the identity of each sample after we'd done the analysis, only the Delta were observed to be in these aggregates. None of the others, nor the control, had spike protein. They were never in these large aggregates that we saw in the Delta.

Barr: How many does the Delta travel with?

Petersen: This is something to look into in the future. In these studies that we did where we just took the culture media from the cells, brought it back to the lab, put it on the EM [electron microscopy] grids, and looked, they had been prepared within at most four hours from the time they were removed from the cells. At that time point, a group would have on average about 30 of the particles in the aggregate. About 70% of the particles would be contained in one of these aggregates. The other 30% would be singles going around by themselves. But we found that if we let more time go by before we looked at them, they would continue to aggregate into bigger and bigger aggregates, which the other spikes didn't do.

Barr: Did the others aggregate at all? In pairs or anything?

Petersen: They didn’t. Okay, you saw certainly maybe 20% or so might be in a doublet or a triplet, or sometimes even four or five, but nothing on the order of the Delta. If you let the Delta go overnight, there would be hundreds in aggregates that reached several microns. This is in a culture dish and in culture medium, and it was not with real virus. The question becomes, does this ever happen in a real-life condition with Delta? That’s for future experiments.

Barr: Can you talk a little bit about that—some of the implications that you and your team believed may be an effect of the Delta aggregation? You talked a little about how maybe this aggregation could be higher transmissibility, but also related to higher virus load and being able to knock out the balance of the host antiviral system because it's such a big group coming at it.

Petersen: Yes, exactly. An aggregate has a bigger surface area, so that means more spikes. Those could bind to the surface of the cell with higher affinity and stay associated with the surface of the cell longer than if a single virus was to contact the surface of a cell but then it happens to let go—off it goes. Being an aggregate could increase the strength that the aggregate binds to the cell with and give it more of a chance for one of those

particles to enter. If more than one virus can enter a cell simultaneously, that's a mechanism called collective infection.

Barr: Do you think that's what was happening since people got so much sicker with Delta than other variants?

Petersen: The thing about Delta is they found that people tested earlier by PCR [polymerase chain reaction]. If they were exposed on a Monday, they might test positive by PCR test in two or three days compared to Alpha, where it was maybe five days. They became positive faster and when they tested positive, they had a higher viral load—meaning more virus in their sample. The aggregate could increase the binding and then it could allow multiple genomes to get delivered to one cell simultaneously. What that can end up doing is creating a burst of more viral replication than if one genome got delivered on its own. Suddenly, the cell would start making many more viral proteins at the same time. You would end up with faster infection and a bigger initial burst of infection—which is exactly what they saw with the Alpha. And since you deliver multiple genomes like this at one time, it's also harder for the cell's immune system to knock it out. It would mount an interferon response, but since there was so much virus having entered the cell, it was too much for the immune system of that cell to counteract.

Barr: Do you think they work together in these groups at all? Is it just a numbers thing, or are there any thoughts that they could be working in tandem?

Petersen: The way that it could be cooperative is if not every virus is functional. You could have viruses produced that maybe just happen to not package very many spike proteins. If it was just up to them, they almost have no chance of successfully infecting a cell. But if it could get stuck into this aggregate, it goes along for the ride. Then it ends up getting in anyway, even though it wouldn't have before, and then its genome contributes to the infection. This deep into virology isn't really my area of expertise—but if multiple genomes get delivered simultaneously, there could also be some genomes that are defective. Those genomes can then get corrected by recombination when the genome starts getting read, and maybe it gets to a spot where it's defective. If it was on its own, that'd be the end of that one. But since there are other viruses getting replicated at the same time, you basically get repair of the genome of that virion. It kind of gets fixed—and off it goes.

Barr: With Delta being so successful with the aggregation, it's very interesting that Omicron did not have that and has taken over. Is there any explanation for that?

Petersen: Yes. When there's mutations on a spike, that maybe gives it some fitness benefits. A lot of times, multiple mutations occur on these variants and there's an interplay between them. Some of them, on their own, might have actually reduced infectivity of that virus or in some way reduced its fitness. But other mutations can increase it at the same time. You end up with a kind of collective effect of the mutations. Delta seems to have some set of mutations on it that lead to this aggregation, we think, and that did impart this benefit to it. Then Omicron came seemingly out of nowhere and it has so many more mutations on the spike protein. It's something like 30—compared to others which might have had five new mutations or 10 new mutations. It has tons. It just shifted the properties of that spike so much. It doesn't do the aggregation, but it has other features

that made it outcompete Delta. I was reading a little bit about the changes in the Omicron spike. It's just so different from other spikes. It has this really high transmissibility and seems to infect a different population of cells—your upper airway cells more than your lower airway cells, which is where the Delta and other variants infected. The Omicron gets in your upper airway and then it just—in my casual reading, I'm not up to date on everything with Omicron—probably spreads better through sneezing and coughing, because it's the upper airway thing. That may be one of the reasons why it can just spread so fast—but I shouldn't talk too much about it, because I definitely don't know the latest on the Omicron. It doesn't aggregate but it had so many other fitness benefits that it took over.

Barr: Has your research prompted subsequent studies on looking at aggregation in the Delta variant as well as maybe aggregation with other viruses?

Petersen: Yeah, we are still interested in what's mediating this aggregation at the molecular level. I worked with some cryo-electron microscopists on the paper we published. We confirmed what I saw, by negative staining them, via flow cytometry and nanoparticle tracking analysis done by Wendy Fitzgerald. Those are machines that can measure the number and size of the particles in the whole solution that you put through the machine—as opposed to what I look at, which is what happens to stick onto the substrate that I put in an electron microscope. Wendy looked at everything present in the culture medium to confirm that we saw larger aggregates in Delta compared to the others that we tested. Actually, Doreen Matthies and Fei Zhou—neighbors of ours from another lab in NICHD that does cryo-electron microscopy, which does give you the near atomic resolution where you can look at the actual conformation of the spike on these particles—are starting to look at the interaction between the spikes at the cryo-EM level to try to get clues about how the spikes are interacting. Are they tip to tip like this? Are their sides interacting? Or are they actually getting pushed out of the way and then you just have the membranes of the particles interacting? We see examples. We see particles at all these distances in the clusters in the aggregates that we see. We're looking at the spike with the cryo-EM. We're thinking about introducing some mutations onto the Delta spike or evaluating the mutations and the surface properties of the spike to generate hypotheses about the physical properties that are mediating this adhesion between spikes. You might think that if Omicron doesn't aggregate, and it's now the dominant variant, then it's interesting about Delta but doesn't matter. I think it matters because some of these mutations can reemerge. We think that for future variants—should there be any, which there probably will be—as we screen new spike variants for their antibody resistance and things like that, you should also screen them for whether they're aggregated and how much because that could be a therapeutic target. If you know that a virus's transmission is facilitated by aggregation, you could find drugs or treatments that disrupt the aggregation. That could knock down the transmissibility of a coronavirus—or any virus that might evolve or arise in the future.

Barr: This is a question based on what you just said. At what point in the emergence of a virus does it start to aggregate? I don't know if that's something your group is interested in, but at what point in a virus's existence does it do this, and what are the conditions that make it do this?

Petersen: Yes, those are probably the most interesting questions to me as a cell biologist. I'm really interested to know if the Delta aggregates during biosynthesis as the viruses are assembling in the cell cytoplasm. Are they getting in close proximity and leaving the cell already as a little group? Or are they meeting each other after they get released and aggregating on their way somewhere? This is a respiratory virus, so this is something that would have to happen—maybe in the mucus or somewhere in the airway secretions as it got released. Or a virus can aggregate on the target cell. It's going to be some effective concentration. They have to get close enough to stick to each other. Maybe that happens on the host membrane. Maybe if several bind at the target cell membrane at the same time, maybe then they happen to encounter each other on the cell surface and stick together. Then maybe they enter the target cell together. I would really like to use electron microscopy to look at the cells producing the virus and see if we see, in any kind of a cell compartment, any signs of aggregation inside the cell or as it is released from the cell, or on a target cell—and see when this aggregation is occurring and in what contexts it can occur. They have to meet each other. There has to be some chance of them meeting each other and getting close enough to bind to each other at some point.

Barr: Are you guys looking at the different variations of Delta? Delta had multiple iterations. Are they similar? Are they different? You saw clusters of 30, which is a lot.

Petersen: Well, we looked at two. We looked at a Delta variant B1.617.2—I can't remember these numbers all the time—and also there is AY variant that came up, AY4.2. They both aggregated. We didn't compare if one of them did it more than the other. If you look at the actual amino acid sequence of the spike, they were very similar. The AY had a couple of additional changes from the Delta. There are databanks where you can look—any time a sample got taken from a person, it could get submitted to these databases for sequencing. There were possibly millions of different ones, and they all get cataloged. Researchers look at the sequences and make a call whether it's more like Delta or more like Alpha and they decide if it's a lineage. It's very complex, but there were many catalogued sequences that got classified as a Delta and a Delta subvariant, like you're saying. One of the things that could be done is to look at all of those Delta-like sequences and find ones that have certain sets of mutations or maybe a couple—missing one or missing two—and compare those. If we find one that really enhances aggregation over another, it'll give you a big clue about how it's happening. You might have heard about "Deltacron." For a little while, there was a spike that had both Omicron and Delta characteristics. Luckily, it didn't catch on. I was worried that it was going to be like Omicron but with aggregation. It's like the perfectly bad spike. But like you mentioned earlier—there were some that kind of popped up and then went away and never caught on. I'm really glad about that, but I was immediately interested in that. I looked up some sequences and I found some Deltacron sequences that had some of the mutations that were unique to Delta. I was really worried that it might aggregate but there were only a couple of isolates, and it didn't become a thing, so we didn't have to worry about it.

Barr: You said that some other viruses aggregate. That's just really interesting. What are some of the other kinds of viruses that aggregate?

Petersen: For instance, enteroviruses. I'm not up on all the virus types. There's many. Another NIH scientist, Nihal Altan-Bonnet, studied the polio virus. What she discovered is that polio virus actually buds out of cells in a

membrane carrier. This is a type of collective infection or grouping of viruses in a bubble. They leave encased in a membrane. That protects them from anything in the immune system that would want to attack the viruses because they're encased—they're hidden inside a membrane—and then it orchestrates this group delivery. HIV also has different mechanisms in that it can undergo group infection. I know it clusters on the outside of dendritic cells and then that cell contacts another cell, which is the target cell of the HIV. Viruses can undergo collective infection by just directly aggregating like what we saw with Delta. VSV [vesicular stomatitis virus], which is like rabies virus, can do that in saliva, and HIV can do that on the surface of other cells or by sticking to fibrils or cell debris. Some viruses aggregate by sticking to something else. Some are in a vesicle, like the polio virus. Another example is a thing called biofilms. For some viruses—this is true of a leukemia type virus—when it gets released from the cell that produces it, it gets released into a film of adhesive molecules and it forms a biofilm. That's a way for viruses to stay in a type of aggregate. They can directly aggregate. There are also other viruses that aggregate on bacteria, and then they ride on the bacteria to look for target cells. It's different mechanisms that viruses can use to aggregate.

Barr: That is fascinating. I wonder, from a history of medicine standpoint, if you could ever go back in time and see if terrible outbreaks of a particular disease occurred because that virus was an aggregation form.

Petersen: I think for viruses on bacteria or viruses in feces, aggregation is definitely a mechanism. This is because viruses and aggregates are also resistant to environmental factors like dehydration or UV—basically because the viruses deep inside the cluster don't get dried and the UV doesn't hit them. We inactivate viruses with paraformaldehyde and things like that. If the cluster is really big, viruses deep inside might not get inactivated. That's another thing. If you know a virus aggregates, that will change the mechanism that you might use to do your disinfection on a surface. It might change the amount of time you run your UV light. The people that study how to inactivate viruses and protocols for sanitization would need to know if the virus is in aggregate or not. I was really fascinated learning all this stuff.

Barr: What were some of the challenges that you and your team have encountered with the work that you have been doing?

Petersen: The challenges…? I would say just that it's difficult to work with the real virus. Obviously, we'd love to do all these experiments with the real thing, but because of the safety concerns we aren't doing that at this point. But I think it's great to do all this preliminary characterization. Often, what people do is first study in a more accessible system before going to the more difficult or more challenging system. Working with the real virus, we're limited at that. Early in the pandemic, the challenge was just working in this kind of scary situation where we had limited access to the lab. You had to work by yourself. But I felt a great sense of purpose. I was so proud to be part of the effort. As a basic scientist— “basic” just means studying these fundamental processes—sometimes you can't see a direct line to helping people. But with this worldwide effort to combat this virus, it felt really important to do what we could. I was really proud to be a part of NIH at this time because I'm just so proud of everything NIH has done during this pandemic. I really appreciate being part of the team and part of NIH and being able to do something, hopefully, to help. It’s very motivating.

Barr: Definitely. Will you speak about some of your contributions to some other COVID-19 research and initiatives you've been a part of, are currently involved with, or are planning?

Petersen: Other work going on in the lab is working with another type of virus-like particle. We would like to study more about the biosynthesis of the virus inside the cells and how it assembles. But again, that's very hard to do with real virus. Another strategy people employ is to use what are called virus-like particles, of which the pseudotyped viruses that I mentioned are a type, but we also have been working to produce virus-like particles that are composed of only CoV-2 proteins. For these other particles that I mentioned, you use another virus's core to make the particles, and you put the CoV-2 spike in it. It's not a pure CoV-2 particle. You make the virus-like particles by just working with the four structural proteins of the CoV-2 spike—what's called an M, an E, and an N protein. Under certain conditions, you express those cells in a producer cell line—a cell that just likes to make proteins. We put plasmids encoding those structural proteins into the producer cells that like to crank out proteins. We try to put them in ratios and under conditions where they will assemble into little empty viruses. They look like a virus on the outside but there's nothing on the inside. Working with those structural proteins of CoV-2, we can then look inside cells and see where they're assembling, how they're assembling, if they're aggregating or not, and study their release. Then you can also study the released CoV-2 VLPs [virus-like particles] for other studies. Making those CoV-2 VLPs has been going on in the lab. I look at them by electron microscopy and try to assess the quality of the particles. Do they look like little viruses? Do they have all the parts? How pure is the preparation? That's one thing we're doing. That's the other main project that I'm working on that's CoV-2 related.

Barr: In addition to being a scientist, you're also a person who has been living through the pandemic. What have been some opportunities and challenges that COVID has presented for you as an individual living through these times?

Petersen: Yes. I think we all kind of recognize that we're really living through a moment in history where someday some kid is going to say, "Wow, you were alive during the pandemic, what was it like?" We're still in it so it's hard to really know all the repercussions it's going to have, but as I said earlier, I've just felt really proud to be at NIH. It feels very rewarding to be able to work on this in real time. I'm single, so I would be at home, watching and following the news. Sometimes they would report scientific breakthroughs that had just come out on bioRxiv [Bio Archive] like overnight—so I would be following the news, looking up bioRxiv for anything that's published in that moment. But then since we were able to work on projects directly related to the virus, I could also come to the lab before a lot of other NIH employees. I would be in the lab doing electron microscopy, but all alone because you couldn't have more than one person in a room and all that. You would maybe see your coworker through the window and wave at them, but for weeks we were just being really careful working alone. It was just all coronavirus, all the time. That was very, very, very dramatic and a little scary. As electron microscopists, you're often working alone, and on the microscope you're in the dark all by yourself. But I was actually looking at the spike proteins myself with my own eyes! It was very intense, but I learned a lot. It was definitely a crash course in viruses and coronavirus. I feel like we're still kind of in the middle of that. I know a lot of people are kind of acting normal again—I'm cautiously optimistic that things will get better, but I'm also aware that these variants can come back kind of fast, right? We'll see.

Barr: Thank you so much for all you've done and everything you've shared. I wish you and your team only the best, and I can't wait to see what comes from your continued research.

Petersen: I really appreciate it. This is a wonderful project you're doing. I also just want to make sure I thank my team members that helped me with this recent publication we had in the journal *Viruses*—my group leaders Josh Zimmerberg; the cryo-EM team that we worked with, Doreen Matthies and Fei Zhou; Rick Wong in the National Cancer Institute, who helped with some of the cryo-EM; Wendy Fitzgerald, who's in Leonid Margolis's lab, also in NICHD; and our statistician extraordinaire, Paul Blank, in Josh Zimmerberg's group as well. Another aspect of this whole thing has been the teamwork and everyone bringing their expertise to the fore and just making it their priority in this intense time. It's just been an honor to be a part of the team—the "local" team and the larger NIH team.

Barr: That is wonderful.

Petersen: Thank you so much. Thanks.

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