# Presymptomatic/Asymptomatic Transmission of SARS-CoV-2

*[Announcer] This program is presented by the Centers for Disease Control and Prevention.*

[Sarah Gregory] Hi, I'm Sarah Gregory, and today I'm talking with Nathan Furukawa, a CDC Epidemic Intelligence Service officer—otherwise known as a disease detective—who's working on the COVID-19 response. We'll be discussing evidence that people who have coronavirus disease without symptoms can still spread the disease.

Welcome, Dr. Furukawa!

[Nathan Furukawa] Hi Sarah! Thanks so much for having me on your podcast. It's a real honor to be here and be able to talk about my work.

[Sarah Gregory] Great! Well, let's start off with some basics. Tell us the difference between COVID-19 and SARS-CoV-2. I know that it's still being confused a lot, especially even in the media.

[Nathan Furukawa] Yeah, I appreciate that. So, SARS-CoV-2 refers to the actual virus itself. And so SARS-CoV-2 causes COVID-19, which is the disease that stands for coronavirus disease 2019. So, sometimes when people talk about COVID-19, they usually are talking about symptomatic disease. And so, sometimes I'll tend to use the term "asymptomatic SARS-CoV-2 infection." But you could also say, "asymptomatic COVID."

[Sarah Gregory] Okay, well let's have some more basics here. What do presymptomatic and asymptomatic mean?

[Nathan Furukawa] Yeah, so SARS-CoV-2 infection comes in two flavors. There's presymptomatic infection…and so that's someone who's been infected, but they're in their incubation period and so they're not yet showing symptoms but they eventually do show symptoms. And the average incubation period is about 5 days, but can be as long as 14 days. And so that's something different from asymptomatic infection, and that's people who are infected with the virus but actually never develop symptoms. And so, when we're talking about asymptomatic or presymptomatic infection, you know, that's also important to note that that's different from transmission. So, infection means being infected with the virus, but transmission means that these people who aren't showing symptoms are actually spreading the virus to others. And, sometimes asymptomatic/presymptomatic transmission can be a bit of a mouthful to say, so sometimes I'll refer to it as "silent transmission." You know, I think others…I

have seen other reports refer to “cryptic” or “undetected” transmission. But I think this might moreso refer to those transmission events that are not counted in official numbers because maybe it occurs in people who don't ever develop symptoms, or maybe the symptoms are so mild they never go get care, get tested, and get officially diagnosed.

[Sarah Gregory] This study involved doing a rapid literature review. Tell us what that means.

[Nathan Furukawa] Yeah, it was indeed very rapid. So, the gold standard for a review is—well, like a systematic review—and that might come with or without a meta-analysis. And so, usually those follow pretty strict PRISMA guidelines that, you know, involve you developing a formalized protocol for finding papers, figuring out how to register that protocol in a registry. And then, when you're reporting what you've found, making sure all the elements are standardized so that, if a researcher wanted to understand “Alright, what was reviewed? What were the gaps? And can I be able to replicate it?” they're able to do that. So, we did a rapid review. And so, this is something that's an abbreviated synthesis of the evidence. And, it's more tailored to provide timely information for decision making. So, systematic reviews can take weeks, months, but rapid reviews can take place over a much shorter time period since, you know, policy makers have deadlines for making decisions. And so, in the setting of a really rapidly evolving outbreak, a rapid review is probably more preferable because we're always getting this new information constantly trickling out, and at some point you've got to take that information and rapidly synthesize it in order to inform a public health intervention. The risk of doing a very systematic approach, is by the time you get through the whole process, some of those findings might actually be outdated based on new studies that have aroused.

And so, for this rapid review, it took place at the beginning of April. And me and my coauthors were actually working at all different levels of the CDC response. We were all following the literature on asymptomatic and presymptomatic transmission, and I was on the clinical team compiling this information to update CDC's clinical management guidance. But, you know, we had just updated the, the guidance and had received some external feedback about a section on asymptomatic infection and transmission. And so, we were all discussing the different papers, which papers were stronger or weaker, and actually, while we started doing this we got a request from the CDC Incident Manager to urgently generate a memo summarizing all of the literature. And, you know, when I say rapid, I really mean rapid. Like, we got this request in the morning with a deadline of later that afternoon. So we really quickly scrambled, we got our writing team together and we made…we made the memo deadline, and then we then generated a more detailed report which was reflected in what was actually published by EID recently.

[Sarah Gregory] I have to say that that's actually incredible. I mean, from A to Z that you got it written that quickly—that it finalized that quickly—got it cleared, and then we got it through our publishing process which involves peer review and copyediting and then posting online. So, good to have this! I'm so glad it worked.

[Nathan Furukawa] Yeah, it was quite a whirlwind process!

[Sarah Gregory] So, what kind of epidemiologic evidence did you find?

[Nathan Furukawa] Yeah, so we kind of looked at the papers and put them into three different buckets. The first bucket was epi studies. We also looked at virologic studies, and then pulled together some mathematical modeling studies. For the epi reports, we found that 11 epi reports that described these clusters of COVID cases where the index case, or primary case, was thought to have transmitted this infection to their contacts during the time that they weren't showing symptoms. And so, this…you know, most of the studies we saw were showing presymptomatic transmission. But we also found a few clusters that demonstrated asymptomatic transmission. Since China had experienced, you know, community transmission first, most of these studies did come from China. Typically it was an index case who had traveled to Wuhan or another city in Hubei, which was the epicenter of the outbreak, and these index cases then traveled to other cities in China before the lockdown. And so, most of these secondary cases tended to be household members, you know, people you'd have extensive contact with over the course of if you were visiting family members in another city. But, we also saw reports of transmission during transient encounters, like sharing meals with other people or even patients being infected in the hospital where index cases visited that same ward earlier in the day. One of the big limitations from these studies, though, is that, you know, since it happened in China and the disease spread quite rapidly, it's, it's kind of hard to completely rule out the possibility that there was some undetected community transmission to explain this disease pattern that we are seeing.

So, what was really helpful was that there were two reports that we drew from, from other countries that did not have community spread at the moment, and that was Germany and Singapore. So, there's an early German report that described a German businessman in Munich, who had a meeting with a mildly symptomatic colleague who was on a business trip from China. And then, during that time period he…after that meeting, he had meetings with two other coworkers who never had contact with that symptomatic Chinese businesswoman. And all three of them went on to progress and become symptomatic and were diagnosed with COVID. So that really provided some pretty strong evidence of presymptomatic transmission. But really, probably the best paper was a paper describing seven COVID clusters in Singapore. And, each of these clusters had an index case where they had a distinct period

where they were exposed to the virus—probably through travel or contact with someone who was a known case—and then a separate distinct period where they exposed their contacts when they were presymptomatic. And so, from these clusters there were definitely examples of healthful transmission, but also transmissions occurring during church services or even singing lessons. I imagine if you're, you know, singing at a quite loud volume that might be potentially spreading respiratory droplets pretty efficiently. So, but, you know, taken together, these epi reports formed a pretty strong backbone of the evidence for asymptomatic and presymptomatic transmission.

[Sarah Gregory] And just for clarification for listeners, an index patient is the original patient that has it that spreads it to others, right?

[Nathan Furukawa] Yes, yes.

[Sarah Gregory] Okay. So how is a SARS-CoV-2 infection usually diagnosed now?

[Nathan Furukawa] The most common way SARS-CoV-2 is diagnosed is through reverse transcription polymerase chain reaction, or RT-PCR, sometimes PCR for short. And that's detecting actual viral RNA from a clinical specimen. And so, that's commonly done on nasopharyngeal swabs or nasal swabs. It can also be done on oropharyngeal swabs at the back of the throat, for sputum. For patients who are intubated in an ICU, you can also do tracheal aspirates or bronchoalveolar lavage. And there's some research studies demonstrating the presence of…of CoV-2 RNA in other body fluids like tears, saliva, or stool, but that's primarily for research purposes and not diagnostics.

But I think it's important to pause here and say I want to be really clear that, just because you detect viral RNA, does not mean that there's infectious virus present. And so, in order to understand, "Okay, does the detection of viral RNA actually mean that there is infectious virus?" we can do viral culture, which is actually taking a sample and trying to extract and grow virus and replicate it just to show that, in fact, this is probably virus that's infectious. One of the downsides is that viral cultures are really resource intensive, so it's not able to be scaled up in a way that we could use it readily for epidemiologic purposes. But what we could use is a cycle threshold, and that's the number of PCR cycles it takes to detect SARS-CoV-2 virus. So you know, if you can imagine, if it's a lower number that means fewer cycles were needed to detect virus and so that would equal having more virus. At this moment, it's kind of unclear what cycle threshold correlates with having sufficient virus to be transmittable. You know, cycle thresholds above 40…probably not infectious. Cycle thresholds above 35…prob…possibly not, but it's unclear. There's not an exact number at the moment.

[Sarah Gregory] Okay. So then, backing up a little bit, you also looked for virologic evidence. So what did you find there?

[Nathan Furukawa] Yeah, so we found some evidence from about six reports that we drew from, and each of these studies showed asymptomatic patients who had SARS-CoV-2 infection. But they had virus and reported cycle thresholds that were low enough that it's possible that they were infectious. You know, two reports actually went a step further and actually took those samples and cultured live virus, and I think having the presence of live virus is pretty strong evidence there's potential transmissibility. I think it's worth highlighting some reports that actually my CDC colleagues worked on and then reported, and it was around the investigation of an outbreak of COVID in a nursing home in Washington state. So, the investigators had noticed that there was a COVID-19 case detected at the nursing home. So they went around and tested as many people as they could consent in the nursing home, and they found 48 other people who were positive. And what's really interesting is that 27, or about a little over half, actually didn't have symptoms at the time that they tested positive. The stroke of brilliance here is that they went back at a later time and they assessed who was initially asymptomatic and went on to develop symptoms and then how many people remained asymptomatic. And it looks like of those 27, 24—or almost 90%—did eventually go on to develop symptoms (so they were presymptomatically infected), whereas about 3—or close to 10%—had remained asymptomatic. Very concerningly, they ran the PCR and showed that cycle thresholds in people who were presymptomatic or asymptomatic tended to be below 35. So they were actually able to culture live virus from people who were presymptomatically infected, and 7 of the 11 samples they tested are about, like, two-thirds actually were positive for live virus. So, you know, it's important to note in this evidence base of virology studies, they didn't document any specific transmission events, but just the presence of virus with the characteristics to be present and culturable really provided, again, some pretty strong evidence of presymptomatic transmission.

[Sarah Gregory] Okay, and so apparently there was also…you mentioned some modeling evidence. First of all, explain to us what modeling is and then talk about the evidence.

[Nathan Furukawa] Sure. I have to admit, I am not a mathematical modeling expert. But, you know, mathematical modeling is…it's a tool that's used and can be used in epidemiology to describe the spread of an infectious disease, and then hopefully be able to predict its future progression over the course of time. And these models, they rely on basic assumptions that are derived from real life or estimated, and they're inputted into this model in order to predict how this disease spreads through a population. And they can be really useful to either explain what we're seeing or predict where things will go, and that can be really important for decision making.

But, you know, some, some people really dislike models because they're easily manipulated by the assumptions built into them, and it can be really difficult to know in the moment, like which assumptions are valid and do any assumptions change over the course of the epidemic as it evolves. There's not an easy way to answer this. So, all the models have some degree of uncertainty built into them, and they're not right all the time. And this can become a little challenging when communicating to the public because oftentimes models are given too much certainty with…by the public than they actually have, and that might generate mistrust if models aren't accurate. But again, models can be really helpful for helping us understand what's happened.

So we used two types of models for this study. The first type of model was one reporting on what's called the serial interval, and this is the time between the symptom onset of the primary patient, and that's compared to the time of symptom onset in the secondary patient. But if you think about a disease that can only be transmitted by symptomatic patients, then that serial interval will always be positive ‘cause the primary case has to develop symptoms before the secondary case does. But on the other hand, if you've got a disease that's transmitted presymptomatically, there may be even cases where that serial interval might be zero (they developed symptoms on the same day) or even negative (the primary case developed symptoms after the secondary case).

So, we found two studies looking at these infector-infectee transmission pairs, and they both estimated the serial interval to be about four days. And so, that's important because if you compare it to the mean incubation time for SARS-CoV-2 infection, it's five days. So since it's shorter, it might suggest that there are transmission events occurring before the onset of symptoms. And then one study estimated that about 13% of transmission events were presymptomatic.

So, the other type of model we reviewed was this….a traditional epidemic model, and these studies took data from the outbreak in China and tried to fit data and assumptions in order to explain the really rapid rate of increase in cases that they experienced. And they noticed that, you know, they…from their model, they detected that a majority of infections that they detected were probably attributable to transmission from people with mild or no symptoms. And so, you know, so you think about it, someone’s asymptomatic or presymptomatic, maybe they're not as infectious to someone with…as someone with symptoms, but, you know, if they have….many more people have undetected infection that's mild or asymptomatic, it might explain some really rapid spread of the disease that was seen early in the outbreak in Wuhan.

[Sarah Gregory] So the aggregate of these three types of evidence means what?

[Nathan Furukawa] Each of these three types of evidence have their own limitations and what they're able to say—and we review that all in the article—but really when you start piecing all of the puzzles together, I think it paints a pretty convincing picture that supports our conclusion that asymptomatic and presymptomatic transmission is possible, but that it's also an important driver of the ongoing pandemic.

[Sarah Gregory] And I think you touched on this briefly at the very beginning, but tell us again why was this research done?

[Nathan Furukawa] Yeah, I had mentioned there was a pretty urgent ask to summarize the evidence, and this report was prepared in part to support some CDC recommendations on cloth face coverings for the public when leaving the household and then also universal face mask use in healthcare facilities. So, since we can't know who's infectious after they've been exposed and usually COVID's not detected until someone's symptomatic, which prompts them testing, really we're looking for interventions that broke the chain of silent transmission. And so, that's where this cloth face covering recommendation for the public came in to effect. You know, cloth face coverings aren't perfect. I, I think the jury is still out on how effective they are compared to face masks in preventing asymptomatic transmission, but if you think about it mechanistically, it can make sense. If you've got something covering your mouth, it can probably capture a lot of those infectious particles and reduce the spread of those particles and risk…and the risk of transmission to others. And there are pretty minimal risks to the wearer. They're probably not sufficient on their own to control the epidemic, but you know, you want to choose everything you can to enhance the efficacy of everything else that we're doing to bend the curve. And again, this is just an instance where, in the middle of a rapidly expanding outbreak, you might not have all the answers but you've got to take decisive action based on the science you know and then try and implement those interventions in order to bend that curve downward.

[Sarah Gregory] What are the public health implications of pre- and asymptomatic transmission? People still talk so much about being careful around others with symptoms.

[Nathan Furukawa] Yeah, I saw that question there. There's a lot to say about this and then I sat back and thought about it. Gosh, I think presymptomatic and asymptomatic transmission has implications for just about every public health tool we have in our toolkit to fight the disease. You know, up to now we've been relying on these broad nonpharmaceutical interventions, which I think you had a guest on your prior podcast discussing them. And then these large, community-based policies that are designed to disrupt the transmission of the virus, and that could be travel bans, doing physical-social distancing, school or workplace closures, cancelling large events; there's staying at home or staying-at-home orders.

And, you know, these interventions were really important for us flattening our curve, but they're quite disruptive and can have very severe economic consequences. And so, as we're shifting from these broad, nonpharmaceutical interventions to more targeted interventions, I think the role of presymptomatic and asymptomatic transmission becomes really evident. You know, one of the first interventions we rolled out was symptom screening at points of entry, like airports. And, while it's helpful in being able to intercept people who have symptomatic disease, it's not as effective when trying to capture people who have asymptomatic infection and maybe just have been exposed. And this sort of symptom screening requires a lot of public health manpower and so, it might not end up being the most targeted intervention we have.

You know, we have isolating ill people once they're diagnosed or before they're diagnosed, and so, this is a cornerstone intervention of field epidemiology and it's important because once someone's isolated you're preventing the transmission of the disease to other people in the community. You know, the less time the people who are infected are out moving around in the community exposing others, the fewer transmission events there will be. But you know, again, here if we're only focusing on isolating people who have symptoms, we might be missing that other group of people who don't have symptoms but nonetheless might be infectious to others. So, in, in thinking about isolating people at home, there's the added complication, you know, that might reduce the spread in the community but that might also potentially increase the risk of household members getting infected, especially if there's asymptomatic transmission. So, some countries like China and Korea have developed these fever clinics or long-term care facilities that allow people to go and stay in until they might have mild illness or no…even no symptoms at all. And they might stay in that setting until they clear their virus and are able to go back and not expose their family members.

Thinking about contact tracing, gosh, you know, contact tracing is when, you know, you've found someone who has been diagnosed with COVID-19 and you go back and interview all the people that they came into contact and consider testing them. You know, for the initial response, contact tracing was really focused on that symptomatic period for the index case and testing was prioritized for those contacts who had symptoms. Which made sense—there wasn't enough tests at the time. But, you know, as we've learned more about asymptomatic infection and transmission, CDC has changed its guidance to…for contact tracing, making sure that late presymptomatic period's included, and also including a consideration of testing of asymptomatic contacts.

But, I think there's just still a lot of questions that remain about what's the optimal contact tracing strategy. You know, should we test all contacts regardless of…of their symptoms or not? Or maybe we

should just focus on those that are at highest risk, such as people who are symptomatic or any close household members. And another question is, you know, what's the best way to quarantine exposed contacts? And, if we can't test everybody, there's a chance that some of these people might have asymptomatic infection and be silent transmitters of the virus.

It's probably not feasible to quarantine all contacts, especially, you know, if it happens in the setting of a healthcare facility where people are essential workers. So, you know, the big question I have is what's the optimum return to work? Is it having people with strict face mask-wearing and monitoring for symptoms? Is that enough to lower the risk of exposing to others? So, as you can see, asymptomatic and presymptomatic transmission really opens up a lot of different questions and has a lot of significant implications for the response.

[Sarah Gregory] But nothing overtly solvable at this point?

[Nathan Furukawa] Yeah, unfortunately there's still a lot we don't know about this virus and so, we're combing the literature trying to figure out what can we learn to implement and improve our response.

[Sarah Gregory] You're study revealed some really important knowledge gaps—critical knowledge gaps. These include the relative incidence of asymptomatic and symptomatic SARS-CoV-2 infection, the public health interventions that prevented asymptomatic transmission, and the question of whether asymptomatic SARS-CoV-2 infection confers protectiveness. How can these gaps be addressed when answered?

[Nathan Furukawa] Yeah, you know, I'll be the first to admit, like, this virus is really humbling and the more we learn about it, the more the more questions that are asked. So, to get to your first point, we don't know how many transmissions are from people with asymptomatic or presymptomatic infection compared to regular symptomatic infection. You know, we've learned from a number of different experiments that, in fact, the asymptomatic infection might…rate might be actually pretty high. There's the natural experiments, like the infections that happened on cruise ships or navy ships; we've seen high rates of asymptomatic infection in homeless shelters and then also during universal screening of pregnant women. But, you know, our understanding of how much transmission comes from this group of people with asymptomatic infection is still unclear. We'll probably have to wait for some other surveillance data or prospective cohort data that uses serial PCR testing or serology in order to get a better sense of these numbers. I'm concerned that many infections are related to asymptomatic or presymptomatic transmission. And so, you know, as I mentioned before, we just have to make sure that we're able to detect these people through enhanced contact tracing and testing.

You're second point was, you know, have…we need to know what public health interventions are most effective at reducing the infectiousness of these people who are asymptomatically or presymptomatically infected. You know, we know that total lockdown was pretty effective at leveling off the epidemic curve but, you know, we can't stay in lockdown forever, and this is likely going to be a protracted fight. So, as we're looking to open up the economy, we need to understand what are the interventions that we can keep in place in order to ensure that there's not a resurgence of infections that's driven by silent transmission events.

And the third point was about immunity. So, you know, we don't know enough about immunity for both symptomatic and asymptomatic infection. You know, I think in an ideal world, all people with SARS-CoV-2 infection would develop complete long-lasting immunity and would get to herd immunity pretty rapidly. But, you know, there's a lot of respiratory viruses out there, like influenza and other coronaviruses, that only convey partial immunity, and then this immunity might wane with time. And people with asymptomatic disease might develop no immunity. So, really understanding whether immunity is full or partial and then durable or transient, that's going to be important for us understanding whether we're going to have some resurgence or a second wave of COVID. And it's also helpful in helping us to determine who's safe to return to work. So again, there's a lot we don't know, but we are searching the literature and trying to understand and fill these knowledge gaps so that we can best figure out how to open the economy, and then how to start recovering from this pandemic.

[Sarah Gregory] So, maybe you've already covered this, but I just need to go back over a little bit. We don't really know then how very mild cases would fit into this scenario. I mean, if you're mildly sick, would you potentially have the same contagiousness as a very sick person or not? Or that's one of the things we don't know yet?

[Nathan Furukawa] Good question. I mean, so we're pretty convinced about the possibility of asymptomatic transmission, but yeah, we don't know the relative infectiousness of people who are asymptomatic versus presymptomatic versus mildly symptomatic. We do know that people who go on to develop symptoms have a peak in their viral load around the time of symptom onset, so people who have presymptomatic or early mild symptomatic disease might have a similar risk of transmission. And that might be different from people who have asymptomatic infection—so that's people who never go on to develop disease—and they might not have the same peak in viral load that people who do go on to develop disease have, and so as a consequence, might be less infectious. But, really I think it's a comment on how little we know about this disease and the way it infects people. And we think like other respiratory viruses, SARS-CoV-2 is spread primarily through respiratory droplets when someone

coughs, sneezes, or even talks. But, you know, people without symptoms might not have a cough, might not have a sneeze. And so not having that means that they're not necessarily propelling these respiratory droplets to other people who are their close contacts.

So, how is it being spread otherwise? Well, you know, we know SARS-CoV-2 has been detected in aerosols and there's some studies that can show it can survive for several hours on surfaces. And so, we don't know if people are getting infected through those mechanisms more than would be expected in a similar respiratory virus. And I think, since we don't know, I think this really reinforces these broad public health messages that we have around physical distancing, hand washing, and for healthcare providers, wearing respirators, especially during those aerosol-generating procedures, or face masks when respirators aren't available.

[Sarah Gregory] We were talking a little bit before about protectiveness and the gap in the knowledge about that, I mean if you've had the virus. But talk to us about antibodies. I hear people saying constantly that they have antibodies or they hope to get antibodies from having had it or soon there will be herd immunity because of all these antibodies. But there's actually no evidence that antibodies will be protective, isn't that right?

[Nathan Furukawa] Yeah. I think, you know, we don't want to rush to judgement. There's still a lot that we're learning about these serology tests and, you know, this, this is a novel coronavirus. No one has underlying immunity, everyone's susceptible. This is a new disease, and so serology will be really helpful in understanding the spread of the disease in the population. But, you know, serology and immunity are two different things. And you're right, they often times get conflated. Serology is a test that's measuring antibodies. But antibodies can reliably tell if you've previously been exposed to the disease, but having antibodies does not necessarily mean you're immune. Like, for instance, I work in HIV and antibody testing is how we diagnose HIV. Because you have an antibody against HIV does not mean you are immune.

Now, there's some evidence coming through that high titers of IgG antibodies might correlate with having neutralizing antibodies, but there's still a lot of variability in the serologic tests and so I think it's a little dangerous to infer, you know, a positive antibody from a serology test means immunity. So, this idea of using, like, serology immunity passports I think is a little risky because you might end up giving people false positive results, and thinking that because they have antibodies they get false reassurance and then put themselves in dangerous situations.

Actually, I've been thinking about that. I heard recently in the news that there were people having coronavirus parties, trying to potentially infect themselves and their family members in order to get immune. This is a serious disease and intentionally infecting yourself and others is dangerous, not just to yourself, but dangerous to the people around you. And, really, we don't even know if being infected conveys lasting immunity from reinfection. So, it's probably obvious to say, but these are not recommended.

[Sarah Gregory] Yeah, Yeah, I just cringe in horror when I hear about those…those parties.

Okay, you work at CDC. What's your job, and what activities have you been involved in?

[Nathan Furukawa] Yeah, so my training is as a physician with a background in internal medicine and I'm currently in the Epidemic Intelligence Service, which is probably something familiar to many of your listeners. But, in case it's not, EIS was originally established in the 1950's by Alex Langmuir. And the goal was initially to have a force ready to protect America from biologic warfare in case that occurred during the Korean War. But since its founding, it's expanded its scope and EIS officers have been involved in, really, most key events in medical history, including smallpox eradication, helping discover the pathogen for Legionnaire's disease, and describing, you know, the early HIV/AIDS epidemic. And there's currently 130 of us that are at CDC or embedded in state and local health departments around the country. And we serve as the boots on the ground disease detectives during an outbreak.

So, normally I work in the Division of HIV/AIDS Prevention, and my research focuses on HIV prevention and PrEP, or preexposure prophylaxis. I've been involved in a couple investigations in responses to HIV outbreaks around the U.S. More recently though, there's been a lot of international outbreaks and so I was…actually, they're working on the COVID-19 pandemic since January. I was initially deployed to the Democratic Republic of the Congo to assist the Ministry of Health with border health screenings for Ebola. But as COVID-19 evolved, I actually got pulled more into coronavirus preparedness activities and did a lot around airport screening protocols.

Later, after I finished my deployment in Congo, I came back to the U.S., I joined the clinical team as part of the CDC COVID-19 response, and I worked on developing clinical management guidance, responded to inquiries from the public and policymakers, and I was involved in a study describing hospitalized patients with COVID-19 in Georgia that was recently published in an MMWR. Now, I'm working with the chief medical officer of the CDC response and our team combs through the literature to ensure that our strategies are being informed by the latest scientific discoveries.

So, it has been a real honor to be able to be able to serve my country during this really challenging time. And EIS has probably been the most hardest but also most fulfilling professional experience I've ever had. I'd say if anyone is on the fence about considering applying, I would highly recommend it.

[Sarah Gregory] I think EIS officers are considered the "glamour people" of CDC, externally and internally. And I'm always envious that I chose a different path. But…

What are you doing to reduce your stress in these very challenging times?

[Nathan Furukawa] Yeah, you know, I'm probably not the best person to ask for advice. Gosh, you know…knowing every moment counts in that early phase of the pandemic, I admittedly haven't done a good job at giving myself personal space to recuperate during this pandemic. I think everyone on this response knows that this is a defining moment for us personally, for CDC, for the country. And also I, like probably a lot of my colleagues, have friends and family who are frontline workers caring for patients with COVID. So, you know, I pause, I think about the experiences they're going through and really it, it pushes me to give this response my all. But thankfully, my wife does a pretty good job at encouraging me to step away from work every now and then. You know, she'll make sure I go to the walk…go to a walk, visit a park, enjoying spending some time together. And I'm really grateful to have her looking out for me, and I owe her big time for getting a really absent-minded husband for the past half-year.

[Sarah Gregory] Aww. Well, thank you so much for taking the time to talk with me today, Dr. Furukawa.

[Nathan Furukawa] Thank you so much. I really enjoyed getting to share what our paper was.

[Sarah Gregory] And thanks for joining me out there! You can read the August 2020 article, Evidence Supporting Transmission of Severe Acute Respiratory Syndrome Coronavirus 2 While Presymptomatic or Asymptomatic, online at cdc.gov/eid.

I'm Sarah Gregory for *Emerging Infectious Diseases*.

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