If you have additional questions, so let’s now turn to our second Speaker. Doctor Ellen Foxman is assistant professor of Laboratory Medicine Ann Immunobiology and Extensive work now really understanding the immune responses and natural responses to respiratory viruses. Which is certainly a very timely topic of research.
Uh, in 2020?
So we were really pleased that Alan could take the time to share her research with us.
So Ellen, thank you.
Thank you. I'm happy to be here.
And now I'm going to hopefully share the screen and it will.
All will go well. Um?
All right?
00:00:35.930 -- 00:00:36.842 Uh, in 2020?
00:00:36.842 -- 00:00:39.355 So we were really pleased that Alan could take the time to share her research with us.
00:00:39.355 -- 00:00:42.208 take the time to share her research with us.
00:00:42.210 -- 00:00:43.462 So Ellen, thank you.
00:00:43.462 -- 00:00:44.400 Thank you. I'm
00:00:44.400 -- 00:00:45.636 happy to be here.
00:00:45.636 -- 00:00:47.490 And now I'm going to hopefully share the screen and it will.
00:00:47.560 -- 00:00:49.426 share the screen and it will.
00:00:49.430 -- 00:00:52.150 All will go well. Um?
00:00:54.660 -- 00:00:55.580 All right?
00:00:57.540 -- 00:01:02.040 Uh. So can you see the slides? Yes,
This actually is not going to be a talk about cancer. It’s going to be a talk about COVID-19, which is also a topic on everyone’s mind these days. Today I’ll be talking about why we are interested in studying the early host responses against respiratory viruses, specifically applications to COVID-19. This is just a disclosure that I’m going to inventor on to patent applications.
or in this case in particular.
SARS coronavirus two,
the virus that causes cobra 19.
I'll give a brief overview
on the basics of Cobra 19
diagnostics an I’ll talk about,
then a project that we’ve been doing
since March on screening using host biomarkers for this disease and then
future directions of the project.
So as I was preparing this talk,
I looked back at some of my previous talks and this is actually an intro slide I had from a talk I gave at the end of
November to the virology faculty group,
and I thought it was kind of.

It looks so different in the lens of our current environment that I thought I would show it.

So I used to start my talk by convincing everyone of the importance of respiratory virus infections, which is a much easier sell now, but actually, even before this pandemic, these infections cause. Over 500 million infections per year in the US, so that’s more than one per person and granted a lot of those are common colds, but some of those are
00:02:54.050 --> 00:02:55.346 serious illnesses such as.
00:02:55.350 --> 00:02:57.074 Influenza with hospitalization or
00:02:57.074 --> 00:02:59.229 hospitalization for asthma attack or
00:02:59.229 --> 00:03:01.414 CEO PD Exacerbation which are very
00:03:01.414 --> 00:03:03.525 often caused by viruses and also
00:03:03.525 --> 00:03:05.691 there has been this emerging this
00:03:05.691 --> 00:03:07.162 lingering concern about emerging
00:03:07.162 --> 00:03:08.690 infections with good reason.
00:03:08.690 --> 00:03:11.042 As we know now and I usually put
00:03:11.042 --> 00:03:13.332 up this photo to describe that
00:03:13.332 --> 00:03:15.357 that’s actually a picture of
00:03:15.357 --> 00:03:17.828 the SARS coronavirus from 2003.
00:03:17.830 --> 00:03:20.679 But now when we see these pictures
00:03:20.679 --> 00:03:22.653 it definitely conjures up something
00:03:22.653 --> 00:03:24.687 else in all of our minds,
which is the 2nd SARS Coronavirus SARS CoV2.

Uh, which causes the disease cobra,

and I just checked on the Johns Hopkins Portal and at the moment there's over 7 million cases and over 400,000 deaths described globally from Cobra 19, so this is definitely having a high impact.

It’s impacting our seminar that were having if I zoom today, it’s impacting our work.

It’s impacting our economy and of course our health and there’s still a lot of unanswered challenges.

were right in the middle of it.

Trying to figure out how to deal with it.
Um, and even when this acute phase is over, there will be long-term impacts, both on the health of the respiratory system in the patients who are recovering, or have recovered and we also have to think what lessons can we learn from this that are going to help us with the next pandemic. So this is sort of a just a screenshot of my labs homepage to remind me to tell you a little bit about what we really focus on the lining of the respiratory tract, the airway mucosa as you see in this picture.
This is actually what the epithelial layer in the upper airway looks like, and these are these cells. The epithelial cells are the target cells of viral infection and viruses replicate in these cells. And these cells also are the first line of defense that recognizes the infection and sends out signals to the immune system to come to the area and also sends out turns on affecter mechanisms to try to stop the virus from replicating. So there are very.

It’s a very highly active tissue.
The airway mucosa.

Our lab is focused on these early steps of host defense, and we’re also interested in repair. Actually, because after the.

It doesn’t constantly regenerate, but rather only when damage

Their way isn’t like the skin.

It doesn’t constantly regenerate, but rather only when damage

Their way isn’t like the skin.

And one thing we’re interested in
right and sometimes goes wrong,

and sometimes when it goes wrong

that leads to cancer and that I

hopefully I'll be able to come back

for a different grounds and talk

But for today I’m going to focus

on the upper respiratory tract.

As the gatekeeper against infection,

so most of the pathogens that

come into our airway come in

through the nose and mouth throat,

and this includes viruses and bacteria.

And often if that infection can

be nipped in the Bud in the upper

respiratory tract that protects
the rest of the respiratory system from that infectious agent getting down to the lungs. So when these offense defenses are effective in the upper respiratory tract, it can really be the difference between miles or asymptomatic illness. Versus a serious illness. And we know that that’s happening all the time, not just with SARS, but other viruses that often there cleared from the become their detectable in a way for a time. A short time.
They and they are cleared without the patient knowing that they were there. That can happen, or you can have the opposite, where the patients in the ICU. So we’re interested in factors that modulate those defenses, and we like to think of it as like a marble sitting on a mountain where this is the very beginning of the immune response. That’s going to recruit certain immune cells in the respiratory system and sort of nudging that marble in one direction. It will roll down the Hill one way,
and you’ll get one type of response, whereas if you nudge it in the other direction, it can have a very different outcome. So we’re very interested in understanding the molecular basis of that.

This is a another picture of this as an upper respiratory tract from a child, and so what’s something that’s kind of interesting about this anatomy is I actually just myself today. Had a swab for this surveillance and we all notice swab goes Kobe 2 and we all notice swab goes.
right in here in the nasopharynx, and that swab also collect some of the patients own cells and some of the proteins made by the patient's own cells. And in a study with Marie Landry of the director of the clinical virology lab back in 2018, we showed that you can actually detect the patterns of jeans and proteins being made in the respiratory tract and the huge changes that occur in the rapid response to viral infection. And if you think about the progression of SARS, Co V2, there’s you probably have all seen a figure something like this.
And of course this will be refined overtime, but the basic idea seems to be that at this early stage of infection we have upper respiratory tract replication and those kinds of symptoms. Then it moves to the long and then in severe cases there’s a host inflammatory response. It causes a lot of damage. At this early stage, what we can find out using these respiratory swabs is what can we think about alternatives and additional things we can do for the best diagnosis an even,
can we understand the difference is an inflammatory response is the very beginning that dictate the way the illness is going to progress? So today I'm not. I'm not gonna talk about bullet .2, I'm gonna talk about bullet .1 today. The diagnosis end. So I'll just start with giving a brief overview on diagnostics for a SARS Co V2. I know we have a diverse audience here an I gave a full length, uh, detailed description of this stuff for one of the Deans workshops that's available online. That this is everything in a nutshell,
so I'm going to describe the test that we are currently doing at Yale. New Haven for this virus. The first test answers a question. Does the patient have the infection right now? And basically what you do for that? Is you do the swab isolate are an RNA. Can you detect viral jeans from the viral genome in this patient sample an if the answer is yes, it means a patient has the virus or the viral RNA and their nasopharynx right now and and that test is
very specific because we’re just looking at the genome of this virus and very specific regions. Sensitivity depends on when your sampling and sample collection but it’s a highly specific test. The other question, of course, is did the patient had the infection? Is there evidence of past infection and that’s serology? So that’s asking has the patient formed antibodies against the virus because they’ve already had the infection? Usually for a minimum of two weeks
to have an adaptive immune response.

And kudos to our clinical lab for having

both of these up and running for awhile now.

Marie Landry in the virology lab,

and, uh, Rick Tourism.

The clinical immunology lab

have set these up and they’re

available to order on the patients,

and this is our go to test to know.

The server balance you know

someone is infected right now.

But there are still challenges.

Are there still a lot of challenges

that we’re facing right now?

One is how to expand testing capacity,
and there’s many different avenues this can go down.

There is a group with Nate groove on an Wiley doing great stuff with saliva.

Testing is one way, but there are other ways we can be screening or expanding testing capacity to help make sure we’re not spreading this virus.

Further, as we restart the economy, another challenge is that some people who test positive by the PCR tests don’t actually seem to be infectious based on a study from South Korea and a few other observations elsewhere of people who recovered and still test positive.
positive for a long time but don’t seem to spread the virus to their Contacts. So how can we tell the difference there and then finally also very important is how do we find new viruses that are going to be the next pandemic that are going around and causing Ellis in our patient under our radar? These kind of questions are why we got into looking at the host response. In addition to understanding pathogenesis. But sort of on the practical side of how can it help us an once is to die for diagnosis.
We’re all familiar with them. I mean the basic one for infection is fever. Fever is a host response to infection and fever. Is fever elevated? Leukocyte count? Those are signs that the patient has an infection. They’re not terribly specific, but they are a host response that has been used for, you know, long time, hundreds of years, even the fever. But now we can get more granular.
NOTE Confidence: 0.912440240383148
00:12:21.560 --> 00:12:22.844 Patterns of gene expression,
NOTE Confidence: 0.912440240383148
00:12:22.844 --> 00:12:24.770 patterns of protein expression using Multi
NOTE Confidence: 0.912440240383148
00:12:24.819 --> 00:12:26.719 Plex Technologies like transcriptomics an.
NOTE Confidence: 0.912440240383148
00:12:26.720 --> 00:12:28.841 The idea is if a patient comes
NOTE Confidence: 0.912440240383148
00:12:28.841 --> 00:12:30.510 in and is coughing,
NOTE Confidence: 0.912440240383148
00:12:30.510 --> 00:12:32.568 you don’t know what’s causing that,
NOTE Confidence: 0.912440240383148
00:12:32.570 --> 00:12:35.234 but if the if that’s being caused by a
NOTE Confidence: 0.912440240383148
00:12:35.234 --> 00:12:37.078 respiratory virus that’s replicating.
NOTE Confidence: 0.912440240383148
00:12:37.080 --> 00:12:37.694 That’s activated,
NOTE Confidence: 0.912440240383148
00:12:37.694 --> 00:12:38.922 the immune system turned
NOTE Confidence: 0.912440240383148
00:12:38.922 --> 00:12:40.150 on antiviral defense is,
NOTE Confidence: 0.912440240383148
00:12:40.150 --> 00:12:41.605 which are different then defenses
NOTE Confidence: 0.912440240383148
00:12:41.605 --> 00:12:43.755 against an irritant or a bacteria or
NOTE Confidence: 0.912440240383148
00:12:43.755 --> 00:12:45.365 other things that cause coughing.
NOTE Confidence: 0.912440240383148
00:12:45.370 --> 00:12:47.836 And if you look at the patterns of Gene
NOTE Confidence: 0.912440240383148
and proteins that the body is making, you can sort of interrogate the bodies own diagnosis and and know what’s going on. And so, this is based on the study from 2018. A very simple question was, are there common patterns to all respiratory viruses that we can look at to say? Is this patient experiencing a respiratory virus infection right now or not? Because you may not know this, but in the winter seasons I’m not talking about this year but in past years between December, and March redo thousands of panels.
00:13:24.600 --> 00:13:26.336 of symptomatic patients testing

00:13:26.336 --> 00:13:28.267 them for 15 viruses to see.

00:13:28.270 --> 00:13:28.636 Uh,

00:13:28.636 --> 00:13:30.832 which virus might be causing their

00:13:30.832 --> 00:13:32.949 respiratory symptoms and only about 1/3

00:13:32.949 --> 00:13:35.182 of them actually have a viral infection,

00:13:35.190 --> 00:13:37.647 so 2/3 of them may have some

00:13:37.647 --> 00:13:39.000 other process going on.

00:13:39.000 --> 00:13:41.254 So we asked whether we can look

00:13:41.254 --> 00:13:43.154 at Biomarkers of the antiviral

00:13:43.154 --> 00:13:45.304 response to identify who those

00:13:45.304 --> 00:13:47.469 patients with viral infection R.

00:13:47.470 --> 00:13:49.690 And this is to this is

00:13:49.690 --> 00:13:51.170 published something to sum

00:13:51.253 --> 00:13:52.689 it up very quickly,
but the idea is that we found that jeans
and proteins that are highly induced
during the antiviral interferon response.
If you detect those in the nasopharynx,
it's a very good indicator that
there's a viral infection there,
and this colored graph just shows kcii 10.
This is actually one of these
interference stimulated jeans.
It's a cytokine.
It goes up many orders of magnitude during viral infection and
the level of it highly correlated
to the presence of the virus.
So this is like the level on a log scale,
that there’s a virus present.

And we did two different studies at two different times of year with two different viruses circulating an in both of those are represented on these pie charts, which viruses were amongst the virus positives and it’s basically any virus that we test for. We could pick up in this way and So what are the potential applications for Koba 19? We want to know do these pan viral biomarkers pickup COVID-19. It’s possible it could be different, and if so,
how can this help us fight the pandemic,
so there's a lot of more ideas
this is a relatively new project,
but I'll just share some of our early
data and this project so far has
been spearheaded by ready chi Marla,
a postdoc in my lab who's been like
side by side with me in the lab
every day since this pandemic hit.
every day since this pandemic hit.
Trying to do the studies I'm going to.
Tell you about and get them down the
road and I also wanted knowledge.
The lab working group.
I'll talk about them again at the end.
Organized by Albert Cohen,
the School of public health who helped us at the beginning all get organised together to get the PCR testing going for research. You sent a support clinical use too. And so this is a graph of Cobra 19 Indiana, the country in our region. Green is the country. The first case was in January. But in our region of Connecticut, in New York, the first case was shown in the blue on March 2nd, Connecticut first case it was in Fairfield County on March 6th.
And our testing began on March 13th, which is actually very fast.

You may recall there is some snafus with the CDC test and they allowed high complexity in clinical labs like ours to do their own test starting on February 29th. Anna Marie Landry and the folks in the clinical virology lab had it up and running by March 13th. So very fast, but nonetheless, we wondered, did we miss any cases in those weeks before our testing started? So we performed a screen of the
about the two weeks before testing

started as shown on this Gray bar.

And, uh, first,

so during this time period a lot

of people have been tested on that

complete panel for 15 viruses and

376 patients who are symptomatic

were negative for other viruses.

So we thought, well,

maybe some of those might have had SARS,

Kobe 2 and we screened with

the button marker.

I mentioned CL 10 and out of

all those negative patients,
only about a tenth of them were positive for the biomarker.

So it seems a good setup like these are people who tested negative for other viruses, but there's symptomatic. It may have a biomarker that a viral infection, their bodies fighting a viral infection. So then we tested all these people for with the PCR test, and it turns out that among these biomarker positive people were four patients who had actually did have SARS, including some surprises like an infant that was seen as an outpatient, and that was a bit of
a surprise to find that. And unfortunately, being here at Yale, we have so many great collaborators with different expertise, we were able to ask Nate Grubaugh slab in the school of public health to sequence those for isolates. This was a paper earlier published by the group lab showing using sequencing of the virus that a lot of the early cases coming to Connecticut were from transmission that were domestic rather than international and the four cases. I hope you can see this,
but the four cases that.

we had picked up in those early weeks.

We had picked up in those early weeks.

Kind of fit this pattern.

Three of the case is shown

with the sort of red lines.

They do a track most closely with North

American other isolates from North

American other isolates from North

America as opposed to other countries.

And then there was one that tracked most

closest to strains from Western Europe.

So this kind of fit the pattern will

also is really interesting to me.

Is that all these for patients that came

within a couple of days the hospital

none of their viruses were directly
related were the same as the other,
so this is independent
introductions coming in,
which was also probably says something about
travel back and forth and things like that.
So that was quite an interesting
bonus of being a in collaboration
with other folks at Yale.
To find more information
about those patients.
Uhm,
but we also had an idea just looking at this.
Well this is interesting.
Like here we used up,
you know 376 PCR test to
00:19:08.942 --> 00:19:10.330 test all these patients.
NOTE Confidence: 0.895463764667511
00:19:10.330 --> 00:19:13.003 But really if we had only tested the 33
NOTE Confidence: 0.895463764667511
00:19:13.003 --> 00:19:15.540 that were positive for the biomarker,
NOTE Confidence: 0.895463764667511
00:19:15.540 --> 00:19:18.308 we still would have found all the cases.
NOTE Confidence: 0.895463764667511
00:19:18.310 --> 00:19:20.122 And so it suggested maybe this
NOTE Confidence: 0.895463764667511
00:19:20.122 --> 00:19:22.130 is a way of expanding,
NOTE Confidence: 0.895463764667511
00:19:22.130 --> 00:19:22.958 like conserving,
NOTE Confidence: 0.895463764667511
00:19:22.958 --> 00:19:24.614 testing capacity or directing
NOTE Confidence: 0.895463764667511
00:19:24.614 --> 00:19:26.946 it towards people who really are
NOTE Confidence: 0.895463764667511
00:19:26.946 --> 00:19:28.466 high suspicion to be positive
NOTE Confidence: 0.895463764667511
00:19:28.466 --> 00:19:30.847 and so we tried that so far just.
NOTE Confidence: 0.895463764667511
00:19:30.850 --> 00:19:31.831 Piloted one day.
NOTE Confidence: 0.895463764667511
00:19:31.831 --> 00:19:33.793 We picked one day in March
NOTE Confidence: 0.895463764667511
00:19:33.793 --> 00:19:36.143 where we were able to get all
NOTE Confidence: 0.895463764667511
00:19:36.143 --> 00:19:37.447 the residual samples from
NOTE Confidence: 0.881870329380035
00:19:37.523 --> 00:19:39.388 testing went 144 patients were
00:19:39.388 --> 00:19:41.954 tested that day for SARS Co V2.

00:19:41.954 --> 00:19:44.730 And did the biomarker test an what you

00:19:44.811 --> 00:19:47.899 can see is again as a smaller proportion

00:19:47.899 --> 00:19:50.908 of people were positive than negative.

00:19:50.910 --> 00:19:53.339 And then we compared this to the

00:19:53.339 --> 00:19:55.852 results from the PCR testing and it

00:19:55.852 --> 00:20:00.779 turned out that 17 people were PCR

00:20:00.780 --> 00:20:02.760 positive for SARS Kobe to that day.

00:20:02.760 --> 00:20:03.750 And 16 of them were among

00:20:03.750 --> 00:20:05.822 the biomarker positive,

00:20:05.822 --> 00:20:07.380 but one wasn’t one was did not

00:20:07.380 --> 00:20:11.340 have the biomarker expressed,

00:20:11.340 --> 00:20:12.990 and that patient also happened

00:20:12.990 --> 00:20:13.970 to have a very low viral load,

00:20:13.970 --> 00:20:15.997 which is kind of something

00:20:15.997 --> 00:20:17.011
we’re following up on.

So if we had had all 17 up here,

we could have said are

negative predictive value.

If you’re negative on this biomarker,

you don’t have the virus is 100%,

but we can’t say that we

have to say 99% because of.

This this one patient out of out of

the 144 that were screened and tested.

Um, so we that got us interested in

biological variables and how they

impact this biomarker that’s induces

approaching that’s induced by viral

replication within the epithelial

cells and possibly infiltrating cells.
And we looked at all the positive patients in our initial study, which was 59 patients. If you look at their age distribution there mostly in the older age groups, and if you look at the symptoms by age group, the people in the older age groups had more serious illness. As you might expect much more likely to be hospitalised and have things like pneumonia and hypoxemia. So what about the correlation with the biomarker?

Well, if you look at, uh,
if you look at viral load

versus the biomarker,

there’s a positive correlation.

As you might expect.

Because, as I mentioned,

the trigger for production of this biomarker is viral replication.

Um, interesting if you look at age versus the biomarker,

where this biomarker is lower and the people with the older age is.

But there doesn’t seem to be a clear correlation between agent viral load in this same group,
00:21:41.370 --> 00:21:43.040 so we’re still investigating this.

00:21:43.040 --> 00:21:45.175 So we actually struck up a collaboration with the Pediatrics Department,

00:21:45.175 --> 00:21:48.695 including Tom Murray and Danielle Pediatrics to delve into this further and see if we can figure out what’s going on with this age correlation.

00:21:51.069 --> 00:21:53.549 I so finally I just want to mention um, what’s ahead for this project?

00:22:00.070 --> 00:22:02.700 what’s ahead for this project?

00:22:02.700 --> 00:22:04.470 I mentioned from these headlines some of the challenges and we would like to know Kenneth biomarker help us to the question of who has live infectious virus versus a persistent PCR positive but not infectious.
Anna question everyone always asked me. I'm just going to preempt it. It would be great to know what this type of biomarker an in general, what the host response to infection, how it’s changing overtime during the course of what can be a long illness. And so we’re actively looking at that right now. And I just want to finish. Briefly got pause. Dan is getting restarted now of trying to find the next pandemic virus before it hits using this strategy.
And this was spearheaded by Amelia Hammer in a Yale School of Public Health Masters student who is in my lab but graduated in 2019. And our idea there was the same idea of let’s look at people who their doctors suspected viral infection sent the test. They tested negative for all the viruses on our panel and see if we can find people who who looks like their body was fighting a viral infection and maybe they have a viral infection that we don’t know so we can find out what other
viruses are causing disease in our patient population that were not catching with our panel. And so Amelia just took one week of January 2017 and screens 250. One negative samples with our biomarker that we talked about here CL. 10 and she had 60 of them that were had high levels of the biomarker at that time. We were not doing testing for the seasonal coronaviruses or parrot influenza virus. so she did that testing an interesting Lee. Half of these patients had seasonal coronaviruses and
that actually tipped our hat.

Let us know that seasonal Corona viruses are circulating in our patient population and actually Marie Landry has now added that to the clinical panel. So now that is those four viruses are on our panel, but this also as a proof of concept that our strategy works of picking up viral infections that we’re not testing for. Um, Interestingly, we still don’t know exact well for some of them we do,
but many of them we don’t know what infectious agents are in the sample, and we’re working that up and finding some interesting things, and we hope this will be a good strategy. Going forward to get an even more comprehensive view of the viruses that are circulating so we can be prepared for ones that we aren’t necessarily testing for right now. So, just to summarize, um, we’re interested in studying the host response to fight coronavirus today. I talked about diagnostic applications I was really interested in getting
insights into early stage pathogenesis.

And how this differs among people who have different outcomes.

Uhm, I talked about a host response based screening test that we've been working on, which allowed us to identify for undiagnosed cases from early March and we're looking at other utilities to sort of fill in the gaps in some of our testing strategies, and hopefully I'll be able to update you in a future talk on our undiagnosed viruses project as well.
I conclude I'd like to thank all the many, many people in this Yale environment have contributed to projects on COVID-19. Definitely could have been done in a silo. It was very great to have lots of collaborators an it still is. I want to acknowledge my lab members including ready tomorrow. I mentioned who spearheaded the project. I talked about as well as Marie Landry on the clinical virology lab, especially Marino in and Robin Garner, who really helped us alot. Dezhen Zou, who's been helping with our bioinformatics,
I didn’t really talk about that today, but he’s been a great help the whole group, all lab and Nate grew bath for their constant participation and help with the molecular Epidemiology. As well as lab working group depicted here from March 2nd which includes Albert Konate, Bhosa Domer Akiko Isaki Marie Landreau. That’s me actually. And this was back when there’s only 45,000 global cases on March 2nd. Uh, so with that? Uh, I think I made up some time. Uh, in in speaking a little quickly,
And if there’s any questions I would be happy to answer them now.

Thank you Ellen. Thank you and congratulations to you and your entire research group on that impressive body of work in a relatively short time to address the pandemic.

and I know we’re just about the top of the hour or so,

One is specifically. I mean, I think the work you’re doing interesting in terms of testing strategy, and you mentioned that you’re
00:27:08.125 --> 00:27:09.750 anticipating one of my questions, which was, how does it change over the course of the illness?

00:27:11.634 --> 00:27:13.749 But I’m curious, do we have a sense of biomarkers that might predict the severity of illness that is almost to predict who’s more likely to need more intensive care at the time of diagnosis?

00:27:13.750 --> 00:27:14.647 Yeah, that’s very interesting people.

00:27:14.647 --> 00:27:17.153 There’s been a some work already published about blood like cytokines in the blood that could be indicated indicative of that we’re looking even earlier.

00:27:19.241 --> 00:27:21.375 I mean it at the early stage

00:27:21.375 --> 00:27:25.440 Yeah, that’s very interesting people.

00:27:23.427 --> 00:27:25.440 There’s been a some work already published about blood like cytokines in the blood that could be indicated indicative of that we’re looking even earlier.
00:27:40.289 --> 00:27:42.234 of infection, the nasopharynx.
NOTE Confidence: 0.932838261127472
00:27:42.234 --> 00:27:44.406 And that’s one reason why we’re
NOTE Confidence: 0.932838261127472
00:27:44.406 --> 00:27:46.278 really interested in this potential
NOTE Confidence: 0.932838261127472
00:27:46.278 --> 00:27:48.093 difference between adults and kids.
NOTE Confidence: 0.932838261127472
00:27:48.100 --> 00:27:49.186 Because, you know,
NOTE Confidence: 0.932838261127472
00:27:49.186 --> 00:27:51.358 kids are seem relatively protected from
NOTE Confidence: 0.932838261127472
00:27:51.358 --> 00:27:53.220 pulmonary disease compared to adults,
NOTE Confidence: 0.932838261127472
00:27:53.220 --> 00:27:53.850 older adults.
NOTE Confidence: 0.932838261127472
00:27:53.850 --> 00:27:55.740 So that’s one reason why we
NOTE Confidence: 0.932838261127472
00:27:55.740 --> 00:27:57.727 struck up this collaboration with
NOTE Confidence: 0.932838261127472
00:27:57.727 --> 00:27:59.887 Pediatrics to try to understand.
NOTE Confidence: 0.932838261127472
00:27:59.890 --> 00:28:02.046 Is there some difference in the robustness
NOTE Confidence: 0.932838261127472
00:28:02.046 --> 00:28:04.350 of that initial response that could you
NOTE Confidence: 0.932838261127472
00:28:04.350 --> 00:28:06.330 know that could possibly explain this?
NOTE Confidence: 0.932838261127472
00:28:06.330 --> 00:28:07.293 There’s many explanations,
NOTE Confidence: 0.932838261127472
00:28:07.293 --> 00:28:08.256 but that’s one,
so that’s that’s the kind of thing we’re going to we’re looking into, but I don’t have the answer yet. This is it’s very rare to give a talk on a project that started like two months ago, but so that’s why there’s a more questions than answers at this point, but we hope to find that out. We’re looking at the whole. The entire pattern of gene expression. Um and not just this one biomarker to try to get it that in some specific groups of patients with different outcomes. So you know just to follow up on that. So do we think that, uh, I mean,
likely the airway response.
NOTE Confidence: 0.908663034439087
It is before the subsequent
NOTE Confidence: 0.908663034439087
sort of larger immune response.
NOTE Confidence: 0.908663034439087
The airway response is likely
NOTE Confidence: 0.908663034439087
very different across ages.
NOTE Confidence: 0.908663034439087
And you think that could be one
NOTE Confidence: 0.908663034439087
of the major explanations why age
NOTE Confidence: 0.908663034439087
is such a strong predictor for
NOTE Confidence: 0.908663034439087
outcome in this illness. Possibly
NOTE Confidence: 0.879945635795593
possibly, I’d like to have the
NOTE Confidence: 0.879945635795593
data to answer you definitively,
NOTE Confidence: 0.879945635795593
so hopefully will have
NOTE Confidence: 0.879945635795593
that soon. Yeah, well,
NOTE Confidence: 0.879945635795593
it sounds like more to follow.
NOTE Confidence: 0.879945635795593
Well, channel and for two really superb
NOTE Confidence: 0.879945635795593
talks and the work that they do.
Thank you all for joining us today.
I know a lot of folks also watch online as we as the labs reopened but.
Enjoy the rest of your day and thank you all for your work.
Thank you very much.