But if you have additional questions, so let’s now turn to our second Speaker. Doctor Ellen foxman is assistant professor of Laboratory Medicine and Immunobiology and received her MD and PhD at Stanford. Her residency training in clinical pathology at Brigham and Women’s Hospital before coming to Yale and joining the faculty and Ellen is done. Extensive work now really understanding the immune responses and natural responses to respiratory viruses. Which is certainly a very timely topic of research.
In 2020?

So we were really pleased that Alan could take the time to share her research with us.

So Ellen, 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? All right? Uh. So can you see the slides? Yes, OK, great. OK, well everyone, I'm very happy to be here even though it's by zoom an be able to participate in my first Yale Cancer Center Grand rounds.

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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.

This is just a disclosure that I’m going to inventor on to patent applications. So today I’ll be talking about why are we interested in studying the early host responses against respiratory viruses,
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
serious illnesses such as Influenza with hospitalization or hospitalization for asthma attack or CEO PD Exacerbation which are very often caused by viruses and also there has been this emerging this lingering concern about emerging infections with good reason. As we know now and I usually put up this photo to describe that that's actually a picture of the SARS coronavirus from 2003. But now when we see these pictures it definitely conjures up something else in all of our minds,
which is the 2nd SARS Coronavirus SARS CoV2.

Uh, which causes the disease cobra,

19 and I just checked on the Johns

Portal an at the moment

there’s over 7 million cases and

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 tissue isn’t like the skin. It doesn’t constantly regenerate, but rather only when damage does it then regenerate, but it has the potential for these stem cells that you see here at the base of the epithelium to proliferate and recreate that issue. And one thing we’re interested in is how come that sometimes goes.
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
00:06:02.862 --> 00:06:04.888 the rest of the respiratory

00:06:04.888 --> 00:06:06.943 system from that that infectious

00:06:06.943 --> 00:06:09.066 agent getting down to the lungs.

00:06:09.066 --> 00:06:11.010 So when these offense defenses are

00:06:11.073 --> 00:06:13.575 effective in the upper respiratory tract,

00:06:13.580 --> 00:06:15.692 it can really be the difference

00:06:15.692 --> 00:06:17.620 between miles or asymptomatic illness.

00:06:17.620 --> 00:06:18.944 Versus a serious illness.

00:06:18.944 --> 00:06:20.930 And we know that that’s happening

00:06:20.990 --> 00:06:22.980 all the time, not just with SARS,

00:06:22.980 --> 00:06:23.610 Co V2,

00:06:23.610 --> 00:06:25.662 but other viruses that often there

00:06:25.662 --> 00:06:27.368 cleared from the become their

00:06:27.368 --> 00:06:29.279 detectable in a way for a time.

00:06:29.280 --> 00:06:30.144 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 and we will roll down the Hill one way, and it will roll down the Hill one way, one way, 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. So, uhm, 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, 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 and 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 detailed description of this stuff for one of the Deans workshops that's available online.
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 and if the answer is yes, it means a patient has the virus or the viral RNA and their nasopharynx right now 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, 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,

are 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 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 about it that we have much better techniques to look at.
Patterns of gene expression, patterns of protein expression using MultiPlex Technologies like transcriptomics and the idea is if a patient comes in and is coughing, you don’t know what’s causing that, but if the if that’s being caused by a respiratory virus that’s replicating, that’s activated, the immune system turned on antiviral defense is, which are different than defenses against an irritant or a bacteria or other things that cause coughing. And if you look at the patterns of Gene
and proteins that the body is making, you can sort of interrogate the body's 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,
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, which virus might be causing their

00:13:28.636 --> 00:13:30.832 respiratory symptoms and only about 1/3

00:13:30.832 --> 00:13:32.949 of them actually have a viral infection, so 2/3 of them may have some

00:13:32.949 --> 00:13:35.182 of them might be causing their

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 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 kc10. This is actually one of these interference stimulated jeans. It’s a cytokine. And 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, and then these bars indicate
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? the first one is 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
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, given the patterns that we see here, 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, 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 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 in the four cases. I hope you can see this,
but the four cases that.

uh,

we had picked up in those early weeks.

Kind of fit this pattern.

Three of the case is shown

They do a track most closely with North

American other isolates from North

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
00:18:41.106 --> 00:18:43.584 related were the same as the other,

NOTE Confidence: 0.895463764667511

00:18:43.590 --> 00:18:44.958 so this is independent

NOTE Confidence: 0.895463764667511

00:18:44.958 --> 00:18:45.984 introductions coming in,

NOTE Confidence: 0.895463764667511

00:18:45.990 --> 00:18:48.454 which was also probably says something about

NOTE Confidence: 0.895463764667511

00:18:48.454 --> 00:18:51.159 travel back and forth and things like that.

NOTE Confidence: 0.895463764667511

00:18:51.160 --> 00:18:52.972 So that was quite an interesting

NOTE Confidence: 0.895463764667511

00:18:52.972 --> 00:18:54.987 bonus of being a in collaboration

NOTE Confidence: 0.895463764667511

00:18:54.987 --> 00:18:56.837 with other folks at Yale.

NOTE Confidence: 0.895463764667511

00:18:56.840 --> 00:18:58.600 To find more information

NOTE Confidence: 0.895463764667511

00:18:58.600 --> 00:18:59.920 about those patients.

NOTE Confidence: 0.895463764667511

00:18:59.920 --> 00:19:00.256 Uhm,

NOTE Confidence: 0.895463764667511

00:19:00.256 --> 00:19:03.740 but we also had an idea just looking at this.

NOTE Confidence: 0.895463764667511

00:19:03.740 --> 00:19:05.124 Well this is interesting.

NOTE Confidence: 0.895463764667511

00:19:05.124 --> 00:19:06.854 Like here we used up,

NOTE Confidence: 0.895463764667511

00:19:06.860 --> 00:19:08.942 you know 376 PCR test to

NOTE Confidence: 0.895463764667511
00:19:08.942 --> 00:19:10.330 test all these patients.

00:19:10.330 --> 00:19:13.003 But really if we had only tested the 33

00:19:13.003 --> 00:19:15.540 that were positive for the biomarker,

00:19:15.540 --> 00:19:18.308 we still would have found all the cases.

00:19:18.310 --> 00:19:20.122 And so it suggested maybe this

00:19:20.122 --> 00:19:22.130 is a way of expanding,

00:19:22.130 --> 00:19:22.958 like conserving,

00:19:22.958 --> 00:19:24.614 testing capacity or directing

00:19:24.614 --> 00:19:26.946 it towards people who really are

00:19:26.946 --> 00:19:28.466 high suspicion to be positive

00:19:28.466 --> 00:19:30.847 and so we tried that so far just.

00:19:30.850 --> 00:19:31.831 Piloted one day.

00:19:31.831 --> 00:19:33.793 We picked one day in March

00:19:33.793 --> 00:19:36.143 where we were able to get all

00:19:36.143 --> 00:19:37.447 the residual samples from

00:19:37.523 --> 00:19:39.388 testing went 144 patients were
tested that day for SARS, CoV2.
And did the biomarker test and what you can see is again as a smaller proportion of people were positive than negative.
And then we compared this to the results from the PCR testing and it turned out that 17 people were PCR positive for SARS Kobe to that day.
And 16 of them were among the biomarker positive, but one wasn’t one was did not have the biomarker expressed, and that patient also happened to have a very low viral load, which is kind of something.
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 one patient out of 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. So, what about the correlation with the biomarker? So, 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.

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,
so we're still investigating this.

So we actually struck up a collaboration

including Tom Murray and Danielle

to delve into this further

and see if we can figure out what’s going on with this age correlation.

I so finally I just want to mention um,

what’s ahead for this project?

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 is 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. 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
00:23:55.928 --> 00:23:57.800 that actually tipped our hat.

00:23:57.800 --> 00:23:59.468 Let us know that seasonal Corona

00:23:59.468 --> 00:24:01.216 viruses are circulating in our patient

00:24:01.216 --> 00:24:04.876 has now added that to the clinical panel.

00:24:04.880 --> 00:24:06.488 So now that is those four

00:24:06.488 --> 00:24:07.990 viruses are on our panel,

00:24:07.990 --> 00:24:10.014 but this also as a proof of concept

00:24:10.014 --> 00:24:12.308 that our strategy works of picking up

00:24:12.308 --> 00:24:14.790 viral infections that we’re not testing for.

00:24:14.790 --> 00:24:15.340 Um, Interestingly,

00:24:15.340 --> 00:24:16.990 we also have half the samples

00:24:16.990 --> 00:24:17.930 where we didn’t.

00:24:17.930 --> 00:24:19.415 We still don’t know exact

00:24:19.415 --> 00:24:21.350 well for some of them we do,
but many of them we don’t know what 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 talked about diagnostic applications were also really interested in getting
insights into early stage pathogenesis.

And how this differs among people who have different outcomes.

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 saw with that before,
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 and 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 a lot. 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, Bhasa 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? Uhm, I think I made up some time. Uh, in in speaking a little quickly, but hopefully you’re able to follow.
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 on sort of the biomarkers is really interesting in terms of testing strategy,

and you mentioned that you're
anticipating one of my questions, which was, how does it change over the course of the illness? 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? Yeah, that’s very interesting people. There’s been some work already published about blood like cytokines in the blood that could be indicative of that we’re looking even earlier. I mean it at the early stage
of infection, the nasopharynx.

And that’s one reason why we’re really interested in this potential difference between adults and kids. Because, you know, kids are seem relatively protected from pulmonary disease compared to adults, older adults. So that’s one reason why we struck up this collaboration with Pediatrics to try to understand. Is there some difference in the robustness of that initial response that could you know that could possibly explain this? There’s many explanations,
00:28:08.260 --> 00:28:10.339 so that’s that’s the kind of thing
00:28:10.339 --> 00:28:12.448 we’re going to we’re looking into,
00:28:12.450 --> 00:28:14.697 but I don’t have the answer yet.
00:28:14.700 --> 00:28:17.902 This is it’s very rare to give a talk on a
00:28:17.902 --> 00:28:20.499 project that started like two months ago,
00:28:20.500 --> 00:28:22.796 but so that’s why there’s a more
00:28:22.796 --> 00:28:24.678 questions than answers at this point,
00:28:24.680 --> 00:28:26.934 but we hope to find that out.
00:28:26.940 --> 00:28:28.550 We’re looking at the whole.
00:28:28.550 --> 00:28:30.536 The entire pattern of gene expression.
00:28:30.540 --> 00:28:33.177 Um and not just this one biomarker to try
00:28:33.177 --> 00:28:35.973 to get it that in some specific groups
00:28:35.973 --> 00:28:38.350 of patients with different outcomes.
00:28:38.940 --> 00:28:41.604 So you know just to follow up on that.
00:28:41.610 --> 00:28:43.991 So do we think that, uh, I mean,
likely the airway response.

It is before the subsequent sort of larger immune response. The airway response is likely very different across ages. And you think that could be one of the major explanations why age is such a strong predictor for outcome in this illness. Possibly possibly, I’d like to have the data to answer you definitively, so hopefully will have that soon. Yeah, well, it sounds like more to follow. Well, channel and for two really superb 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.