



Webinar: The Science Behind COVID-19
The Lakshmi Mittal and Family South Asia Institute
Panelist Transcript
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Moderator

- **Dr. Jennifer Leaning**, *Professor of the Practice of Health and Human Rights, Harvard T.H. Chan School of Public Health*

Panelists

- **Dr. Caroline Buckee**, *Associate Professor of Epidemiology, Harvard T.H. Chan School of Public Health*
- **Dr. Sheila Jasanoff**, *Pforzheimer Professor of Science and Technology Studies, Harvard Kennedy School*
- **Dr. Victoria D'Souza**, *Professor of Molecular and Cellular Biology, Harvard University*

This panel will provide a deeper understanding of the science behind the COVID-19 virus. Panelists will explore the place of science in the COVID-19 response, as well as how to track transmission of the virus throughout South Asia through the use of mobile network data.

BEGIN TRANSCRIPTION:

Chelsea Ferrell: Hello and welcome to today's seminar on the Science Behind COVID-19, I'm Chelsea Ferrell, the Assistant Director of the Lakshmi Mittal and Family South Asia Institute at Harvard University. The mission of the institute is to engage through interdisciplinary research to advance and deepen the understanding of critical issues relevant to South Asia and its relationship to the world. As part of this engagement, the institute is running a series of stream on a number of topics related to COVID-19.

Before we get started we have a couple of housekeeping items for today. During the Question and Answer session, you can submit questions directly to moderators via the Q&A function on Zoom. Due to the large number of attendees at today's seminar, we unfortunately will not be able to cover all questions. There will be a short survey automatically sent to you at the end of this session. We would ask that you kindly fill this out. Finally, today's session will be recorded.

Without further ado, I would like to introduce the moderator of today's panel Dr. Jennifer Leaning. Dr. Leaning is Professor of the Practice of Health and Human Rights at Harvard T.H. Chan School of Public Health and Senior Fellow at the Harvard FXB Center. As Associate Professor of Medicine at Harvard Medical School, she is a faculty member at the Department of Emergency Medicine at Birmingham Women's Hospital. Dr. Leaning served as the Director of the Harvard FXB Center from 2010 to 2018. Prior to her appointment in 2010, she served for 5 years as co-director of the Harvard Humanitarian Initiative. Dr. Leaning's research interests focus on issues of public health and international law, in response to warning disaster, early warning for mass atrocities, and problems of human security in the context of force migration and conflict. She has field experience in assessment issues of public health, human rights, and international humanitarian law in a range of crisis situations. Dr. Leaning, thank you so much for being with us today.

Dr. Leaning: Thank you very much Chelsea, and a warm welcome to all of you in the audience. And, I would like to welcome you to a what I perceive to be a very important discussion from three scientists, with tremendously different expertise and it gives you a sense of how wide science could be and the extent to which their different zones of knowledge contribute to our understanding of the science that we need to understand when we consider COVID-19. I will start with introducing each of the panelist, and then I will turn to the first panelist in our set up and begin by asking her a question and then we'll proceed in that way. Each of our panelists will then respond to a question or begin with an opening observation. They will have somewhere around the order of about 10 minutes each of them to talk. I will then come back to them in reverse order and ask out sort of brief common question, have them respond to that, and then we will have sufficient time for responses to audience questions.

So, the first person we will be hearing from is Dr. Victoria D'Souza, she is Professor of Molecular and Cellular Biology at Harvard University and a Howard Hughes Medical Institute faculty scholar. She also serves as a Director for the Molecule Cells and Organisms Graduate Program at Harvard. Her lab is interested in RNA



Biology, and conducts research on RNA viruses, including retro viruses, like HIV, and coronaviruses, like SARS. Specifically, she is interested in how these viruses, their structures, their genomes, not only efficiently copy, but also how the structures interact with host cell machinery for viral production. When we say 'host cell machinery' here, in the setting of COVID-19, we're talking about how people get this virus and how they interact. So, Victoria, please if you could begin and tell us what you were interested in your own lab, what's so special about RNA viruses that makes it intellectually stimulating for you, and as I gather, a major focus for your career.

Dr. D'Souza: Sure, you know, I have been interested in RNA viruses for a very long time, since I was a grad student. What's fascination about these viruses are like they technically typically tend to be tiny, in terms of, we're talking about 10,000 nucleotides — do get a little bit bigger but still about a 30,000 nucleotides, and they are interacting with host cell that have millions of base pairs of DNA. The viruses are making, let's say, about 13 to 28 proteins and in the host cell you have many ends of these proteins, yet the virus is able to efficiently navigate that complex host cell, sometimes completely change what the host cell behavior is doing and manipulate it completely so it can reproduce and make a lot of itself. So this constant battle between host and viruses, I'm very extremely interested in that and just the complexity that can come from very tiny genomes and very little proteins and how they literally take over a massive system that has evolved billions of years, right?

The other thing fascinating about RNA viruses is that these viruses don't use their genomes just as carrying the genetic information, these genomes actually fold into three dimensional structures. So, if you've seen the images of the virus that is floating around on the media, you would have noticed spikes coming out of it, these are actually three-dimensional structures that the proteins are making. Similar structures will form just with the RNA and in many cases, RNA viruses will use these genomes to actually use this structures to go into the cell and manipulate the cell. So, if you take the example of COVID-19, for example, it comes with 30,000 nucleotides, and it's one big piece of genome. And the basic dogma in the cell that is infected, it says that, the cell says that if I have one message that's coming in then I can only make one protein out of that. That's what eukaryotic cells typically do, and yet this virus is coming up with one message inside the cell that's telling the cell to make 28 different proteins. Something like that really fascinates me because it's telling the system to actually behave a little bit differently for itself.

And, one of the processes that we're very interested in is if you think about the most important enzyme that the RNA virus needs is sort of copying its genomes to make many, many copies of itself, is its most important — about 2/3rd of the genome it uses to make — case, and the most important protein there is the polymerase that's copied the genome. And the only way this polymerase can be made is if the virus, genome that's coming in is somehow telling the cellular machinery inside the cells that take a message and sort of in three unit cores are making proteins. And what this message, with the help of the structure, is telling the virus to do is after you reach a certain point in this message, stop making the protein like you're supposed to make and go back one nucleotide, so that now the entire message is in a different frame, so you're making a different kind of protein. So, if you think about this sort of, this is called frame shifting. This is a mechanism called frame shifting, we're very much interested in this, and if you think about if your cells were, if the proteins machinery in the cells were frame shifting at that frequency that the virus is making the host cell do, we wouldn't know evolution as we know it, because you just would be making nonsensical protein in your host cells.

Yet those genome structures are coming in and telling this machinery to precisely shift back one nucleotide so I can make my polymerase. So that's the kind of science that is very basic, and it's more to do with once the virus enters the cell. But of course, there is a whole lot of biology that goes before the virus enters the cell. And, my lab particularly doesn't look at it but I've become fascinated also just in the terms of being sort of a structural virologist, how do cells recognize the host surface that they want to go into. In terms of, like I said, if you've seen virus pictures, there are things poking out of the cells of the virus that are called spike proteins, and this protein can then look at a host cell and find a receptor that it can bind to so that the virus can enter the cell and that receptor is the angiotensin converting an enzyme receptor, we've been able to record time, identify what a receptor is and we know a lot about COVID biology in a very short time because the way the science has taken the issue very seriously, and we know a lot about what cells it is binding to.

There was a very recent study that just came out a couple of days ago that looked exactly the two receptors you need for the virus to enter the cell and where these are, and it's clear that some of the cells were going to be in the lungs, that was obvious. And, the study has now found that cells in your nasal pathway that are



making mucuses, those cells are also harboring these receptors, so that could be the first mode of entry. There are cells in your intestine that are also harboring these receptors, so in the end I'll take any questions you guys have, but to think about viral biology has been pretty complicated, right now what the world is listening to is more as to how the virus looks on the outside, how it's gaining entry to go into the particular host cells, but once it enters the cell there is a whole other magic going on there which is going to be really interesting to study for COVID-19.

Dr. Leaning: Thank you, that's fascinating. You brought us back to the clinical picture, which people reading the newspaper are beginning to understand, the nose, the lungs, and some of the GI symptoms that people have and how this little beast of a virus that is so complicated and so powerful, how it is actually approaching these tissues and creating illness. So, thank you for that. Okay, we'll come back.

Our next speaker is Professor Caroline Buckee. Professor Buckee joined Harvard School of Public Health in the summer of 2010 as an Assistant Professor of Epidemiology and was promoted to Associate Professor in 2017. In 2013, Dr. Buckee was named the Associate Director of the Center for Communicable Disease Dynamics. The Buckee lab uses mathematical models and data science to understand the mechanisms driving the spread of infectious diseases, with a focus on pathogens like malaria that affect vulnerable populations in low-income countries. She has worked on this modeling and data science in a number of parts of the world, including India, and other parts of South Asia. And, what, there are many questions I could ask you Caroline, but let me begin with one. Could you explain why social mobility and migration generally are so critical to understand the epidemiology of transmittable disease while also speaking to the notions of crowds and diseases because your models are aggregate ones and yet you have many insights you can derive from those models about what is actually happening on the ground.

Dr. Buckee: Um, yes, thanks for having me. So, um, just like Victoria studies how viruses get between cells, infectious disease epidemiologists study how pathogens spread between people and through populations. And a lot of the work that we do in my lab is actually working with policymakers on questions of resource allocations. So, where should I be sending my drugs, or bed nets in the case of malaria, which populations need them, when to expect outbreaks, for example, of dengue and other pathogens. And, so, those questions are fundamentally spatial in nature, so you have to know where all of these diseases are, and where populations at risk are. And, so, I think the human mobility drives a lot of the spatial patterns of disease spread, and so a lot of the work that we have been doing is trying to understand human migration patterns that are relevant for the spread of infectious diseases using different kinds of data.

Often, the mobility patterns are the piece of the puzzle that's missing when you're trying to understand the risk of imported infections and likelihood that the disease would spread from between populations. So, for a long time we have been working with mobile phone companies to use aggregated mobility data to understand those patterns of the population moving around and we've been working for a long a long time to make sure those data are aggregated and they conform to privacy protocols so that there's no possibility of reidentification of individuals and the data is safe, which I should say is a big distinction between those kinds of analyses, and for example, contact tracing apps where individual level data is the name of the game. We're dealing with aggregated data on a population level to try to understand broad patterns of movement between places and of course things like seasonal migration and mobility are very important for infectious disease outbreaks, and indeed we've seen in this outbreak that travel for the New Year spread the disease significantly, leading up to the major outbreak in Wuhan. And, we will continue to see those types of large scale movement's impact this spread of diseases in general, and COVID-19 in particular.

Of course, Jennifer, that's where we've overlapped because large-scale population movements are important for other kinds of natural disasters and how to provide aid to people, how to know where everybody is. Um, I think that there is an important distinction between endemic infectious diseases like malaria versus emerging infectious diseases like COVID-19. So, in the context of an endemic pathogen where we have diagnostics, we have treatment, we have prevention options, when we're using these kinds of approaches, we're asking the question okay, but where do I send my bed nets, I already know what to do, and I already know how to monitor the number of infections that are happening in my population, where do I, you know how do I act. In the case of an emerging infectious disease, about which we don't know very much and I totally agree with Victoria that there has been an incredible amount of science done in a very short amount of time to understand what this virus is, how it's related to other coronaviruses, and so on.



But still, in the early days of the outbreak, there were many, many uncertainties about the basic epidemiological parameters of this pathogen, and that meant that a lot of the efforts to model how it might spread and what we might do about it were riddled with uncertainties that were really basic science uncertainties, things like how long does it take from getting the disease in my system to having symptoms and how can I spread the disease to other people in that time-period before I know I am infected. A major uncertainty is how many people have the infection and they have no symptoms at all, and so they are spreading the disease in the community and we don't know about them. Um, some of these basic parameters have made it very difficult to assess what is going to happen with the spread. And, of course we have seen that it spread very quickly first out of the central hotspots of the epidemic through international travel to our major cities, and then subsequently it spread dramatically within communities. And, of course there is a very strong risk associated with elderly populations and other underlying comorbidities.

Again, those epidemiological features we knew early on but we're still refining what the overall fatality is going to be, how dangerous this virus is. And again, that will help us with our planning. In the early stages of this outbreak, in the absence of drugs, in the absence of a vaccine, which we won't have for a long time, a year, a year and a half. Um, one of the only responses that you can have to try and control the spread of the disease like this that's directly transmitted between people is using non-pharmaceutical interventions and the world has largely adopted social distancing or physical distancing interventions to try to slow down the spread of the disease. So, shelter in place interventions, closing schools, stopping large gatherings, these kinds of approaches are some of the only ways to stop the epidemic from spreading out of control and overwhelming our health system and causing a huge amount of mortality.

Now, that links back to my previous work, because of course, what that means is that people stop traveling around as much, and in some cases, travel restrictions have also been imposed, quite severe ones to try to limit spread of the virus. So, one of the issues with that is it's very hard to measure the efficacy of those interventions in the absence of data. Uh, and so the approaches that we've taken in the past using aggregated mobile phone data to look at, you know, general patterns of spread of mobility that you can link to the spread of disease. We have started to do the same thing here, where we're just monitoring how much people are traveling in the communities on quite a core spatial scale and that's going to provide us not only with one of the only ways to measure what's working and you know how much reduction in mobility do we need in order to prevent how much transmission of the virus. But also, as societies get through this very first part of the first wave of the epidemic, and start to ask how to open up at least a little bit so that people can retain their opportunities for livelihoods, and feeding their families so on, we need to be able to monitor what's going on in response, and if we don't have that data linking the intervention to the number of people that end up in the hospital then we won't be able to do that in an evidence-based fashion.

So, we've been trying to kind of use those data in a way that informs, can inform policy and help us think through what's going to work because we will have to, we will potentially have to start opening up in advance of any pharmaceutical interventions, which poses a very big risk. If we look back at the 1918 influenza pandemic that killed so many, in many cases when places started to think that it was safe, there was an enormous resurgence in a massive second wave, which in some cases killed many more people than the first wave. So, we really need to be very careful when we're thinking about how to implement non-pharmaceutical interventions, try and gather data that we can, we can systematically link to the epidemiology and then make sensible decisions. And those decisions, I think from the epidemiological perspective, we've seen epidemics, um, the trajectory of epidemics is very different in different places, these are local problems, and so the local context is very important when we try to think through what types of interventions we might need, different areas are at different stages of epidemic, some are just beginning, some are way into the first wave, um, and the decision about how you combine different interventions in local context is going to be challenging because the epidemiology itself, the contact rate that underlie the spread of disease are going to be very dependent on the place that you're talking about.

So, I'll stop there just to say that as we, as we move ahead and think about how interventions should be informed by data and by models, it's really key that we recognize that although the science is going really fast, there's still a lot of uncertainty, and although we have to make decisions with incomplete data, it's important to keep in mind that there's still basic parameters that we don't know, and we're going to have to adapt in our interventions and planning.

Dr. Leaning: So, thank you. The two of you have raised a number of questions in my mind but I will hold them and then we'll come back and have a discussion after I've had the chance to introduce Professor Jasa-



noff and then we'll come back as a group, okay? So thank you, Caroline. So, our next speaker is Dr. Sheila Jasanoff, she is the Pforzheimer Professor of Science and Technology Studies at the Harvard Kennedy School. A pioneer in her field, she has authored more than a 120 articles and chapters and is author or editor of more than 15 books, including *The Fifth Branch*, *Science at the Bar*, *Designs on Nature*, and *The Ethics of Invention*. Her work explores the role of science and technology in the law, politics and policy of modern democracies. She founded and directs the program on science technology at Harvard.

Previously, she was founding chair of the program at the Department of Cornell, she set up that program in Cornell. She has held distinguished visiting appointments at leading universities in Europe, Asia, Australia, and the US. And, I have sat in some of her lectures and courses, and consider her an abundantly resourceful intellect on a wide range of topics. So, Sheila, would you like to begin and talk about your work and how it relates to this discussion of COVID-19. I know that's a general question but please take it from there.

Dr. Jasanoff: Thank you, Jennifer. I thought you were going to point to something a bit more specific in my work but that's an open-ended entry point. First of all, let me begin by thanking the Mittal Institute and to you for hosting it and it's a pleasure to be in this rather gender unbalanced panel, talking about science. That too is a kind of first of kind for my appearances on this subject. So, Victoria and Caroline have spoken about the ways in which existing scientific capability has been energized and revved up and a lot has been learnt very quickly about the virus on the one hand, and its behaviors, and about transmission, and how it relates to human behaviors, and in particular mobility.

One of the things that we consider in my field that looks at science and technology in society is sort of third piece of the transmission. So, not just, you know, what happens at the protein structural level and not what happens when a complex, invasive agent is fed into human societies that have their own dynamics, but when all of that is fed into a set of institutions and social response mechanisms that ultimately decide what we're actually going to do. I mean we as human beings with our particular limitations of knowledge and understanding about particular needs, whether it's to go out, or exercise, or eat or whatever. Where are we going to get our advise and who's going to tell us what to do. So, one of the things that Victoria and Caroline have both stressed is that, in a way, uncertainty, non-knowledge is the flip side of knowledge. That you, it's almost like you enlarge the surface of the sphere that is knowledge but the aura or penumbra of ignorance increases as you get a bigger sphere with more things that are unknown and unasked. And, yet we have very specific needs to, you know, as my colleagues at the Kennedy School say, we have to get up Monday morning and make a decision. A decision is very specific, it's very particular, you tell people what to do and to some degree I feel enormous sympathy for today's public servants who are having to guard us on simple-minded things. I mean, like you take your newspaper and it suddenly become like a medical advisory system, you know what are your FAQs, can you go outside, should you wear a mask? But there are translation agents for what is happening in the political realm.

So, it becomes a serious concern for the social sciences, how does the knowledge that gets produced in the basic science lab or in more of a clinical field like epidemiology, how does that find its way into the minds of decision-makers who then have to come back and tell us, you know, how we should be thinking about certain things. There was a very simple model abroad at the turn of the, sort of at the mid-century, about how science relates to politics, and the sort of, little formulaic way of capturing that was speaking truth to power. So the idea was that scientists, their responsibility is to find out the truth, and to some extent their moral ethical responsibility is to speak the truth.

Two different things: finding it and speaking it, but the idea was that power sits somewhere separate and then power exercises itself by making policy, by making prescriptions, and telling people what to do. Well, those waters have been usefully muddled but I think also complicated the muddled over the seven decades since 1950ish when this idea of speaking truth to power was prevalent in the world. For one thing, just because we know a lot more about a lot more, we've also discovered a cortisol that we also know a lot less about a lot more and therefore making decisions in uncertainties, making decisions in complex systems, these things have arisen as bigger problems for society. And although we have more powerful tools like what Caroline already mentioned data science and computer modeling, nevertheless, those tools that are good at dealing with massive invasions of data don't necessarily work equally well at producing clear-cut prescriptions for how we go about doing things.

So, my work has focused for a long time in an area of the social sciences that's devoted to studying how knowledge waking intersects with political practice and political response. And one of the things that is strik-



ing at the moment, particularly in the United States is a kind of gap that was not supposed to happen here. So, culturally speaking, America is a country that has been especially devoted to science-driven policy making. I do a lot of comparative work across countries, and one of the things one finds is that where other people have political controversies, political parties fighting each other, or social movements, we do that on scientific territory.

So if you take something like many people around the world are opposed to nuclear power, for legitimate reasons because they think that nuclear waste has not sufficiently been dealt with or they are worried about, in essence, having reactors that are protonuclear bombs planted in their backyards and they don't know how these things are going to behave. So, we have had nuclear mobilization across the world, but it's taken very, very different forms, only in this country, only in America, have we had the kind of protracted spectacle for over now 60-70 years of scientific controversy over particular solutions. So like, should we bury nuclear wastes at Yucca Mountain. Some people call Yucca Mountain the most expensive piece of real estate in the world just because of the amount of money we have invested in trying to learn the science of the geology in which people are going to bury those waste. So that is not about coronavirus, but it is relevant as a piece of history that in America, we have a tendency to translate political conflict into conflicts of our data, and political polarization gets reflected in controversies around science.

Nevertheless, for such a science-hungry, science-loving, science-admiring country, it is striking the extent of which rifts have opened up between public health authorities and political authorities in this country. Maybe it reflects a little bit what Caroline was talking about, that is, the extreme difference in the nature of the transmission and the spread as they occur in different localities and different local circumstances. So, across the world today, of course, if we look at the case fatality rate for COVID, if we look at the mortality figures, why is it that Italy and German, which are fairly close together and all members of the European Union, have such drastic differences, such that, even though orders of magnitude they are at the same numbers of recorded cases of COVID infections in both countries, well more in Italy, but it's not significantly more but the number of deaths is four to five times higher in Italy than it is in Germany.

So, what is it that accounts for these differences. Now, that set of questions, which might be one for epidemiologists, has enormous repercussions in the route. So that whose model are you going to believe, at what level of government should the governance be occurring, whose TV shows are you going to click on to to find the right facts, is it going to be the President of the United States, or is it going to be the Governor of New York, or for that matter is it going to be the Mayor of New York. And, we're in a situation where in such an information-rich world where we supposedly have everything at our fingertips, so, the decision who you click on to or who you key on to gives you a very different picture of the world. And, I think that for the social sciences in this moment, it is absolutely key and crucial to understand where those differences arise from and what we're going to do about them because without a much closer kind of partnership between the basic knowledge generators about the way that the physical systems and the physical entities are behaving and moving and the social sciences that say something about our institutions and their capacities, and people's own willingness to believe in those institutions, we are not going to get the right public health solutions.

Now, I do think and I'll just say a couple of words about this and stop in this first round that we know a lot. I mean, just like Victoria was stressing, we already know a lot about viral behaviors, and it is meaningful to ask the question, you know, sort of like the passover question why is this night different from other nights, you can, you know, also ask why is this virus different from all other viruses and you can ask why is the set of responses to this virus so different from responses to other crisis. But it also opens up a comparative terrain, and we can look back at other crisis and say these things generically have not worked and these things generically have worked. So one point that I would make and stress is that the post hoc analysis of crisis always, and I think this is almost axiomatic, always shows that things were known about the likelihood of such a crisis and even how such a crisis would occur.

So, after the Challenger disaster, it became known that we knew how that defective O-ring would behave under certain kinds of weather conditions, like freezing point temperatures. So, the question becomes not 'Did we know or Didn't we know' but why is what we knew not transmitted to the places where we act and that opens up a whole set of questions in the social sciences that I'm happy to come back to. But, one another, sort of, corollary thing to point out is that how knowledge works its way through institutional structures and political systems is extremely culturally specific. So, you can't just say as a universal matter, we know this fact therefore people are going to behave in response to this fact as if they are rationally programed



computers or robots. I mean that is not the way things work. The knowledge gets filtered through particular cultural ways of understanding sometimes that make some claims credible and other claims not.

In my own work, I've referred to this property of political cultures as civic epistemologies, that is, I think we as publics have, depending on which political systems we were brought up in, we have particular ways of understanding what the authorities tell us, particular ways of holding knowledge itself publicly accountable. And many of my Chinese students and colleagues have told me that they're more authority-trusting culture has produced less controversy about whether to take something at face value, is this kind of isolation, for instance, a legitimate thing, when the authorities tell us that this is the right answer, then we accept that. Um, we have a right now exceeding the polarized political culture, which coupled to this information-seeking society that we are, has tended to breed conspiracy theories, like where is this virus actually coming from and a tendency to feed into social divisions and fragmentations that we already have.

So, when we begin to pick up the pieces and try to think how do we become a wiser society, in the moment for sure, because we have a great deal of variation state by state in what people are doing, but we're talking about second waves and third waves. And, right now, there is a huge question whether we'll be any more adept at dealing with wave number 2 than we were at dealing with wave number 1 and it'll depend on the lessons we draw from this wave number 1 and predominant in that set of lessons is how do we actually act on knowledge. We may have the very best scientist producing the very best results, we will not have a wise society unless we have a reasoned way of responding to the knowledge that's coming out of all our incredibly intense and talented scientific enterprises. So that was all for the moment.

Dr. Leaning: No, Sheila, thank you. There's much to think about in what you've just raised, and we're getting some very good questions from the audience, so I want to reserve a bit of time for that. But I'm going to go around now, one more time, back to each of you and I will begin with Sheila this time and ask a follow-up question, what you've just said led quite directly into it. As you know, I've very interested in, from a disaster perspective, in decision-making under stress, and I would say we're here in the limelight of stress in terms of the consequence of policy-decisions on the large number of people affected and the uncertainties we have touched upon and may delve deeper into, but yet as you say at the Kennedy School and for disasters 'On Day X you have to make a decision.'

So, could you say something about the leaders of the government or state levels, we're getting pictures of how they made decisions and I had some thoughts about how they're gathering up data, how they're then communicating it to the general public. But could you say something about the issue of massive inertia that's involved here. We're talking about a governor of a state that has millions of people in it and he or she is going to say something that will affect millions of people, and it will take them some time to figure it out what they want to do, they'll be to-ing and fro-ing, then the decision is made and then a week later or maybe a new piece of scientific evidence that is essentially becoming understood and welcomed but it actually undermines the decision that was made.

In other words, a hesitancy to make a decision now, in my mind, as I observe major leaders, is that they have good data to some extent but what they are being asked to do will affect people who will then have difficulty turning around quickly, like a massive aircraft carrier. So this is always the case in decision-making under stress when you're dealing with public issues. But, could you comment from your perspective about what's particularly acute now, or does it seem to you part of a common process?

Dr. Jasanoff: No, I think you're pointing to obviously common and recurring thing. I mean, how do you make anything as complicated as a society turn on a dime. But actually, I would look at the present situation and say that it's an object lesson in how you can make society turn on a dime and I've called attention to the kind of debates that have been going on that I know you know about the on climate change and the sort of disasters that are confronting us as a result of not much more slow-moving crisis. If you look point by point at what people have said we need to do in response to climate change, there are things that we have put in place overnight in response to COVID, almost overnight. I mean so, fly less, eat more locally, consume less of all kinds of commodities, travel less overall, maintain a sense of communal responsibility toward each other. I mean these are prescriptions that could be taken out of the climate change playbook, and the answer to why we don't do it for climate change has always been well we couldn't.

And there's two sides to the coin: one is it would be economically disastrous, and the other is we as human beings and human societies could not. So, I think that the COVID case has thrown up a counter example,



previously thought a counterfactual to the second piece, we've obviously shown that we can. I mean, most of the, even the voluntary restrictions and you and I live in a state where the governor has not introduced a mandatory lockdown but voluntary guidelines. But compliance has been relatively good, so I saw one of these maps of a sort that I'm sure comes out of Caroline's work among others as the telephone and cellphone data being used to create a regional map of drops and mobility in different parts of the country, and I think that Massachusetts was, in one thing I saw early on, an A graded place because our mobility had actually reduced to the levels that were recommended by the public health specialists whereas other parts of the country like Wyoming where you can't do anything without getting in a car, obviously had very different patterns.

So, I think we've shown that people can turn on a dime if they think the risk is bad enough and in this country we particularly have been incredibly conscious of health risks. That's partly because we have not created the social welfare programs that put a floor under the people, and people are terrified of health problems we've left them unprotected and on their own. All these people who are losing their jobs now, they're also losing their healthcare, so we have, in some sense, a society that is very capable of turning on a dime but it's like they're still capable of turning on a dime because we have given them so little infrastructure in the past. Infrastructures that are sticky, not the individual behavior.

I think that your crisis scenario, as modified by this case, shows that individual behavior given high enough fear of threat can be modified, but it doesn't necessarily translate into institutions. It doesn't necessarily translate into the collective norms and the collective judgments by which we will end up guarding our lives once this particularly tragedy has passed. You know, I keep thinking as the scope of this thing as sort of Biblical and I keep thinking that maybe Egyptians had a coronavirus epidemic and when the hand of God passed over and took the firstborn of the Egyptians but not of the Jews maybe that was a coronavirus epidemic that has come down to us in those terms. But will there be a parting of the Red Sea, so that some sort of promised land is on the other side, and maybe one shouldn't carry oneself with the metaphors too far but I really do wonder whether the institutions have the wisdom to learn not that the individuals have the capacity to behave.

Dr. Leaning: Thank you, that's very interesting. So now I'm going to turn to Victoria and this again is going to be, I'm asking you to make a disposition on uncertainty in your own scientific endeavors. And, perhaps bringing up on a point that Sheila was raising, the psychological or emotional dynamics of your being at the closest proximity to the engine room that is this virus and knowing how much ways on what you determine is not just accurate but true. So, how does a scientist think about uncertainty, and a basic scientist think about uncertainty in this context?

Dr. D'Souza: Of course, I mean that's a question that most scientists are starting to get trained on to be very diligent. If you've thought about a lot of people that get published in really good journals also get retracted because there are certain mistakes — but there is always a way for science given time to self-correct, right. So, I will publish data based on my work in the lab with as much statistical analysis that I can do to say that these observations that I'm seeing have this much statistical significance value attached to it and that's how confident I am about my data. But, it's always put in the context like this is the best explanation I can come for what, how — given the data that I've seen. And, when scientists do get things wrong because it's still people working in the lab, sometimes we — agent from lab to lab don't behave the same given time though on a particular topic we always tend to self-correct, and the principles that come out that we finally all agree on are usually solid.

The problem with the pandemic like this is that what people are looking at more whole lot these days and what the policy is basing stuff on is what are things going to look based on the data that we currently have from different country and a lot of model building to that. And, model building unfortunately, you know, always an inbuilt consent, like the more data you have, the more you can predict your model. So, the point here is if you have a pandemic like situation, there is no time to do the rigorous science for every topic. Let's just take example of transmission, there are some studies that say these are liquid droplets certain size, yet some studies say they could be aerosols, which means that they can migrate more than the six-foot guidance that we are getting.

Given time, we will get the answer to this, but you need a lot of science to go in many different labs to agree to a certain point. And I think that's where we don't have that kind of time line that we are looking for. So, uncertainties will always be there, but that doesn't mean that each scientist is doing a bad job, it's just each

scientist's experiment is happening at different hospital and different ventilation systems just given the idea of the aerosols. So, like, uncertainties will be there, but the point is science is generally done in a rigorous enough fashion that eventually you get to the answer.

Now, certain things, however, like, if you think about what will get us out of the stickiest situation that we are a potential is a good vaccine in as quick a time as we can. For that you needed to know what are the receptors, what are structure of it, that's happened in record time, and that I would like to say is not an uncertain thing. That is what the receptor is because we'd already studied SARS which is close to COVID-19 and that happens to be the same receptor that people had studied for a long time in that, so it was logical that's what we would go on seeing is if that is the receptor for COVID-19, and of course it is because the two coronaviruses are closely very related.

So, I don't believe that there's an uncertainty in that observation because it's piggy-backing on certain things that we already know. So, I think, but to take things from one step of the science to the next step of the science, there are certain things that you have to be certain about and then move to the next route. Again, I want to iterate that the time that it took for the sequences to come out from the first time the virus was isolated, from the time we got to identify the receptors, from the time we actually know what cells are interacting with it. This used to happen in the time scale, like a year or two, we're doing it in like months. So, that is the beauty about it many people work together at the same topic then the timeline does reduce and we get to certainty from those uncertainties fairly quickly.

Dr. Leaning: Thank you. I mean this is a setting where the crisis is extreme and is felt by the experts, and in a very short amount of time you're going to have skilled members of a large crowd basically come together and resolve a lot of issues in relatively record time. No?

Dr. D'Souza: I totally agree with that. We haven't come to a, we've never faced a situation like this any time in the recent history that the current scientists are working on. What is good about, so a lot of the basic scientists are still sitting home, you know, we don't have the luxury of going and looking at this viruses right now. The people who are working are the one that are at the frontline, where they are directly looking at drug targeting, directly looking at vaccine development, where the research will bear fruit within six to twelve months, right. The rest of the basic science lab that can contribute right now cannot go to the lab to actually do the work.

What is good about viruses though right, what we've typically thought of looking at the viruses as a virology problem, like a person like a virologist studies this. But my contention, or like, people always thing about this, once the virus enters the cell, it is cell biology, it is interacting with the factors that are there in the cell. So, my gut feeling is, like, the scientist who contribute also to this problem is the thousands of great cell biologists that we have, and even if each lab just gives like 10 percent of their resources to like, to take that field of expertise and you know make the contribution to COVID-19, not change the entire lab structure, but put in the effort which they are already experts in.

A very good example is when the virus enters the cell, it does something different. Coronaviruses set up these large factories in the cytoplasm which look at the strange tabun membrane. So, it's a different, um, they're taking the host cell and they're organizing it differently. Now, I think the best people to study this is not like the virologists, like the cell biologists that were doing membrane biology in like other context. So my belief is that we will make progress on this really fast just because of the scale of the pandemic and you really need to understand these viruses really well really soon. Because if you think about 1918 pandemic, and you know, then we had a huge number of decades where there wasn't something so bad. If you look at just the last 20 years, we had SARS, we had MERS, now we have COVID-19, and maybe its deforestation, maybe it's just we're living with animals much more closer than we used to, so the cross-jumps, or the species jumps may be more and more likely.

So, the idea is that if we know that things like coronaviruses and flu viruses have the tendency to jump between species, we need to understand this thoroughly, completely so if a different strain comes out, and that's just a matter of time, I don't think COVID-19 is the end of, you know like, we're going to see things like this again, and the idea is can we set up a background trial with the whole world working together to work on novel ideas. Can we make vaccines broadly to all coronaviruses, is that even possible, things like that are going to be really critical. But my feeling is that scientists in general will just step up to the play, just given the scale of this.



Dr. Leaning: It's encouraging, very encouraging. And your enthusiasm actually comes through, how you are describing this, so it's great. Caroline, there have been a number of questions that have come up from the audience and they relate to what I was going to ask you as a follow-up question. So, perhaps I could begin and ask you about two of them or perhaps three of them. Is that okay?

So, I'm going to say these questions and then you think through because some are related. So, there are a relatively large number, at least the person I've seen the questions of is observing this, and at least we hear about the asymptomatic, but the question is very is there a very large number of asymptomatic people in this pandemic than perhaps we've seen in other viral epidemics in recent times.

First of all, is that your sense of how the curve of this virus is moving that is a relatively large number of asymptomatic and secondly, can you say a little bit more precisely what that means in terms of the challenges, you've said some of that but that's one question. And then the other is, can we, it's a bit related, how are your models, as you're evolving them now for this virus, how are they applicable to South Asia, let's say India, that's where I think the questioner comes from, where we don't have large nursing homes.

We do have in India large population clusters, for sure, there's now seasonal migration going back and forth and we have this problem of people being trapped halfway to go home, you know with Modi asking for a lockdown with relatively little warning. So, people are caught between do we stay some place, do we go some place and in the midst of this there's very little testing going on. And, so this concern about who is asymptomatic is going to come to the fore because you have no other ways of, you can try to protect yourself but you really don't really know what's going on. So, both countries and the world, but there's certain question about India and the United States, this issue of the asymptomatic person is very perplexing in terms of model and disease prevention. So, it's a large question but could you unpack it for us?

Dr. Buckee: So, the fraction of people that are asymptomatic, and by that I mean, you know, truly asymptomatic. You have no symptoms for the whole duration of your infection. That is one set of people who are potentially transmitting the virus that we need to measure. The other set of people that are pre-symptomatic, so not yet symptomatic but still able to transmit. We still don't know how much those categories of people are spreading the virus. We also don't know really what fraction of people have mild disease because they're not going to get picked up in clinics or hospitals because they won't get treated and not stay home. So, those are still very uncertain, one of the ways that we can start to measure that is through antibody test where you are looking for an immune response specific to the virus.

So, you're trying to say what is the cumulative incidents of this disease, how many people have had this infection so far, and that will help us to understand the denominator, so the number of people overall who have been infected. And that's very important for the models in terms of understanding where we are, now we also still don't know whether having antibodies to the virus means that you can't be infected again and you can't get sick again. That's a really key piece of basic science data that we need to be able to understand what it means if we open up, what it means, you know, how much should we be locking down all of these things relies on this basic science question, which is are you immune, are you protected against reinfection and disease if you've got antibodies to this virus. We don't know that, so, we don't know how your clinical severity, whether it's asymptomatic, mild infection relates to those categories of protection moving forward.

So, and again I want to draw distinction between the types of certainty that Victoria was describing. So these are biological realities that we can measure, okay, we might not be able to measure them yet but we will be able to measure them. So, when we talk about the randomized control trials, many dozens of randomized control trials for treatment and so on will happen for vaccines too. That's trying to ascertain a biological truth to some sense, right? Does it work, are you protected, what is the specificity? Those are biological facts. Models not, there is no correct model. Models are a way of asking 'what if'? So what if immunity is complete and we lockdown for this long, and we make this assumption about how people will behave, what will happen? And so the types of uncertainty that we are dealing with when it comes to modeling are very different from the uncertainty around the biological reality but rather, it's a way to think through all the possible spectrums of outcomes. So communicating that uncertainty and talking about what it means and why models are uncertain, it's a slightly different scientific question because model itself is just a way of thinking through what happens under a certain set of assumptions.



Now, as far as the lockdowns go, we've seen happening in multiple places, it is very often when a travel lockdown is announced, there's a massive surge of people that move out of the epicenter, from cities to rural areas, that's happened around the world. And, we've seen that as a social response to some of these interventions. We still don't know what that will do to the epidemic and whether it's a good idea or not. We can start to explore how that relates to the epidemic as it progresses and then estimate what that means but we still don't really know how to plan these interventions. With respect to the asymptomatics, because of the uncertainty of how much they actually infect other people, we also don't know whether some of the interventions are going to relax or going to have a big impact.

Again, I think what this speaks to is the need for the, so as Sheila was talking about, as we make this models built on the basic science that's happening and try to make policy decisions that are informed by these uncertainties, the question is a) In particular context how will people behave because that's going to connect to what happens with your epidemic regardless, and that's going to be very context specific, but then b) How adaptable is the system overall to monitor the possibility of a resurgence, so what are the surveillance systems in place and that relies on testing, if you don't have testing capacity, you're going to have trouble making sense of whether a resurgence is happening and where. But how adaptable is the system to be able to respond when resurgence does start to happen. Can we have a quick enough feedback loop between the data, the surveillance system, the modeling and the policy makers to pivot quickly.

We spoke about the fact that people can change their behavior quickly, my concern is that with all of the uncertainties and the fact that the models are constantly being redefined, there's going to be another context-specific piece which is how well can we flexibly respond to changing parameters because there isn't going to be a model will go with that model and we do a bunch of policies around it. That's now how modeling works and that's not how humans work. So, we have to be flexible, and the extent to which we can be flexible depends on testing and how well we can feed back the information that we're getting from the refinement of the model based through basic science to policy makers. So, I don't know if that addresses the question but I think it's a difficult and broad type of question.

Dr. Leaning: I think it addresses it very well. There's a question that I will ask you and then ask Victoria because it's quite simple question but it's complicated answer. There's the observation that this virus has spread very rapidly, and the question is, is its rapid spread a result of biological characteristics of the virus, and I would say, the virus-host interaction or is it a function of how societies operate, and societies operate quite differently, of course. India and the United States might be good ones to refer to. I think it's \quite important question, so get ready Victoria, I will ask it to you but Caroline first.

Dr. Buckee: So, I would say that this virus spreads fast but it's a virus like other viruses, and so, for example, the reproduction number which you probably heard about in the media, you know, that's how much, how many onward infections we expect from a single person infected. That's within the range of viruses that we know even though it is quite transmissible. Some of these parameters like being able to spread before you're symptomatic that makes it particularly hard to contain. As far as the asymptomatic piece, actually many infections cause asymptomatic. So again, although it's a kind of unlucky combination of factors here, these are not unknown and unheard of in terms of the epidemiology. With respect to how fast it spreads, I would say it is both biological and tightly linked to the population.

So, if you think of the spread of the disease through a population like a fire spreading through a forest, you know, the density, crowding of places, allowing a virus to spread efficiently, that's a huge factor in how it's going to spread. And, in fact that's why these mad dive physical distancing interventions work because you're chaining the social structure, not the biology of the virus. So, it's both things, and I think this virus is not necessarily unique but it has a combination of factors that make it able to spread very quickly through populations. And, in our globally connected world and our very dense populations, we have a perfect storm for a pandemic like this. In fact, epidemiologists have known that this could happen for many years, and we've been trying to create pandemic preparedness protocols and so on but generally speaking we've been reactive in the past rather than preventative.

So, that's what I would say, I think it's both biological and social of course. And, the hope is that we can tame the biology a little and identify treatments and vaccine that work so that the social interventions that we've put into place can be relaxed a little bit in an evidence-based way.



Dr. Leaning: Thank you. Great. Victoria, what is it about this virus that, I mean, we are naive, human beings are naive to this virus, and so that's one way of starting this answer as to why it's biologically so potent. There's another question, two other questions have come up related to, what about mutations into this virus and how might that short-circuit vaccine development or not. So if you could ponder on those?

Dr. D'Souza: Yeah, I mean, I think Carol really did a wonderful job in explaining what's different, if there's anything different. I think it's just an unlucky combination like Caroline said. You know the only reason I think SARS was easily contained, it still took a year to contain the whole thing but people got sick unlike this virus where some people are not getting that sick, they can be spreaders. But with the first SARS infection, people got really sick which means that they went to the hospital and that is quarantine, right. That is automatic, built-in quarantine, so you don't spread as much. So the possibility of curtailing it is easier.

Other than that, I don't think the biology itself is that different, we are just naive to a slightly different set of combinations or the spike protein then, you know that was there in like SARS. The way the human body is interacting with it where there is such a discrepancy in the way each individual is behaving to this is what makes it that difficult. And exactly why, why different populations, why people with hypertension, why people with diabetes are most susceptible. All these are basic science questions that will be answered in a decent amount of time. The concerns with the vaccine are, the good news about, you know, coronaviruses is, it's not like we don't know anything about coronaviruses.

We have four coronaviruses that circulate among humans but they're not a nuisance, they cause common cold, seasonal common cold and there are some patients that do get pneumonia etc. but the mortality rate and the effect of these coronaviruses are mild, we don't take them seriously. We could have taken SARS seriously and there was already a vaccine in Phase I but as soon as the virus got contained, we stopped looking for the vaccine anymore because vaccine development is incredibly an expensive business. And, so, you know, we had an opportunity and we didn't follow it through.

But the good news about coronaviruses like they do mount an antibody response, which means that you could make a vaccine for it. It's a very promising candidate to make vaccine but as we know like vaccines in some viruses or like vaccines some they give you immunity for a lifetime, some give immunity for 10 years, then you need to take boosters. And we know a bit, like flu, it mutates so much every year that these are seasonal viruses that you have to constantly reengineer your vaccine and take it every year.

So people have actually started to look at the mutation rate, there are studies that have looked at, you know, different strains, I mean different seasons in Spain, Italy, Switzerland, US, and looking at what the mutation rate for COVID-19 is, it's not that high compared to flu. And, there's a reason for that — coronaviruses were always strange in the case like for season this family of viruses have been able to break through the size barrier of how large can an RNA virus get, these viruses for some reason have become really big. And the one reason for that is the enzyme that makes or copies like the genome over and over, RNA viruses have a very sloppy enzyme that means they make a lot of mutations. These viruses as part of the coronaviruses have come up with a way to have a proofreading function. They have come up with an extra piece of protein which is in exonuclease, which if an enzyme is making a mutation, this piece will come in and take the mutation out and put the correct thing in.

These are the only family of viruses in which this proofreading functions. So that was in our favor, in the sense that there's a proofreading function and people have looked at based on the different sequences out there, and they feel like you're going to get about half the mutation rate that you would get typically from a flu, given the amount of mutations per year that will come up and depending on the size when you compare the flu verses the coronavirus. I don't think we're looking at a vaccine like the flu, that you'll have to take every year because it mutates so much, but like what Caroline said, coronaviruses, the reason they are seasonal, that is the ones that are sort of entrenched itself with like human biology is that they come season they give you the common cold.

What that means is if this eventually mutates to being like a coronavirus thing, it's most likely not going to be something that's going to keep your immunity up for decades. This is at the max you're talking about, maybe 18 months to like two years. And again, these are just numbers based on other things that we have seen but certainly nobody knows. And we have to go and lab would do the exact, exact test.



Dr. Leaning: Thank you. Thank you, very much. There are a couple of questions that I will encapsulate into one and ask Sheila, if you could answer. We're approaching the time where we'll have to say goodbye and I will have to probably if there's time ask each of you to say something short at the end. But let's take this one question, and it's um, maybe the one that Caroline and Victoria want to comment on but Sheila, there's a question, there are several questions about what is going to happen, what do you think will happen if there is a second wave and a third wave?

Are there going to be policy issues that will make a second or third wave more likely but regardless we're going to have to at some point begin people move out of social distancing and self-quarantine into the work space, and there'll be a gradual process for that probably, or it will be somewhat hard to control, we don't know and even in this country but elsewhere. There is a big concern that is already facing policy makers as well as scientists is the large number of people who have not been exposed as far as we know and this gets back to who is asymptomatic all the way through but the people who will be susceptible to say come out.

And, I wouldn't ask you to predict whether there'll be a second wave or third wave but there's quite likely to be another wave because you can see it's starting a little bit in China and Singapore that they're both seeing a resurgence as they relax social mobility issues. What do you think about that from your perspective of policy, human behavior, messaging, risk communication. It's a very hard question, but do you have thoughts on it?

Dr. Jasanoff: I mean it's hard at some levels and easy at others. If we're taking epidemiology as guide then the chances that a pandemic has a second wave are quite high. So, one might actually say that one can be a little bit predictive. The Spanish Flu gave us at least two pretty sizable waves, so, I think the way it impinges on my work and on the social sciences is on the side of learning and uptake institutional response, collective behavior, those kinds of things. So, it's not so much will there be a second wave which as you said you weren't going to ask me anyway but I think one should plan for it. But what are the ways in which we even internalize what the lessons are and how do we walk away with them?

Now, as you know even in our own employer institution which boasts as we've seen some of the best basic knowledge, institutional response is completely up in the air. I mean that is, how should we, Caroline's been talking about modeling but let's remember that modeling goes deep, deep, deep into society. We operate with models, I mean those of us who have partners or children or whatever, close family members or friends, we have models of who they are and we have a kind of instinct for what load they will bear, we even decide whether to talk to them about the truth or not the truth, depending on what kind of personalities. I mean, so we're operating on models all the time, right. So, Harvard University is operating on a set of models and, you know, as you know pretty well that's completely uncertain.

Are we to model ourselves as a cruise ship, are we to model ourselves as a group with herd immunity, are we to model ourselves as relatively home safe because we're relatively youthful population? Should we, for instance, take our most senior faculty and tell them that the way to prevent a bad case fatality rate is by taking out everybody over 65 and saying that they're furloughed for a semester and the kids can be back and do their things and the people who are most likely to get hurt won't be there. All this depends on normatively what model do we think is right and this is a dimension I don't think we have touched on enough that it's one thing to say there are uncertainties, there are different ways you can model things, some model may be useful or not useful but there's a great deal of uncertainty.

But, against that backdrop, every choice we make is a set of moral choices as well. How many people do we put in danger, is it in fact okay to say, you know, the economy is worth more than a small handful or a large handful of lives over 70. I mean these are the questions that are already out there in the public sphere. One of the things that I would maintain is that there is no point, no matter how deeply you get into a scientific side of an evidence based policy so called where you wash out the norms that it evaluates all the way down. So, I think that part of the answer to your question how do we behave in a second wave is how do we clarify the values that we're bringing today even on this first wave, and there are, just looking at our own institution as a microcosm, one answer is that we have to get very detailed, very detailed about the ethnography almost, the anthropology of behaviors and the who is heard and who is not question.

So for instance, we took an almost wartime mobilization decision across many universities in the country to send kids home. So, our response to a viral invasion was dissipate the population so that they are not going to turn into super spreaders or create super spreading environments. But what we didn't think about, and



what we now have a lot of background information on is what does it mean to send people home. The homes are not all equally equipped for receiving, for instance, remote education. Universities are a special case but the fact is that you could relate this to any social organization, you're used to working in a certain environment, you suddenly pull out that infrastructure.

We metaphorically talk about pulling the rug out from under people's feet. But what we've done is almost in a physical sense is pulled rugs out from under millions of millions, tens of millions of feet and so, for me, the second wave question which really has to do with pandemics in the future and collective behavior in the future is how do we reintegrate our moral, and ethical, and political learning of how to implement these large-scale policies based on inadequate knowledge, low previous feedback from the situations that we are in. How do we aggregate that, I think we're not going to aggregate that by data science, I think we're going to need a lot of the interpretive social sciences to come along and you know the humanistic sciences.

I think in terms of policy we're going to have to be recursive, we're going to have to be experimental, we're going to have to not have silver bullets type solutions because likely they will not work, we're going to have to think, I mean, I'll finish with just one thing I read in Le Monde yesterday that ten or twelve days ago Macron was saying we're not like Germany, we're not federal like they are, we're going to have a national solution, and then yesterday, he was saying no no no, we're going to have a regionalized solution. Well there was a turnaround, and Macron is being advised as we know by an expert advisory body which has very different characteristics from the British one. There was an article in the Times yesterday about how the British one is incredibly secretive to the point where you don't even know who the members are. And then you ask yourself who is the advisory committee that's advising the President of the United States.

Dr. Leaning: Well, that's another question isn't it? I think we should leave that one not the last word as a question or a topic area. But, I would like to say that we're hitting the last few minutes of this talk. You all know that this is going to be recorded and available for other audiences, including the one that we've had. I'd like to thank the audience for their wonderful questions and participation and I hope that you've found this to be an invigorating, thoughtful and oddly optimistic conversation. At least that's how I'm feeling in terms of the energy and acumen of the three of you that's involved in this understanding of this disastrous epidemic but also in commenting on it and guiding it to closure.

It comes to a sporadic set of closures, we all know. All of you are going to be in the frontline of either explaining or understanding or designing new insights on various levels of science and society as we approach this epidemic. We're talking about a second wave when it's already still completely perplexing many societies around the world, including ours, this first wave, with an enormous amount of grief and sorrow and loss that will be part of our consciousness, and our social expectations and social burden coming out of this one, this first wave. And then, having to let people know there will be a second, I think has scientific obligation but also moral and emotional responsibility so that people are prepared for what is going to be a long haul.

And, I just have to thank you, the three of you for your really brilliant discussions and the ways in which we have unpacked some of these topics of the science behind COVID-19, and we'll bring this session to a close, and thank the audience again, thanks to the Mittal Institute for sponsoring this and let's leave it at that okay. Thank you very much. Bye-bye.