

@ This Moment | COVID Conversations with Ken Stedman, Brooke Napier, Lynne Messer

- **Do you think we'll be back on campus by fall term** and what if so, what might that look like?
 - Ken: No idea, I hope so. It depends on lots of things. I suspect that big lectures (like many of mine) will not be happening.
 - LM: some classes probably. But agree with Ken - not all
- How does the **variation on response (i.e. no effect vrs. eventual death) aid the spread of a virus?**
 - Ken: Love to get Lynne's take on this, but, in my understanding, from an evolutionary perspective, killing off your host if you are dependent on a living host to replicate is probably a bad idea. Also the experience from SARS(1) was that it was controlled because transmission only happened from symptomatic individuals, which allowed them to be traced and isolated until the virus "died out". So probably fewer effects would help the spread of the virus. However, there might be some things, like inducing a cough for a respiratory virus that could increase spread. Rabies is probably spread better by rabid dogs because of behavioral changes that happen in the dog due to the virus infection.
 - LM: absolutely agree with Ken. Killing off host is a bad idea. Inducing behavior in a host that will aid the spread of disease is a smart plan.
- Dr. Kimberly Brady, a dermatologist, wrote a letter-to-the-editor to a newspaper in Douglas County, MO. The letter is entirely speculative and is comprised of only "what if" questions, raising doubts about the efficacy of vaccines. **Are all medical doctors reliable sources of information regarding viruses such as COVID-19 or should the public be more discerning of which medical doctors we regard as experts?**
 - Ken: As I think that we closed with "be skeptical", this is true for medical doctors, scientists, politicians, etc.. Do your best to analyze what this person's experience is with what they are discussing, and try to determine if they have any ulterior motives. Some of the more misleading things that I have seen online are from doctors, but some scientists as well. I would not like to get advice on trimming my trees from a doctor or an appendicitis from an arborist. It sounds like you are already doing a good job being skeptical and wanting to see data.
 - LM: Be skeptical. Look for multiple sources. If only one or two people are saying one thing but the bulk of the medical community (and CDC...and WHO) is saying another, I'd put more faith in the more consistent messaging, as opposed to the rogue messaging.
- In order to prevent future outbreaks, **will China have to clean up their act with the "wet" markets** and interactions between people and animals?

- Ken: The first (retrospectively diagnosed) cases of COVID-19 were not associated with the wet market in Wuhan and many of the first cases did not have a direct connection to the market. Interactions between humans and wildlife in their natural environments, particularly due to habitat degradation are probably equally likely to cause problems.
- This recent structure biology article showed that some viral proteins could bind to the porphyrin and attack heme on the 1-beta chain of hemoglobin, dissociating iron to form porphyrin. I am currently working on Gallium protoporphyrin (similar to hemoglobin except having Ga instead of Fe) effect on Coxiella which has been promising in certain other intracellular infections by inactivating Fe-containing enzymes. **Since I have very limited understanding of coronavirus, Can anyone explain the possible challenges/ongoing efforts, regarding nutritional immunity during viral infections?**
 - Ken: No idea.
- What **factors would cause some people to be asymptomatic** for COVID-19?
 - Ken: No idea. Asymptomatic is a difficult definition and very subjective.
 - LM: Differential vulnerability. Differential expression of disease.
- Is it very possible that the **second wave of covid-19 could be worse** than this wave?
 - Ken: Distinctly possible, yes, but we have “bought time” to get some things better taken care of, more PPE, hopefully better plans in place, more testing capacity, more drugs tested, etc..
 - LM: We also have some experience, now. It will likely be coinciding with the next flu season, so people’s immune systems may be already stressed. But maybe the seasonal flu will occur differently because we are all more careful about handwashing and cough / sneezing hygiene.
- RE: Lynne's comments, **what is the trajectory of this disease in Latin America and Africa?**
 - LM: COVID-19 is going to be especially devastating in parts of the world with high density and low levels of preventive capacity. So the parts of Africa or Latin America characterized by inadequate resources (hospitals, ppe, ventilators, soap, water, ability to physically distance) and high population density are likely to be very hard hit. COVID-19 may have a higher case-fatality rate in those parts of the world without the medical infrastructure to treat people who become infected. It will likely be grim.
- Do you **wash your vegetables or take out containers?** From Clare Quinn.
 - Ken: Not more or less than I did before for vegetables, I don’t bother with take out containers. Just wash my hands when I come in to my house from outside.
 - LM: Same as before for me too.
- I just wondered whether **it is the virus which causes the deaths or it is the host immune system**, as I came to know that the antiviral medicine Remdesivir shorten the

time to recover and at the same time the antiviral medicine Hydroxychloroquine which is also used to treat autoimmune disease is doing good.

- Brooke: Generally, it is the immune response to the virus that leads to severe respiratory failure and pneumonia, which are generally the causes of death in COVID-19 patients. Remdesivir has been shown to decrease recovery rate from 15 days (without drug) to 11 days (with drug). These are promising results, but clearly it's not the "wonder drug" people wanted it to be.
- Brooke: Hydroxychloroquine has been proven to be ineffective, and it has been shown that higher doses of this drug correlate with a significant increase in rates of death. These studies were done on small cohorts (<100 people), but mostly clinicians have pulled this drug from use on their patients because it is too dangerous and has not shown any improvement in patient outcome, except for the original study from France that published hydroxychloroquine might help COVID-19 patients, but this paper is frown-upon because they didn't use proper control subjects.
- What could we be doing as individuals to help out researchers as they work to find a cure/vaccine?
 - Ken: It really depends on your skill set. Something that everyone can do is to slow the spread of the disease by staying home and continuing to practice physical distance as much as possible. In fact I heard from an infectious disease doctor the other day that we are all heroes, because by practicing physical distancing we did "flatten the curve". Oregon has also been very good at this, better than most other states, from what I can tell.
- How can we as public health students respond when objective public health data become weaponized politically, when their objectivity is questioned, and when science itself is distrusted?
 - Ken: Great question! My personal approach is to be data and evidence driven, communicate a lot, and admit when you make a mistake (which I do often). There has been some quite good work on how to address anti-vaccine arguments, a lot of it has to do with identifying as much as possible with whomever you are talking to and to listen to them. One thing that I do as far as vaccines are concerned is to always mention that I vaccinate myself and my family.
- What are the features of this virus and the host immune response that lead to such individual variability in clinical manifestations and outcomes?
 - Ken: I'd love to hear Brooke's take on this, but I think that we are seeing a large variation in manifestations and outcomes mostly because so many people are infected. I think that the vast majority of manifestations are respiratory and outcomes are bad for people with "underlying conditions" (whatever that

means) and older people. But when you have so many people, you will see some “outliers”. We also need to bear in mind that we are probably missing many if not most cases, so things may change as we learn more. This virus was only discovered about 5 months ago and the number of cases of people who had similar diseases (MERS and SARS) were very low compared to this disease.

- How likely is it that SARS-CoV2 was circulating in communities prior to the first identified case?
 - Ken: Likely, see my answer above about wet markets, but probably not very widespread.

Questions asked and answered during the webinar:

- What are all your thoughts on **developing antibodies after exposure and the possibility of not having immunity** thus contracting it a second time?
- I’m interested in the wide **variety of clinical manifestations of COVID-19, which seem to be very population/host specific**. I.e. ARDS for older people and people with chronic disease, coagulopathies in healthier people ie cases of stroke in healthy folks in their 30s, “covid toes” in children, reports of toxic shock type responses in kids, cytokine storm?, loss of sense of taste and smell (neuro involvement?) versus totally mild symptoms or no symptoms. I’m imagining this has something to do with host response. Does Brooke have insight into the **wide ranging symptom picture that seems different across different types of people**?
- What is **one thing we have learned from this pandemic** that we can incorporate **to help us prepare for the next pandemic**?
- Some people are infected & have little effects such that we don’t know they are infected and possibly contagious. Other people get sick and recover. Other people get sick and die - even with excellent medical support. Why do we have these 3 or more different circumstances. Was the treatment & support different from those who quickly recovered hrs those who had a hard time & still recovered? (**Why is there such a huge spectrum of severity of illness?**)
- I have not read up much on the new journal articles that are coming up about COVID-19. But I did read about **ACE2 possibly being an entry point for SARS-CoV-2. This was related to the use of ibuprofen and other drugs that upregulate ACE2 and some news articles recommended avoiding those drugs. Can someone speak to this?** I was just curious of expert opinions.
- What is it about the COVID-19 virus that makes it so unknown about whether it will mutate, like the Flu virus does? Also, what is it about Flu virus and the COVID-19 virus that prevent people from being immune once they’ve had it, like measles or chicken

pox? (How is **COVID** different from the flu (does it mutate) and why aren't people **immune once recovered** like with measles and chickenpox?)

- I'm curious about the **development of the COVID-19 vaccine**. Can anyone speak to that - either info about the **timing** of it, or what goes into its **production**, etc?
- I heard that COVID-19 may be **spread as an airborne aerosol**, like SARS. Do you know if this is accurate?
- It's curious that SARS COVID1 is so "similar" to SARS COVID2 in terms of genetics/SPIKE/ACE2-binding/crossprotective antibodies etc...but that SARS COVID2 is more pathogenic...why do you think SARS COVID2 is so much more transmissible/pathogenic/or deadly?
- What do you see as a misunderstanding of this pandemic?