Academy of PHYSICAL MEDICINE

Virology

with Nicolas Locker

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TRANSCRIPT

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Steven Bruce
I'm joined by Professor Nic Locker. Nic joined us five months, ago right at the outset of the Coronavirus problem or close to it, I suppose, to talk about viruses generally, he is Professor of Biology at the University of Surrey. I went through a bit more of his bio last time to show just how expert he is in these things, but Nic, it's fantastic to have you back with us and to spare some of your time for us again. Perhaps you can tell us all the progress you've made towards a vaccine for COVID-19?

Nic Locker
Yes, good afternoon, it's my pleasure to be with you again this afternoon. So, in terms of vaccine, I'm afraid I haven't got any breakthrough news to report. I think we are part of one of the 180 different teams that are trying to develop their own vaccines at the moment. There are a lot of larger teams that have made tremendous progress over the past, I would say, nine months. And I think, for everybody listening, this is the positive that I would take at the minute, we currently have nine vaccines that are in phase three of clinical trials. So, this is basically the last stage collecting and gathering data before applying for regulatory approval. And if you think about it, I know we're all desperate for a vaccine, I am desperate for a vaccine, you know whether it comes from Surrey or whether it comes from somewhere in the US or somewhere else in the world. But if we reflect on what would be normal timelines, it takes about 10 to 15 years to develop a vaccine for a common microbial pathogen. And this is because there is a lot of preclinical work that has to go into the design of the vaccination, of the vaccine itself. And what has been allowed with the COVID-19 outbreak is to focus really an unprecedented amount of resources in trying to speed up those preclinical studies and to push bolder ideas faster. And what it means is that teams across the world have been able to, instead of taking a staggered approach where you would do the first phase of trials, then the second phase and the third phase and then ask for the regulatory approval, everything has kind of started in parallel. Of course, we've all the appropriate safety measures in place, but really to minimise the delay and to minimise the amount of time it takes to move from one stage to the next. And this is why we're in that situation today where we have companies and in the UK we seem to have a lot of publicity around the work done by Oxford and AstraZeneca because they are local to us, right. But there are nine vaccine projects that are basically in that final phase, pre-approval. I guess where we perhaps need to be a little bit careful is that, although this is very, very encouraging. You know, the reason why we've been able to progress quickly with those studies is that because they are focused on so far, a small amount of people, mainly people that are, so for the trials mainly people that are healthy and that are between 18 to 55 years old. So, we are in small cohorts up to 1000 people. So, though we have positive outlooks with these studies, of course, we need to know how mass vaccination could impact on population. And how it could impact for example, on the elderly, because at the moment we have really no idea on the safety of the vaccine for people that are in their slightly older years, and for our children as well. So, this is where we have a gap in knowledge, right. And so, this will be addressed by the studies that are being done at the moment with those nine key candidates that are in the final phase. But of course, it doesn't prevent any of the other 171, if my math is correct, vaccines from also progressing to a positive outlook.

Steven Bruce
Just out of curiosity, how big is your team working on this vaccine?

Nic Locker
So, at Surrey what we have is, it's basically a team of a team. So, it's more, you know, different individuals that are grouped together, we probably have six to seven people working on that. I have to say the efforts that we are making at Surrey at the moment are also focused a lot on the testing and how to improve the testing.

Steven Bruce
Oh, good. I'm pleased about that, because I want to ask about that later. But what I was going to say, though, is that given that you mentioned that there are nine prominent vaccines out there that must affect the sort of, I don't know, the drive, the mood of your own team, if they feel that they're not out there with the prominent players.

Nic Locker
Yes. And all those nine vaccines have the backing of either government, like the efforts that are done in Russia or in China with their three vaccine candidates, or the backing of a major pharma. So, all the key players like AstraZeneca, GSK, Pfizer, they are all backing one particular vaccine development. But it's not because you have a smaller team and perhaps different idea that it's not going to contribute overall to the potential development of something successful. I think, at this stage, the more ideas we can push and the more ideas we can develop, the better it is, because one thing to remember is that although I sound positive, there are a lot of very serious viruses for which we don't have a vaccine. Right. And I always use HIV as an example. We've been dealing with HIV for over 40 years with a lot of very clever people working on an HIV vaccine. And we still don't have one, simply because of how our immune system fights HIV. So, the more approaches and the more groups and teams of collaborators worldwide are engaged in that vaccine quest, the better it is.

Steven Bruce
That leads on to a couple of questions which have been asked by our viewers actually, Nic. One of them, whose name I don't know, has said what makes the Coronavirus different to other viruses? And Daniel has said, has there ever been a successful vaccine for a Coronavirus in the past? And I'm not sure, is influenza a Coronavirus?

Nic Locker
No, it's not. It's not a Coronavirus. It's a respiratory virus like The Bug, as we call it for the show, but it's not a Coronavirus. So, the second question is very easy to answer: has there have been a vaccine for a coronavirus? No. Ironically perhaps, if we follow up with a simple question, which is, why? It's because nobody cared. People who have been working on Coronavirus for the past 20 years really, until recently, had a lot of trouble securing research funding to develop a vaccine against SARS or MERS, which are the other two big Coronaviruses. And because Coronaviruses are normally only causing mild respiratory infection, there was no real investment on vaccine development. And now, of course, I've forgotten the first question, which was...?

Steven Bruce
Which was, how does it differ from other viruses?

Nic Locker
Okay, how does it differ from other viruses? It doesn't really, it's just new. So, in terms of the way it replicates, I can't really, I don't want to go into the details of molecular biology. But that virus doesn't really have any peculiar property that makes it stand out. I think the reason why it's spreading so fast in the population worldwide is because our immune system and our behavior is totally naive to that virus. Okay, but the virus itself, it works like any other virus or most. And this is why we've been able to implement those testing strategies and those vaccination designs so quickly. It's because we can use our knowledge of other viruses to study, understand and fight SARS-Coronavirus 2, which is the trigger for COVID-19.

Steven Bruce
We've been asked about specifics on drugs that might help protect us from the virus, ACE inhibitors and angiotensin receptor blockers. Martin has asked whether those actually provide any protection?

Nic Locker
So in terms of clinical trials, the data is very, very limited. What we do know from test experiments in cell culture, so rather than looking at the response of an individual, you take an isolated cell away from the body and you use those inhibitors and yes, they do prevent the entry of the virus into the cells and they completely block viral replication. Now the data we are missing are basically data on patient safety and efficacy in patients. And this is where perhaps, if we think about the allocation of resources, we need to think I mean, "we", governments need to think about allocation of resources and perhaps, what is going towards vaccines and what is going towards a drug that can be used, maybe in the shorter term, in order to manage patients in the clinic. And it's true that at the moment, the resources are really much more focused towards vaccine development. And I think a lot of people would like to see maybe a little bit more balance in where the efforts are going.

Steven Bruce
What about the mutations of the virus? Because obviously, the vaccine presumably will work on whichever version of it you're testing at the moment. Is it easy to adapt a vaccine to a mutation? Or is it less likely that mutations will be dangerous? I think that's what happened with Ebola, isn't it, it mutated and became less dangerous, is that right?

Nic Locker
Not necessarily with Ebola but for instance, if we think about the influenza vaccine that we get, or we should get, every year, it tends to mutate and we tend to have different strains. So different flavours or versions of the virus circulating every winter. And the way we work with influenza is that based on what we know, or what we see, with viral mutation, we try and predict which way the virus is going to mutate and we tailor the vaccine for the following season on those predictions. So, these can be adapted, you know, from

one year to the next and this is why we have a new vaccine every year. And it's true that viruses are mutating a lot, SARS doesn't really mutate more than any other virus of its kind, of its family. At the moment, we do not have evidence that there has been mutations that, for instance, have increased the pathogenicity of the virus. So, the fact that some isolates could make you more sick or less sick. There does seem to be evidence that the virus has been mutating a little bit to transmit better or faster. But this is what we would expect of any vaccine as well, that basically adapts to the host and it evolves in order to transmit more efficiently from one host to the next. So, you know, we have seen headlines on a particular SARS strain that evolved in the past with a single mutation that had evolved in the past couple of months. Yes, it is true, but it is not unexpected and there's nothing really, I would say, scary around that.

Steven Bruce
You make it sound as though it's easy to predict the mutation and therefore adapt your vaccine in advance. Is that being a bit simplistic?

Nic Locker
Yeah, perhaps I'm a bit too trivial with that. What is easy to predict are the proteins that are the protein constituent of the virus that are going to mutate because we know which ones are under selective pressure from the host responses. For example, all the proteins that are exposed to the surface of the virus and that are interacting with these receptors are the ones that are going to have a tendency to mutate more, as are the proteins that are involved for replicating the viral genetic material. So, all the viral machinery, replication machinery, is also usually under pressure to evolve, selective pressure to evolve. So, let's say that it is easy to know where to look at. So, we know which parts of the genome are more likely to mutate, then what we have to do is to make predictions of what mutation would be valuable for the virus, would they confer an advantage? And this is how the predictions are based.

Steven Bruce
Right? Okay, so you make it sound easy, but I suspect it isn't. John's just asked how much this new Coronavirus has mutated since we last spoke? And I think you said a moment ago that it hasn't changed, is that right?

Nic Locker
Yeah. So, what has been characterised, but it's really a long-term mutation, it's something that happened, I think, during the past six months is one particular mutation that has been reported in the news that seemed to be helping the transmission of the virus in the human population. And this is all that has been characterised. But you know, what needs to be clear for people listening is that there is a lot of variation in the viruses circulating. And this is why there are a lot of genomic studies to sequence, do full genome sequencing of viruses. Because for instance, if you and I were sick at the moment, it's very likely that the genome sequences of the viruses infecting both of us would be really markedly different. And it's simply because every time the virus replicates, it makes error. And this is just a natural process of viral replication. These viruses are actually not very good at copying exactly their own genome, they always make errors. And this is why as the virus replicates in my body, and as the virus replicates in your body, if we have been

infected by let's say the same isolate on day one, it is very likely that by the end of the week, when potentially we contaminate somebody else, we are contaminating these other people with different viral sequences, if you will. So viral mutation is really a natural process.

Steven Bruce
But that would not be in a way which would necessarily prevent the vaccine when we get it from being effective?

Nic Locker
No. And likewise, if I may add, in terms of testing, all the testing methods are basically targeting those regions of the genome that we know are not mutating, and that are very conserved.

Steven Bruce
Well, I'm afraid this is probably a politically charged question, Nic, but I guess it's kind of predictable. How good are the tests? They're PCR tests, aren't they?

Nic Locker
I think the tests are as good as what you make of them. So, I think that's also a politically charged answer? I've been pretty open on my Twitter feeds on what I think about the current management of the situation. We've had a lot of time to, to prepare for what's coming or for the situation we are in right now. And clearly, there have been a lot of shortcomings in that preparation. The tests are accurate, the tests are relatively fast. I think the issue is how you coordinate testing on the national level and how much you empower local authorities, right, to deliver those tests.

Steven Bruce
Well, Nic, you said the test is accurate. We've got an audience who understands sensitivity and specificity, what's the best we can expect at the moment from the best test we've got?

Nic Locker
So, if we speak about nucleic acid detection, so the PCR test. I mean, efficacy is higher than 99%. So, we are very close to hundred percent, you know.

Steven Bruce
Is that sensitivity or specificity?

Nic Locker
So that would be for sensitivity and for specificity. So, the amount of false negatives and the amount of false positives that we get with those tests are really-

Steven Bruce

That's very remarkable, isn't it? Because normally one will be better than the other for obvious reasons. And I realised the sensitivity was always quite high, but I thought specificity was down at about 95.6%.

Nic Locker
The specificity is very high, especially with the latest tests that have been approved by PHE. I think we started, so there has been some change in the exact primer sequences that are used for detection and that has been approved for PHA, for use by PHA in this country. And with the latest changes, we are really satisfied with what we can do with the testing.

Steven Bruce
Right. The reason I asked the question is because there was a time when the margin of error in the specificity of the test exceeded the numbers who were being found positive, which of course means that the results can't be really relied on. That's been overcome now, I take it?

Nic Locker
That's been overcome now.

Steven Bruce
What do we make of the latest figures then? What do you make of them? How should we be interpreting them? Let's ignore today's figures, because I think they've suddenly been bumped up by 15,000 extra cases, haven't they?

Nic Locker
Yes. Similar to what happened yesterday. I think, so the first thing is there was always that question around how big the second wave would be. Right? And if we just look at the curves, that we can see on websites or during the news, of course, what we see now is that the second wave looks like it's going to be, or looks like it is, bigger than the first wave. Now, what we need to remember is that we are now testing much more people then were tested in March, April, and even towards the start of May. So, I think, the curves and the graphics are really under estimating the size of that original peak that we had in the UK in the spring, especially around Easter. So, the visuals are scary, because we see the amount of people that are being infected now. I think the figures that we actually need to look at are hospitalisation and death. And, of course, there's always a delay between that rise in infection and the rise in hospitalisation and death. But I would say that within the next 10 days, probably is when we are going to get a bit more of a clear understanding of the impact of that second wave, because we will be able to compare what I would call like to like figures.

Steven Bruce
It surprises me that we need more time, because the spike started a good two weeks ago, didn't it, or longer than that. And sure, there's been a slight increase in hospitalisations and deaths, but nothing like the numbers that we saw right at the beginning of the virus.

Nic Locker
No, but this is also because a lot of people that are being, I think, again, a lot of those people that are being tested now, are perhaps people that are asymptomatic, but part of the family, or people that are younger and that would not necessarily have been tested back in the spring. And people that are so and that younger part of the population being a little bit more solid, I would say in the face of the infection.

Steven Bruce
A chap called Daniel has sent in a couple of questions, and I think I can see where Daniel stands on this particular argument from his questions. But he says that there's been 650,000 flu deaths per year for however long it is, and I'm going to rely on his figure because I can't remember what it should be, that's despite the vaccine for influenza. Do we really expect this to be more effective than that vaccine? Should we be especially concerned about this particular virus?

Nic Locker
Well, I think yeah, perhaps it's unfair to relate the amount of death for influenza to the existence of a vaccine because it's not like the MMR vaccine that every single child has. The flu vaccine is something that you can choose to get or not. So, you can't really, I wouldn't personally make a direct comparison and I don't think one could. I think we could be able to compare if we knew that more than 80% of the population was vaccinated against influenza and still, we had that big disparity in the figures. So, would the vaccine make a difference for SARS-Cov-2 and for COVID-19? Personally, I'm convinced that it will make a huge difference, especially in preventing the spread of the virus even further in the population.

Steven Bruce
Are the two, this is a very naive question perhaps, are the two at all comparable? The two viruses? Is SARS-Cov-2, COVID-19, is that more lethal now, now that we've got to this stage of our knowledge of the virus, than influenza?

Nic Locker
I think it's difficult to compare the lethality, although you can compare the fatality rates. I think the thing that we really want to be able to understand in the next few months is as we reach the influenza season here in the UK, how the two infections are going to coexist together. So, for example, for people that have influenza and that already have some sort of inflammation of their upper respiratory tract, how is that going to impact on potential infection by SARS-Cov-2? Is that going to increase the fraction of people that are symptomatic as opposed to asymptomatic? This is what we have absolutely no knowledge of and no feedback yet. So, we, unfortunately, we are going to live and learn for that interaction.

Steven Bruce
There are lots of members watching through Vimeo at the moment, I'm told, who are intrigued to know whether we should really be treating the latest bug, SARS-Cov-2, more seriously than influenza. And I suppose, again, this reflects what you've just said, I suppose, we don't test for influenza and yet, if you test for coronavirus, the new one, presumably you're picking up any number of coronavirus virons in people's

respiratory tract, which are not causing problems. If you did the same for flu, you'd probably get the same high number of positive results, wouldn't you?

Nic Locker
Possibly yes, absolutely. The amount of people that are getting what they call the flu during the winter, but that don't report it to their GP or that don't visit A&E, fortunately, is incredibly high. So, it's really difficult to put a number on this figure. Now, what's important is that the test that we use for COVID-19 really discriminates between SARS-Cov-2 and the other coronaviruses that are routinely circulating in the population and only making us mildly sick. So, we are able to keep, and for this winter season, we are really going to be able to keep track of what are the respiratory infections that are due to this coronavirus as opposed to all the other coronaviruses that normally circulate in the UK at this time of year.

Steven Bruce
Yeah. Finola's asked a question about what happens if we don't ever get a vaccine for this?

Nic Locker
Yeah, so that's a very good question. That's a question I get asked a lot. So, I think this is where we actually need to be able to test and contact trace very efficiently people. So, if we don't have a vaccine, what we need to be able is to eliminate the routes of transmission for the virus. And we come from a culture where, perhaps a year ago, if we had seen somebody wearing a mask in the street in the winter, we would have looked at that person like somebody completely weird and not really understood. Whereas I think for us in in the future these are practices that are known to reduce the spread of respiratory viruses that our neighbours in Asia have been using and applying for decades and in those parts of the world you don't think about it, it's a routine. It's the winter, you have a fever, you have a bit of a cough, you wear your mask, it's as simple as that. You don't need to educate children on it, it's part of the culture. And I think for us in the UK or even further in Europe, it's something that we are learning that is new, but that is going to be part of our routine and I'm assuming that our children that are growing up, will grow up in a country where, when you're sick and not feeling well, you wear a mask, you disinfect your hands all the time and actually, what we may end up doing is not only reduce the spread of COVID-19, but also reduce the spread of other infections. Not just respiratory infections, but better hygiene practices we also reduce the spread of norovirus, for instance. So, what can we do, in short, what can we do without a vaccine? We can try and block transmission.

Steven Bruce
Yeah. Jackie's asked whether it's true that some reports last week said that there's a very minimal risk of contracting Coronavirus from hard surfaces?

Nic Locker
Yes, I've seen I've read some of those reports. I think what is difficult is what do we consider hard surfaces? And I think perhaps for the professionals that are working here, whether we consider plastic or metal, the data are really fluctuating at the moment. You know, I've seen reports measuring the survival of SARS on

iron for something like 72 hours, I've seen reports of nine days, I'm afraid that at this stage, we don't really have a definite answer on that. So, we also don't really have clear data on surface, not really surface disinfection with liquid, but for example, UV radiation of hard surface material is something that is currently being investigated, but there are no final words really on how efficient those disinfection processes are. Because I'm assuming that this is perhaps where the question was heading to.

Steven Bruce
Gominda has asked whether you believe that UV light is a good way to disinfect surfaces. At the moment we're fogging our clinics with whatever it is we fog them with. What does UV light have to offer?

Nic Locker
So yeah, so in terms of disinfection, that's what I was alluding to. So, UV is really good at inactivating viruses, any kind of viruses, because what it does is induce mutations and induce breaks in the genetic material of any viruses. It actually destroys our own genetic material as well. And it means that the virus cannot replicate. So, it can bind and attach to a cell, but it's not going to be able to replicate and lead to the propagation of the virus. There are a lot of different UV devices that are currently being tested for disinfection and I'm not aware at the moment of efficacy figures. And the reason why I'm saying that is because I am involved in one of these studies looking at deactivation of sSARS-Cov-2 with a particular handheld UV light and we have no point of comparison sadly. So, this is something where unfortunately, the professionals have to wait a little bit more before they get a clear answer. Now what is true is that UV light destroys the virus. Yes. Or inactivates the virus. Yes.

Steven Bruce
So, I suppose what our clinicians will be wanting to know is, are they okay if they use UV light instead of fogging their clinic? To which your answer is well, we don't have the evidence to say whether, you know, what strength of UV light or how you administer it or any of those other factors. Karen's asked whether the virus is weakening as time goes by?

Nic Locker
We have no sign of this at the moment.

Steven Bruce
Okay, and a related question, I suppose, is Mike has asked whether, if you get infected with two viruses at the same time, can they in some way help each other to evolve to mutate?

Nic Locker
So, help each other to evolve and mutate, I would say no. In terms of virus, so virus combination or you know virus A and virus B coming together to create a new virus is not really something that happens, apart from closely related viruses. For example, this is how influenza virus evolves. So, you can get infected by an avian influenza virus or swine influenza virus and they can both recognise receptors on human cells and they can create chimeric influenza viruses. For SARS, we have no evidence that this virus uses such a

process just because of what we know of its replication cycle. Now, if the question is about interactions between two different viruses? The short answer is that we don't know. The slightly more elaborated answer is that I would expect that a virus that already weakens my upper respiratory tract, or in fact, lower respiratory tract, will exacerbate the symptoms of COVID-19. So I was mentioning influenza, but we could say the same of other viruses, perhaps common Coronaviruses or rhinoviruses, that are causing throat infections, perhaps having that local inflammation where your immune system is already pumped up and exhausted by fighting that one virus will not be so good at coping with a second wave that hits it with SARS-Cov-2, and this is where perhaps, we will see that the amount of people that are presenting symptoms in the winter is going to go up, as opposed to what we had in the summer, because of these interactions.

Steven Bruce
You mentioned inflammation there and also the winters related to this. We've had a couple of questions about the effectiveness of vitamin D in minimising the impact of coronavirus.

Nic Locker
So, there's no scientific evidence that vitamin D will help. Sorry, that's a bit blunt. But there was a there was a bit of controversy over the summer around vitamin D. I think from my knowledge of the current literature, the evidence as, been circumstantial at best on the positive effect of vitamin D.

Steven Bruce
Okay, but it is known that vitamin D contributes as an anti-inflammatory component, isn't it? So, I guess, since it's recommended by most sources, that one in this country particularly should supplement vitamin D anyway, particularly in the winter, it's probably a good thing to do, but it's not necessarily a cure for COVID-19.

Nic Locker
Exactly. It's not going to do any harm but it's not going to do anything specific for COVID-19.

Steven Bruce
Samantha's asked whether new information suggests that it's not a cytokine storm which causes the problem, but a Bradykinin storm now? What do you think?

Nic Locker
No, we are still, I think most of the community is still pretty fixed on the cytokine storm for COVID-19. In particular, secretion of pro-inflammatory cytokines, such as IL-6 or IL-10, that are responsible for that pathogenicity that hits the patient. So, I think we are still with that hypothesis. You know, I think one of the problems, it's not a problem, but because as scientists, we are trying to communicate very fast a lot of results, we do tend to have university marketing and media departments that will promote very quickly on social media any paper of preprint, and I'm not blaming colleagues, we're all culprits in that, that will promote preprints very quickly, when in fact, we need to wait for those studies to be published and to be

peer reviewed by colleagues and to be published in a reputable journal so that we can make a little bit more conclusions. Otherwise, it's very easy to get a head start on ideas like that.

Steven Bruce
Jackie's asked an interesting question. Do you think that this sudden, massive increase in sanitization, disinfection and all that, do you think that will affect our overall resistance to any sorts of bugs?

Nic Locker
Yeah, definitely. So, it's not good to live in a sterile environment, right. Our immune system needs bacteria there, it needs viruses. One thing to consider is that we have, I believe, over 10 trillions of viral particles in our body, just as part of the natural interaction between viruses and human cells and it's the same with bacteria. So, we are constantly interacting with bacteria and viruses. Of course, we speak a lot about the gut microbiota and how this is good for you, this is good for your health, this is good for digestion. So, a constant sanitised environment is, I don't want to say that it's going to be bad because it's going to be useful in reducing the spread of the virus. But I think we need to be aware of the fact that some viruses and some bacteria are good for you and we need these around. So, we can't live in a sterile environment, that would be bad.

Steven Bruce
I've had a question flagged up to me that a number of people have asked about the research around what COVID-19 actually is, and in particular, they've said, is there any research which says that SARS-Cov-2, the virus, causes COVID-19? And I thought that was a given.

Nic Locker
Yes, because that is a given. Yes. So, SARS-Cov-2 is the pathogen, so it's the etiological agent for COVID-19.

Steven Bruce
Yeah, I think when you were last on the show, actually, we mentioned the fact that there's quite a lot of confusion over whether we talk about Coronavirus, which of course means a whole lot of different Coronaviruses; COVID-19, which is a specific disease; or SARS-Cov-2, which is the latest version of the Coronavirus, the one we're worried about now.

Nic Locker
Exactly.

Steven Bruce
I'm glad I got that right, means I was listening last time.

Nic Locker

You got that right. Exactly. You remember correctly. COVID-19 is the symptom, the manifestation, the disease. SARS-Cov-2, is the agent responsible for that disease.

Steven Bruce
I'm gonna go back to something you said earlier on now, because Matthew has sent in a question asking if you know what Dominic Raab was talking about when he said the PCR testing has a false positive rate of 93%? Was that just a politician getting confused?

Nic Locker
I would assume so.

Steven Bruce
A false positive rate of 93% is a bit excessive, isn't?

Nic Locker
I personally have stopped taking too much interest in what the politicians are saying about the science. We should remember that we have been promised mass testing months ago, we are still waiting for that to happen.

Steven Bruce
One last question from our audience, because we're out of time almost. John says, historically there's never been a contemporaneous vaccine. Do you have confidence that individual research groups will cooperate in the event of a breakthrough?

Nic Locker
I would- so if I understand that correctly, what your listener is stating is that we haven't found a vaccine recently in a very rapid setting. I would dispute that and take the example of Ebola. Ebola outbreak 2014 in Western Africa, collaboration between academics in Europe and in Africa and the WHO, we now have a vaccine that is used in the current Democratic Republic of Congo outbreak of Ebola. So actually, what happened is that we have learned from the Ebola outbreak, how to rapidly develop in the field a vaccine.

Steven Bruce
Okay, thank you, Nic. Sorry, one more I just want to put to you, one of our viewers has said is hand washing with soap as effective a disinfectant? I thought hand washing with soap was actually more effective than disinfectant and probably better.

Nic Locker
Absolutely, absolutely. So, the soap is a detergent and it's really good at breaking the structure of the virus and it will inactivate/destroy Coronaviruses, and in fact, any other enveloped viruses. So, if you want to use soap, by all means, use soap. It's just more difficult to use on the spot when you're travelling or in the outside environment.

Steven Bruce
No doubt Donald Trump will be advising that we all either inject it or swallow soap in the near future. Nic, there were a few other questions I didn't put to you because I don't think they're your area of expertise, which is about the rules that are being imposed on us to do with Coronavirus and I think it would be unfair to put you on the spot there because your expertise in the virus itself and it's fantastic of you to come over a second time and explain what's going on and what we now know about The Bug, particularly since you're actively involved in saving the world from Coronavirus, the new Coronavirus. So, thanks again for your time.