Academy of PHYSICAL MEDICINE

Virology

with Nicolas Locker

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TRANSCRIPT

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Steven :

Good afternoon and welcome to the Academy of physical medicine once again for another great lunchtime session of CPD. We're going to get scientific with you today. I am joined by professor Nick Locker who is the reader in neurology at the university of Surrey. He set up and now runs a laboratory there whose main question is, and I've got to read this, how do cells or pathogens maintain a specific translational program in conditions where general translation is compromised or targeted for regulation? So if that is a question which has been preying on your mind recently, then now is the chance to get all the answers you could possibly wish. Of course, there's another reason why we want to have a virologist on the show today. Nick's particular interests are virus host interactions and RNA protein interactions, which is close to all our hearts right at the moment, Um and he's eminently qualified to talk to us about that. He's written papers on countless subjects including HIV, norovirus and diarrhoea in cattle, so he's very, very well educated and ideally placed to help us out in our current problem with coronavirus. Nick, welcome to the show.

Nick :

Where'd you go? Thank you.

Steven :

Tell me just you were discussing this earlier on before we went on air, but I think you're somewhat undermanned in your laboratory at the moment aren't you, what's going on there at Surrey?.

Nick :

So at the moment, most universities in the country are closed for all usual businesses. So we have most of the staff working from home and the students are already engaged in a remote learning activity. So, you know, the campus that is normally thriving with maybe 20,000 students and staff is completely empty. They are the central activities that are related to the on going, covid pandemic. So the only facilities that are open are the ones that are contributing to the research efforts, meat through diagnostic work or, trying to understand better, the mechanisms by which the virus causes, disease in humans and, vaccine work. My lab that has normally 10 to 12 people working, constantly is currently empty with just myself in the office/the lab doing experiments and participating to those efforts. So it's a very strange time, indeed.

Steven :

So are you directly involved in research into vaccines for Covid19 at the moment?

Nick :

Yeah, so we are jointly involved in two lines of work related to Covid19 to summarise it broadly. So on the one hand, we are at, and this is probably what is the closest to, something applicable for the population in the short term. We are developing, a cheap and portable test, for sars covid 2, so the virus responsible for Covid19. Cause I guess if you and the viewers follow the news, you hear constantly that we have issues with testing capacities, with the NHS, having access to, reagents and kits. So there is a huge bottleneck in our testing ability, down, which is down to the actual material that we are using for testing. So what we are trying to do at Surrey is to develop novel ways of testing that are faster and cheaper so that perhaps we can go in the community and test people and get results very, very quickly.

Nick :

So that we understand more the spread of the virus and in the more longer term we are involved in various projects that I can't reveal about for confidentiality reasons that are all aimed at adapting existing vaccine and repurposing them for, efforts in controlling, covid 19. And I would say, you know, that probably what every single academic biology lab in the UK is doing at the moment is repurposing its research activities to Covid 19, either from the testing strand or for the vaccine, strengths. Because this is where

New Speaker:

Is it centrally coordinated because obviously there's a possibility that all these labs will go off and try to do their own thing and get to the answers first and therefore duplicate effort, or is somebody making sure that it's all targeted properly?

Nick :

So you would hope that it's coordinated. It is not centrally coordinated by the government, um or any organization. What is in a way, what the outbreak has brought, out of the research community is really a fast exchange of ideas and communication of putting in our data on various channels. Publication of pre prints. So basically studies that are not peer reviewed yet but that we put out on the web on, archived website sharing of ideas via Twitter. And basically this is how the centralisation is done. So for example, we have networks of biologists, that simply message each other on Twitter and okay, have you, have you thought about this? Have you done that? And we take the interesting ideas off line, but this is pretty much, very dynamic process that we are coordinating ourselves. And you know, from the academic perspective, if there is one thing to emerge out of this outbreak is perhaps, mow to better, communicate and how to better collaborate between ourselves other than doing other than walking in silence, and in isolation.

Steven :

I imagine that that should be a little bit easier with Coronavirus because as I understand it, there are no financial incentives to people keeping secrets because everyone I've read about has promised that their research will be freely available as soon as they get it. Is that the case with you?

Nick :

Yeah, absolutely. All the data generated is available on request. The material, we will engineer in our labs is available on demand. Of course this has the risk of turning some labs into factories because we are, we can be overwhelmed with requests from collaborators, which we are obliged to fulfil. So there was already a little bit of a, you know, of time management and resource management to come into the equation. But you know, definitely it's, it's bringing a whole new collaborative meaning to biology researcher from, from my point of view.

Steven :

Yeah. Just for a layman like myself what are the stumbling blocks to getting to acquiring a quick and cheap test for Coronavirus or the new strain? Because to me it seems you, you poke a swab into something like a mucus membrane and then you take it out and you see if it's got the, the virus in it.

Nick :

Exactly. So what you summarised here a is how to get the test to work and that's exactly what we are all working. And you can use different techniques, different physical ways of engineering that response. And this is what this is what people will or will try and do in different labs. Now if you want to be able to hold out these tests nationwide or worldwide, you need to be able to rely on these tests. Not 90% 95%, 97% but 100%. And so what you need to be absolutely confident, we believe is that somebody that hasn't got coronavirus will test negative. And that somebody that has the virus will test positive.

Steven :

So, specificity and sensitivity, which we would apply in our orthopaedic tests, but we would rarely get a hundred percent in any of those.

Nick :

Exactly. So this is what we would call false positive and false negative detection. And basically the, the bottleneck for all the alternative tests that are being developed that we want to make faster and cheaper than the current approved one is, is literally to make sure that we detect you know, that our tests are accurate and sensitive because if you go and tell someone that he hasn't got the virus, you want to be sure of that code. Okay. You, you know, you can't take seven guesses and this is where the, this is where the bottleneck is currently aligned,

Steven :

Are you also looking at testing for markers that people have already had covid 19?

Nick :

Yes. So that's an interesting point because it's sort of part of our strategy to get people back to work. Right? and you know, if I had to think about the most single frequent email or text or WhatsApp message I have received from colleagues, members of the public or friends, it's can I be tested for coronavirus antibodies? And when can you do this? And again at the moment we have capacities, maybe I shouldn't say that, but labs have the capacities to run the test to say whether we have antibodies, so whether we have been infected for coronavirus. Anybody can do that in a lab with basic science equipment. The one thing that nobody knows, okay, and I insist on that, nobody knows, is whether having had the virus in the past is a predictor for not having it in the future.

Steven :

Yeah.

Nick :

There are many coronaviruses that were circulating in the population before this current outbreak started. So plenty of coronaviruses are causing the common cold. They are making us sick for a few days but you know, it doesn't kill us. Whether the, the tests that we, that are being developed for testing whether we have covid 19 or Sars covid 2, so the virus responsible for 19 in the past, whether those tests cross-react with the coronaviruses that are already circulating in the population is the problem for interpretation. And whether having a positive test basically gives you, you know, a safe card for going out and going out to back to work is another question. And we simply don't

know this because you have to remember one thing. In December or in November that virus didn't exist on the radar of scientists. So what we are doing now is basically trying to scale up research that normally occurs on several, the timeline of several years to decades to understand immunity, to understand how our immune responses are building against a particular virus, how we can be protected. So we are trying to do everything at the same time. And why, you know, you see all those reports popping up in the news about tests being created somewhere in the world and then a few weeks after the tests are deemed not sensitive enough somewhere else, it's because we are lacking the background to understand and test really accurately, immune response of our body against Sars covid 2.

Steven :

Somebody has sent in a question, Nick, asking exactly how reliable the tests have to be before they can be considered useful. You said a hundred percent earlier on, but I would imagine that our government would quite happily accept something a little bit less than that if it helped with managing the, the outbreak.

Nick :

So I've said so it's usually a reasonable compromise. Okay. So at the moment, because we have tests that are extremely accurate, so between 99.5% to 100%, this is the accuracy Public Health England is working towards. So this is the benchmark we have to achieve if we have to develop novel tests.

Steven :

Okay. maybe if I can, can I ask you to take us back to basics? I nicked this diagram this morning from the New York Times, so I hope people don't ask me to share it online because that would be a bit naughty of me without New York Times permission. But essentially it's a, it's a simple diagram of how the virus gets into the body and what it does. Could you run us through that process, please?

Nick :

Yeah, so I mean, basically what, what you're saying here is viral particles, so an envelope virus, with some spike proteins. So the red triangles are the viral proteins that are exposed on the surface of the virus and these proteins have affinity for the particular receptor that some of our cells, but not all of our cells are expressing. So in the case of Sars covid 2, the spike protein of Sars covid 2 is able to recognize a receptor called ACE 2 that is expressed on a number of cells from different organs, but it is particularly present in the respiratory tract and in the epithelial cells that are lining the respiratory tract. And so it's that, initial step of the virus binding to those receptors that's going to cause the cell to be infected. What's actually very interesting is that, this is the same receptor that your regional Sars virus that emerged in 2003 was able to use.

Steven :

Is that the same for the MERS virus as well?

Nick :

It's a different receptor for Mers but Sars and Sars Covid 2 use the exact same receptor. What's interesting is that the strength of the interaction, so how tightly the spike protein and the receptor bind together are different. So the, the Sars Covid 2 ACE 2 receptor interaction is much stronger than the reception, then the interaction that was observed for Sars. And this is why the virus has

been able to spread much more efficiently in the population and much more widely as compared to Sars. And we think that it's, there are other reasons, but we think that it's because the entry portal is actually more efficient for Sars Covid 2.

Steven :

Sorry, go on please.

Nick :

No, no, ask your question.

Steven :

Is there any truth in the report, which when, which started early this month, but actually nicotine smoking, um, to some extent blocks the access of Sars Covid 2 into the cells.

Nick :

So

Steven :

The focus seemed to have been less affected by covid 19 than

Nick :

Yeah, so, so it first comes from epidemiological evidence that among the people that were hospitalised the prevalence of smokers was less present. And it's also down to the fact that these receptors are part of the family of proteins that are involved in binding and recognizing nicotine.. So the idea being that if nicotine already binds to the receptor, it kind of prevents the virus from binding, right, to make a really lay summary. At the moment, there are no direct evidence to show that you can block entry and inhibit viral replication by adding nicotine to cells that would display the receptor. But what people are doing are, really collecting meta analyses, so trying to increase our knowledge of how much smokers are affected by Sars Covid 2. What is the proportion within the Covid 19 displaying a cough really on a more global scale, how many are heavy smokers, medium smokers or non-smokers so that we gather more evidence?

Steven :

I think that original report was based on evidence from France, in fact, wasn't it, where a high proportion of them are smokers? Yeah. Does it annoy you that people like me use terms like novel coronavirus cells, Covid 2, coronavirus and Covid 19 interchangeably?

Nick :

Not at all. Because to be fair, it's, I would, I would find it confusing as well. The best analogy I can make, and it's usually one that people can relate to easily is that Covid 19 is the disease, Sars Covid 2 Is the virus.

Steven:

Okay.

Nick :

HIV is the virus. AIDS is the disease. It's, yeah, you see, it's that, that sort of, you could tell me, but you know, you can, you can use one or the other without any issues. When we speak about detection tests we tend to speak about Sars Covid 2, . And for patients, we tend to speak about Covid 19 because it makes a little bit more sense for biologist. But this is just being pedantic.

Steven :

I suspect that one of the areas, the other areas of confusion for people like myself is where the expertise of a virologist such as yourself ends and the immunologist and epidemiologist starts, and I know that nobody's going to be able to read this slide, but it's available freely in lots of press at the moment. It's just indicating the number of deaths from Covid 19 in the various countries. And do you have any thoughts on why, for example, Britain's deaths exceed those of Italy and are less than certainly less than you'd expect given the population of the America or of America by comparison?

Nick :

So what, so if I can go back to your first to your first point, I think one of the things you've highlighted very well is that because a lot of data are available freely and everywhere and you know, a lot of my colleagues are putting their own data out on Twitter, it's really easy to try and patch out some piece of scientific information and then try and try and make any sort of scientific comment based on our limited understanding. And so for example, I'm a biologist but I'm not an immunologist. What's important is that there are there are terms that we need to be careful with. For example, with the graph that you are showing. Okay. The first thing that somebody would try and do is to extract the mortality rate. Just thinking, okay, if I know how many people are infected and how many people have died, I would have a mortality rate. And I would say you can get the ballpark figure, but to really understand the mortality of a virus, you need to be able to take into account the fact that the death that you observe at one point in time are actually only reflective of the infections that have occurred to date. Okay. But for example, infections that would have been reported on the same day will only impact on death that will perhaps occur in month. Okay. So it's really difficult to get a very accurate mortality figure. Now what we can really, easily look at are the raw numbers and what I would say is that the differences that you see between countries are the direct, impact of governmental policies.

Nick :

Okay. So one of the things I've said numerous time on various TV channels throughout the course of this outbreak is that, you know, typically the US and the UK had a head start on the virus. Okay. So our country and the U S were in the fortunate position that we could see what was going on elsewhere. We had minimum number of cases when Italy China and Spain were already in the midst of the outbreak and we could have used those data to already design control measure, Now for what I believe are political reasons this was not done. And this is why we end up now with the figures that we have if you look at for example the implementation of physical distancing and lock down measures that occurred in parts of the U S or in the UK, you know, these measures were recommended by the WHO and by biology experts weeks before they got implemented. And the numbers that we see on that graph actually reflect that late implementation.

Steven :

I also, I did, I tried to do a little bit of my own amateurish research on this and, and you know, on here it says Italy 32,000 deaths Britain, 35,000 more or less. But actually the populations of the two countries are not too dissimilar, but the population density is much greater in Britain. So it's occurred to me that population density surely must have an effect in spreading a virus.

Nick :

Absolutely. And you know, if you take a very particular example that's very close to my heart, which is the number of cases in Surrey we can see that in the, in the cases reported, we have a very high proportion and we've had since the onset, a very high proportion of cases in the Southeast. And if you think about it, okay, London and the Southeast you have three airports, you have wealthy, or wealthier people that all went on holidays for half term, that very likely went to France and Italy for skiing, where basically the outbreak was brewing, and where the number of cases were very high. And all those cases flew back to the UK in highly populated and densely populated area and transmitted the virus. Right?

Steven :

Nick, we're in danger here of saying that skiing is bad for you and smoking is good for you at this rate!

Nick :

Absolutely.

Steven :

Let's hope it doesn't come to that. Caroline's asked a question. She says, is there any research going on in the UK as to the individual's immune response to Covid? It appears that some individuals overreact and she's asked your opinion on neutrophil extracellular traps.

Nick :

So when we are saying that individuals overreact so basically one of the, one of the clearest things that we know related to a mortality and the reason why people are actually dying of Covid 19 is because the immune system goes in overdrive. Okay. So we have our immune system has really neat and efficient ways of clearing out, out pathogens. Okay. So at the site of infection we will infiltrate neutrophils and macrophages that we select that we secrete cytokines that will then mediate an immune response, attract B cells and T cells that are there to on the one hand mop up the infected cell and on the other hand ensure that antibodies are selected, and synthesized against the virus. Now, what can happen in some individuals is that that secretion of cytokines and inflammatory cytokines, so production of localized information goes uncontrolled in overdrive.

Nick :

And what it means is that for some patients, their lungs become sites of hyper inflammation. There's a damage of the lung tissues, there's a damage of the vascular tissues on the lung, and this is what, then subsequentially leads to respiratory failure and the multi organ failure that is associated with mortality and morbidity. And so definitely there are. And so this is what we want to understand. What makes people actually go into overdrive? What are the genetic variants? So are some people

more prone to these inflammation? Is it because of differences, subtle differences in, the viral sequence between these individuals? So we have in the UK research in trying to analyse that. And we have research in trying to map the quantity and types of cytokines because these cytokine storm as we call it really represents a cloud of dozens of cytokines sensitized. So what we are trying to do at the moment is to map for each individual cytokines how much of it and which exact one is made for different clusters of severity of disease. And we hope that this will help to understand the immune response to the virus.

Steven :

I remember reading somewhere that coronavirus or the novel coronavirus actually destroys T lymphocytes. Is that the case? Is it an immune response?

Nick :

So the virus is a cytotoxic. So what it means is that as long as the virus can enter a cell, it will damage and lyse the cell and be cytotoxic for the cell. So for example, it can enter, replicate and kill monocytes, microphages, some but not all the T cells. The receptor is also expressed and this is something that hasn't really been studied a lot, but the receptor is also expressed in subpopulations of cells that we have in the brain. So scientists are interested in understanding potential long term neurological damage for survivors of Covid 19, as this is something that we will have to live with. In the short term we want to be able to test and trace people that are infected. We want to be able to protect people that are in NHS, facilities or that are in ICU. We want to get these people out from ICUs, but we also need to understand the long term damage beyond damages to the respiratory tract.. What are the damages to cognitive function? We know that liver cells also express the ACE2 receptor. What are going to be the damage for our liver function in the long term? And these are all questions that unfortunately that we are not going to be able to answer now, but these will be questions that we learn in full in the years to come when, survivors of Covid 19 are treated, develop other types of syndromes, other type of pathologies. And this is where it will actually start to be really difficult to understand the direct contribution of Sars Covid 2, of the virus to these associated pathologies. Whether they would have emerged in those patients anyway,

Steven :

Angelica has asked a couple of specific questions. And certainly one of them is very close to my heart. The first is what's the point of wearing gloves? Because actually if soap destroys the virus as I believe it does, then you might just as well wash your hands before we treat our patients and then wash them again afterwards. And there's no point in sticking a pair of nitrite or latex gloves in between those two processes.

Nick :

So it's true that soap is, soap or any alcohol-based solution is efficient at killing the virus. And this is simply because on that diagram that you showed that the virus is coated by a layer of lipid. Okay, So just below those red spikes you have, a layer of membrane and the soap will act as a detergent and disassemble the viral particles. So, yes. Soap is efficient. I guess why do you need the gloves? It's basically to provide layers of protection. So you can wash your hands. Yes. But if you have to do it, not one time, you know, as opposed to a member of the public. It's not as if you had to do it once, getting out from the house, and maybe once coming into the house. You're going to have to wash your hands 20, 30, 40, 50 times during the day. How good are you going to be with that process?

Are you going to really go for your 30 seconds in the nooks and crannies between your fingers? It's difficult to guarantee that you would do that very efficiently. So wearing gloves is actually a way of protecting yourself and protecting your clients and making sure that um the unfortunate event where you wouldn't have cleaned your hands as properly as you should have, you're not going to put yourself or clients at risk.

Steven :

And there's a well known test that the NHS uses and others I'm sure, where you coat your hands in some substance and then try and wash it off, and then you stick them under a UV light and it shows all the bits that you've missed. And I guess the same applies to a virus, doesn't it? You could easily miss some, so the gloves add an extra layer of protection as you said. What about clothing? She's asked about people obviously have a dislike of using plastic and we're being advised to use plastic aprons just as the NHS are. And she's saying, well, why not just change our tunics, our clinic coats in between patients, and shove them in the wash.

Nick :

I think you have to think about what is practical in the long run, right? Because you are probably going to have to live with those practices for, I would say most likely the next 18 months, if we are conservative. So what you want, what we would like people to adopt is a standardized approach. But of course there needs to be some flexibility for individual practices. I think trying to stick to what the NHS is advocating is probably the best practice that we can recommend, at the moment. So disposable, aprons. UI think the other questions, masks and goggles..

Steven :

Right. Angelica makes a very good point. She's not asking you to change the guidelines, she just wanted to understand them better, so we understand the mechanisms better to make our own risk assessment and decision making process. We have been asked about the virus survival outside the host in other environments. And it occurs to me that last week we were talking about whether carpets on the floors of our treatment rooms pose a potential hazard. How, what is the risk? Do we kick up the Corona virus every time we walk across the carpet, or does it just sit there and do no harm?

Nick :

So as you know, a virus is a inert, right? So it's not going, it's not like a bacteria that will grow on the carpet or a fungi that could grow on the carpet. If there was one viral particle on the carpet, it will not disappear, but it will not be infectious, unless it reaches a living cell that is expressing that oronavirus, that, uh, ACE2 receptor. Now in terms of how long coronavirus can survive on different surfaces, there are limited studies that have been doing with the actual virus we work on. There are a lot of studies that have been done inferring from what we know of other coronaviruses or other respiratory viruses. And depending on the type of surface we are speaking about, so plastics, wood, hard metal, carpets, it can go from anything between 12 hours to, I believe the most conservative figures I've seen is eight days.

Steven :

Eight days. Gosh.

Nick :

Eight days.

Steven :

Yeah.

Nick :

Right. Eight days on a, I think it was on a hard metal surface. Okay. So I think the, the one of the issues with, you know, carpets or floor surfaces is that you can't disinfect between every patient. Okay. You can't clean, you can't deep clean fully at the end of every single day. But what you can do is to think about how the virus is transmitted. So it's respiratory droplets and aerosol that are produced when people are sneezing, coughing. Um, the, the most likely best practice to make your workspace safe is to basically have both yourself and your patients wearing masks and potentially, u, for yourself, definitely gloves, and for the patient, disinfecting their hands as they enter the practice.

Steven :

Yeah.

Nick :

So, you know, for example, when I have people coming to the Hope Centre in our facilities as they enter, they have to wash their hands, put a mask on. Okay. And that's, I would say the minimum level of protection I would be comfortable with.

Steven :

Yeah. Somebody's asked about the advice that we have that we should air our rooms, get a through flow of air in between patients. Any idea? I mean, I suppose it depends on how much air is flowing. So it's one window or two windows, whether it's windy outside or not, but how long do we need between patients to minimize the risks satisfactorily?

Nick :

So there is so there is no data on that. There is no modelling on how fast the virus can be can move within a room and actually what's with airborne transmission it's quite interesting, there are a lot of conflicting data. What we've, what we know for a fact is that from people that have been so from infected people that have travelled in an enclosed environment, so people that were on a plane with a patient that was sick, we have very limited evidence of individuals that were not in direct contact. So there's then one meter infected. So there is potentially airborne transmission, but it's not very efficient. So I think the air flow and you know, trying to get your room with a lot of air current is probably a good practice. Whether it's going to make an actual difference and impact on the risk for you, I think at the moment it's hard to speculate on that. It's, it will probably do more good than harm, let's put it that way.

Steven :

You won't be surprised, I imagine that an awful lot of our viewers are very keen to find remedies, preventative treatments, which don't rely on artificial drugs. And so I've got a couple of questions which perhaps we can finish up with, cause I realize we're getting close to time. One is from John

who says what do you know about the research being done in China into treatment with high levels of vitamin C, vitamin D, zinc and magnesium. New Zealand, apparently, has similar research and something I sent you earlier on, one of our members sent in an article about metadichol, which she claims to be very effective in overcoming the virus. Do you have any thoughts on those investigations?

Nick :

So I think at the moment there are a lot of reports of different, um natural products or food supplements that are effective against Covid 19. And being in the biology and antiviral field for a long time, we constantly have reports of natural products or food supplements that can help treat or cure or prevent symptoms for a lot of viruses, and most viruses in fact. What we really lack at the moment, is evidence to show that giving those substances in comparison to placebo treatment is efficient. So if you look at vitamin C, well it's not going to do any harm. Right? But is it going to specifically block viral replication? We don't know that and there is no data, and it's the same with vitamins, it's the same with sorry, the compound you just

Steven :

Metadichol was the name that was in the news release. The press release I saw.

Nick :

Yeah. so we, we don't have controlled evidence on patients to show that taking these drugs or compound is actually going to reduce the ability of the virus to replicate. I think this is where the difficulties, you know, reading reports that they may help because okay, 10 people have taken that drug and they haven't developed symptoms. Okay, fine. But would, would have they developed symptoms anyway?

Steven :

One of my favourite doctors, Malcolm Kendrick, who I mentioned to you earlier on, he does talk a lot about vitamin C and vitamin D supplementation. And he does point out that I don't think anyone's ever died from taking vitamins. And the worst that can happen is that you end up with very expensive urine.

Nick :

Exactly.

Steven :

So it's worth, it's worth. It's worth taking them, even if there is no evidence. But be cautious about products which are based on basing the evidence on a press release.

Nick :

And I would say in particular members of the public should be cautious of where they source the product. So, you know, of the other Quinta compound can be can be formulated in all kinds of ways that are not necessarily good and can be toxic for the human body. So this is where we have to be cautious. You know, obviously, you were mentioning those compounds. We can also without making a bad analogy, you know, we can also think about the comment that President Trump did

about bleach. Yeah. Bleach. Bleach is killing any type of viruses including coronavirus. The WHO has had to issue a statement that bleach was actually toxic and people should not ingest bleach in order to protect themselves from coronavirus. And this is where I think that those press releases or those, um tements from people that perhaps have a lot of media exposure can be a little bit dangerous.

Steven :

So there was always the great spike in morale when people thought that president Trump was drinking bleach.

Nick :

For other reasons, yes.

Steven :

We shouldn't get political. One very final question, and it's probably a yes or no question. It's coming from somebody who remains anonymous. Does ultraviolet light kill the virus?

Nick :

Yes,

Steven :

It does! So actually that's a useful thing to do in our clinic rooms to sanitize them?

Nick :

So one of the, one of the way we inactivate viruses is by UV irradiation. There are very specific length and strength of treatment that are published that people can access there are guidelines for plenty of viruses. And this is simply because what the UV are going to do is to break down the genetic material of the virus. So it's going to affect the RNA molecule that are making up the genetic material of the coronavirus, and therefore it creates an inactivated virus so it can bind the receptor. It can be internalized so the virus can still get in, but it won't be able to replicate because the genetic material of the virus is damaged. Now this only works if you apply the right irradiation protocol.

Steven :

We've taken you away from your laboratory where you're doing literally lifesaving work and I'm really grateful for your time, and I'm very conscious that I don't want to take any more of it than, than you agreed to. That's really, really kind of you to spend 45 minutes with us talking about Corona virus and Covid 19 today. We had, it was described to me on my list here, a squillions of questions came in and I'm sorry we weren't able to cover them all, but I'm grateful that you've covered what you did and I wish you the best of luck with the work you're doing at the moment. And I'd be really grateful if we could perhaps talk to you in a few weeks time about what progress you've made and what developments there have been.

Nick :

Absolutely. That would be, that was my pleasure today, and that would be my pleasure to speak again.

Steven :

Thank you very much for your time.