# Transcript

**Ask the experts Coronavirus fake news & medical terminology**

**RACHEL MCMULLAN:** Good afternoon. Thanks for joining this live Open University Ask The Experts Session on COVID-19. My name's Rachel McMullan, and I'm a lecturer in health sciences at the Open University. I'm going to be putting your questions to our panel of four Open University experts today.

In a moment, I'll hand over to our experts to introduce themselves, and then each of them will give you a brief introduction before I open up the floor for your questions. If you'd like to submit a question for our panel, please email it to the address at the bottom of the screen. That's STEM-News@open.ac.uk. So to get us started, I'll hand over to the panel to introduce themselves.

**HARITH ALANI:** Hi. I'm Harith Alani. I'm a professor of web science at the Knowledge Media Institute. My work is primarily focused on analysing social media to better understand various social phenomena online.

**JON GOLDING:** Hi. My name is Jon Golding. I'm a senior lecturer at the OU. I'm a cell biologist, and I run the Open University module on infectious disease and public health.

**HELEN WIMALARATHNA:** Hi. I'm Helen Wimalarathna. I'm a lecturer in health sciences at the Open University. I'm an infectious disease epidemiologist with a background in evidence-based medicine.

**DAVID MALE:** And I'm David Male, professor of biology at the Open University. I've got a particular interest in immunology, immunology of infection. And I can also talk a little bit about testing and the tests that are being used for the virus as well.

**RACHEL MCMULLAN:** Great. Thanks, everybody. So we're going to start by letting our panellists talk very briefly about their particular areas of expertise around COVID-19. And we'll start with Harry.

**HARITH ALANI:** So back in January, the European Commission started a rapid response for the outbreak, which was probably one of the first big agencies of research to tackle the problem. So they raised about 50 million euros to fund around 17 projects to look into the pandemic from many different angles. So for example, some of them are looking at vaccine development. Some of them looking at treatment, diagnosis.

But our research, our project, which is called HERoS, which stands for Health Emergency Response in Interconnected Systems, was luckily one of them. And it's mainly to look at trying to understand the pandemic from many different angles or many different factors, adding those to the models.

So for example, although there are many models to give a representation of the spread of the virus, we don't have many that add the factors of behaviour. And that's how that influences the spread of the virus, or more importantly for us in this project, the impact of misinformation, and how does that impact the actions that people take, how they respond to the virus, how they respond to the recommendations, and how does that, in general, affect the resilience of society and of communities.

So this project is an international one. It's coordinated by the school of economics in Finland. It includes 11 partners. One of them is the Italian Red Cross. The other one is Project HOPE, which is a global health and humanitarian relief organisation. And it will involve a very kind of a big diversity of work.

So some of the partners will be looking at the problem from ethnography work. They will be doing lots of interviews, contacts with people, face-to-face meetings, if they could, in this climate, to better understand the actions and the impact of those from various organisations and policymakers.

Our work at the Open University will be mainly focused on monitoring the spread of COVID-related misinformation. And as you know, there's tonnes of it. Perhaps this is the biggest amount of information that any event has witnessed in the last decades, I would say. And partly because, obviously, it's an event that impacts the entire globe. It's something that's evolving. So not everyone understands it very well. The information that we have about it is still scarce and developing all the time. So unfortunately, there is a lot of misinformation that's also spread around it.

What we would like to know in this project is, first of all, how can we better monitor that spread? How can we understand which of these types of misinformation is spreading more widely than others? Or why a certain type might be spreading in one part of the world better than the other ones.

But also, we know that there are many organisations who are battling misinformation continuously, including the WHO, for example. But we do not know how effective these actions have been in reducing the spread of misinformation. And if they have, where were they more effective? Or where were they less effective?

So in the project that we've already been working on for the past two years, which is called Co-Inform, which was also about misinformation and kind of general misinformation, rather than any health-related ones, we've been very busy developing tools to help us do that kind of tracking and analysis. So for example, we started to find out that there are some factors, such as age, such as the geographic location, such as your familiarity with the topic of the misinformation, and the impact of those on your judgement whether to share that or not.

Another piece of work that my team has done is to look into your social values and how you use that to make a judgement of whether you share something or not, regardless of whether you believe in it or not. So there are many factors that I don't want to talk too much about.

But one of the things, for example, I found today was that there was a 20% rise in the price of garlic back in February. And if you look for that, you will find that many websites, videos on YouTube, they're linking this to the myth that garlic with help prevent you from catching the virus, which is obviously false.

But then there are some other ones which are more trustworthy, like the financial terms, who would give a better explanation to that, which is that, well, actually, 80% of the production and export of garlic is from China. They were heavily impacted by that. It was a disruption in the supply chain, and so that might have led to that rise. We do not know, of course, whether misinformation had any impact on that. This is what we would like to understand-- how misinformation and the disruption to supply chains could come together to give us a better understanding of how these things are going.

The last thing I wanted to mention is that we are computer scientists. We can develop, for example, models, AI technologies to help automatically identify whether something is likely to be misinforming or not. But that's not what we're focusing on. What we're trying to focus on is that, look, there are many experts out there who are constantly fact-checking, like I mentioned the WHO and many others. How can we use their outputs to intervene a little bit more effectively, to spread the correct information more widely, and more perhaps targeted a bit better to the people or to the social media or to the environments where they are most needed? So how can we use tools, use technology to take all of that output from them to help us understand not only what is spreading and why, but also how we could develop effective strategies in tackling them?

I'll stop here and pass it to others so that others can speak.

**RACHEL MCMULLAN:** Great. Thanks, Harry.

I can see we've already got a couple of questions coming in that relate a little bit to fake news. So we'll come back to those once everybody's had a chance to speak. So I'll move on now to Helen.

**HELEN WIMALARATHNA:** Hi. So as an epidemiologist, until very recently, when I've told people what my job is, they've looked at me a bit blankly and said, but what is that? Now every day we turn on the radio, the television, we pick up a newspaper, and we can see commentary by epidemiologists. And we're flooded with epidemiological terminology in the mainstream media and in social media. And we're really asking a lot of people suddenly to be understanding these concepts that were completely unfamiliar to most people just a few months ago.

So I've been lucky enough to be involved in some sort of public communication about what's going on and how we understand the coronavirus epidemic. And I keep meeting the same questions being asked again and again. And they really are reflective of the fact that people are trying to understand the news, and perhaps not with the resources that we have maybe as scientists to understand.

So if I just run through very quickly the sort of three broad topics that keep coming up, a lot of people want to know why aren't vaccines and drugs being rolled out quicker. And this, I think, is very natural. And I'm sure Professor Harry could say more on this. But I think it's very natural with all of this bad news going on, when there's a glimmer of hope, when there's a news report about a candidate drug, people want to grab onto that. And they think, well, why shouldn't we just throw away everything at this horrible virus and just try it?

Obviously, we know that there's a very stringent process of clinical testing trials that either vaccines or drugs have to go through. And there's a very good reason for that, because we need to know about the safety and the efficacy of the drugs. But it's understandable that people would feel frustrated by this if they don't have that background knowledge.

I also keep coming across people basically generalising from the specific. And we've seen over the past few weeks some really harrowing stories of awful, but unusual events taking place. So we had, for example, a couple of nurses, two hardworking, dedicated nurses in their 30's dying of this awful virus. And it's right that these things should be reported. But it does tend to give people a kind of skewed idea of what the risks are to certain groups.

So I was then asked-- I was in fact rung by a few media channels asking for an explanation of why so many health care workers are dying. Well, when you actually have a look at the figures for the first two months of the epidemic in the UK, you'll actually see that London bus drivers were at something like roughly a 25 times higher risk of death from coronavirus than were nurses in England. Now, this is obviously not to belittle the risk that NHS workers are being put at, but it shows the reporting bias and how we have to be careful and we have to be responsible in what is put out and how it's framed so that people perhaps without an epidemiological background, without a scientific background can make judgments for themselves about how to interpret the information that they receive.

The biggest question, which I keep hearing, and which is really relevant today-- in fact, it's probably about now that they're sitting on a Skype conference like this discussing much more important things-- when can the lockdown be lifted? Obviously, everyone wants to know this. This is tough. It's only been what? Two and a half weeks so far, and it feels like a lifetime.

And so people want to know why the lockdown can be-- when the lockdown can be lifted. Now, this is a great example of when a little bit of knowledge is perhaps more dangerous than having no

knowledge. So I think a lot of people have a notion that an epidemic will peak and then fall. So this is true.

I'm about to be photobombed, and I apologise.

So an epidemic will reach a peak, and it will fall. And we think of the classic examples-- forgive me-- for example, of cholera in the 1850s in London.

**CHILD**: Mummy, have a look at this.

**HELEN WIMALARATHNA:** One second. Excuse me. Darling, just go and see Apati.

**CHILD:** Do you like it?

**HELEN WIMALARATHNA:** So we think of the classic examples of cholera in the 1850s.

**CHILD:** Mummy, do you like the picture?

**HELEN WIMALARATHNA:** So we see a peak, and then we run out of susceptibles, and the epidemic is over. OK. This is basically true of a closed population.

Now, by isolating ourselves, by shutting ourselves in our house with our kids at the moment, working from home, we're basically closing the population. If we reach the peak, which is supposed to be over the next week or so, we're supposed to see the beginning of the peak. If we reach the peak, and then suddenly lift the stringent rules on social isolation or physical isolation, then we're going to see a second peak. And it's going to be larger, because we're basically going to be flooding the population with new immunonaive individuals. So we're going to get another huge peak, and we're going to be in a much worse situation than we were in.

So I think it has been stressed by government that we're waiting for the evidence. And I think we can't get enough of it. It is all about the evidence. But I feel it's very frustrating perhaps for people to be watching this without a scientific background, without a knowledge of how these things are actually processed, and to try and understand what's going on. And I think that lack of understanding feeds into some of the sort of conspiracy theories and things that I think we're going to touch on throughout this conversation.

Thank you.

**RACHEL MCMULLAN:** Thanks, Helen. That's great.

Just before we move on to Jon, don't forget you can submit your questions for our panel to answer by emailing the email address that's at the bottom of your screens, which is STEM-News@open.ac.uk. OK. So I'll pass over to Jon now.

**JON GOLDING:** Thanks a lot, Rachel.

So I'm just going to talk a little bit about a couple of articles we have written for Open Learn, which is-- I think you're going to be receiving the web links to those. The first one was written by David and Martin Bootman and I. And it was an article in four parts really just talking briefly about, what are viruses? What is coronavirus? It also dealt with aspects of things you might have heard, such as herd immunity, which Helen will be able to tell you more about, and the response of our bodies to viruses, which David is an expert on and will be able to tell you more about.

But I can tell you a little bit about viruses in general, and then specifically about coronaviruses. So the word virus is everywhere at the moment. And everyone's getting all panicky about it. It's as if they've just appeared from nowhere. They've been around for millions and billions of years from the very dawn of life.

They are small strands of genetic material. So they're either DNA or RNA. And they're in a little protective package that allows them to be out in the open.

Coronavirus, for example, is an RNA virus. It's about 30,000 bases. Now, put that in context, the human genome has got about 10,000 times more genetic material than coronavirus.

Viruses, they're very, very tiny. They're about 1,000 times smaller than a human hair, or about 50 times smaller than a cell. And they're not alive. I mean, people think that these are alive organisms. They're not really. They don't have any kind of cellular machinery. They don't have any kind of power supply, any energy of their own.

What they do, though, is they rely on getting into a host cell, and then using their genetic information to reprogram the host to make more of the virus. And they infect everything. It's not just mammals or anything like that. They infect all cellular life. Bacteria have got viruses. Plants have got viruses. Animals have got viruses. And there are thousands of different types.

And if you, for instance, look in just a millilitre of seawater, you'll find about 100 million viruses in that. And if you look in lake sediments, for instance, you've got about 20 billion viruses per gramme of mud of lake sediment.

Because of this little package that they're inside, they can remain viable outside of a host for several days or weeks. If you keep them in clean, cold water, they can last for more than a year.

There are loads of theories about where they actually originated from. It could be that they came originally from small strands of DNA that are able to cut and paste themselves within the genome. These are little sequences. They're common in plants and bacteria, but there are some also present in animals. And it's thought that it could be possible that a bit of this transposable DNA, if it acquired a protective coating, then it could easily be transferred from one cell to another. That could be the beginnings of how viruses started.

And of course, they're not all bad. Everybody talks about viruses as being some sort of evil thing that's going to kill you all off. They're not that. They can be used for good purposes.

So for instance, with antibiotic resistance problems at the moment, there's a whole field of work looking at viruses that infect bacteria and kill those. They're being engineered and looked at as a way of overcoming antibiotic resistance. And they're called bacteriophages.

I would just tell you a little bit about the coronavirus itself. So this is a family of enveloped viruses. So it's got a kind of an internal crystal structure called the nucleocapsid that's got the RNA inside it. And then outside of that is an envelope or a cell membrane that's been taken from the host cell.

They use RNA as their genetic material, positive strand RNA. That's exactly the same type of RNA that gets used in ourselves to make instructions for proteins. So this virus is immediately ready to go as soon as it gets into a cell. It can start making protein immediately. It doesn't have to do anything else like some other viruses do.

And it's relatively simple. Although it's got quite a big genome, it only is encoding between 8 to 14 different proteins, depending on the type of coronavirus. So the bigger ones, SARS, and this new one, coronavirus 2, they make 14 different types of proteins. The main ones are called membrane and enveloped spike proteins. And they sit in this outer membrane part.

And that's why it's really important to be using detergent and handwashing, because detergents will break apart that bilipid membrane. And they just break the whole virus apart. So it's essential to be using handwashing with soap and not just water. Soap will break that apart.

And there are loads of different types of coronaviruses. So there are ones that infect mammals and birds. And they're called slightly different diseases. So you tend to get more kind of gut diseases in cows and pigs. And in chickens and humans, you tend to get more of the lung upper respiratory diseases.

There are seven different types that infect humans. There's the awful one, the severe ones, which are SARS and MERS, and now this new coronavirus 2. And then there are more mild ones. There are four of those. Two of which are thought to cause around about 10% to 30% of the common cold. So if you've had an ordinary winter cold, it's possibly been a coronavirus that's done that.

But they belong to a much larger group of RNA viruses. And those include things like influenza, hepatitis, measles.

In fact, the name coronavirus comes from the appearance of this sort of crown on the virus, which are these spike proteins. And the spikes are very important for the virus. They allow it to interact with a protein on the surface of mammalian cells called ACE-2, which is Angiotensin-Converting Enzyme 2.

And the spikes also contain what's known as a cleavage site. So once they're attached to the cell, this thing acts like a knife, and it allows it to get into the cell and dump the RNA inside.

So that's just a little bit about the biology of these things. And I think I'll probably stop there, because there's lots of stuff that could be said about that, but I'm not going to.

**RACHEL MCMULLAN:** Great. Thanks very much, Jon.

So our final panellist in this session is David. So I'll just hand over to him now to just introduce his area for a few moments.

**DAVID MALE:** OK. Thank you, Rachel.

Just very briefly, the immune response to a virus is very variable, depending on the virus. But for this particular virus we're talking about, it's an acute virus. And therefore what happens in the early stages of infection is particularly important.

And one of the key things about the immune system, especially with a new virus like this, is that there aren't very many cells available to actually fight the infection. So in the first phase of the infection, what happens is the immune system expands the number of cells that are able to deal with this particular virus. And while this is occurring, there's a race on as the virus is trying to reproduce and so are the cells of the immune systems. And during this period, there is a whole series of delaying actions that take place, mediated by some sort of cellular and intercellular signalling systems.

Later on, one of the things that I get a lot of asked a lot of questions about is, how long will immunity last once we have it? Why is it that some people are getting better really quite quickly, whereas other people are progressing on to quite serious pathology?

Of course, with a new virus, we don't know the absolute answers to this, because we've only known about this for three months. But we've got some leads from previous coronaviruses, like the SARS and MERS that Jon's just mentioned.

So for example, there has been some considerable discussion as to whether the difference between whether a person recovers and doesn't recover is whether at a particular phase, about maybe about a week into the infection, whether the strength of the immune response is actually more damaging than the immune reaction itself. Now, clearly, the immune reaction is very beneficial and absolutely necessary, but there has been debate over this. It's sometimes called the cytokine storm theory.

However, if a person has got antibodies, developed antibodies, they should give them quite persistent and long-lasting immunity. Antibody lasts for many months, or are detectable even for years. And also, the immune system develops what are called memory cells, which are able to reactivate very quickly should a virus return again.

That, of course, depends on the virus not mutating so that it can evade the immune response. And viruses do have a lot of tricks up their sleeve to evade immune responses. So that's an area that you might like to ask questions about.

And very briefly, the other thing that turns up again and again is I think people have got to the stage where they understand now the difference between a test for the virus, which is looking for the virus genome, usually on swabs from the nose or the throat, and the possible tests for the antibodies. Antibodies arise much later at the time that the virus disappears, but they can tell you whether you have been infected by the virus. So I think people now understand the difference between those two types of tests.

But what there is still a lot of confusion about is whether these tests have to be done in laboratories or whether they might be able to be done at home, a home testing kit, like a pregnancy test kit. That is, to my view, some way off.

And then, of course, further down the line, as Helen mentioned, why is it taking so long to get a vaccine? And we can talk a little bit about the various phases of clinical trials that people have to go through before they, first of all, realise that a vaccine may be efficacious. And you don't want to be taking all the world's vaccine production facilities over with a vaccine that is not effective and actually scaling up to produce a number of vaccine doses that are needed is going to be another considerable effort. And that is why this is taking longer than people might imagine it ought to.

I'll leave it at that, Rachel.

**RACHEL MCMULLAN:** Lovely. Thanks very much, David. So thanks very much to all of our panel for their comments so far.

What I'm going to do now is I'm going to open up the floor for the panel to answer some of the questions that you've been submitting. So I'm going to start with one that's been submitted by a couple of different people, including Mark Duffy. And it's a question probably slightly based on the fact that the weather for this weekend is predicted to be really good, and we're all thinking about this summer. And it's "Is the spread of the virus likely to be affected by warmer temperatures? And if so, why would this be?"

**JON GOLDING:** Well, there's been a study out that often gets quoted looking at how long the virus survives outside of a host. And they put the virus, coronavirus, this particular coronavirus onto various different surfaces, and then they test it after days whether it's still able to infect some cells in a dish. And they found that on smooth surfaces, metal surfaces, plastics, it would remain infectious for about four days. On cardboard or paper, a couple of days. Of course, if you get it near any detergent, then it's gone within about five minutes.

But also-- so it will survive for different amounts on different surfaces. But one of the things that generally tends to kill off viruses quite well is ultraviolet radiation. So drying ultraviolet will tend to deactivate viruses.

If you have very cold weather, so if you keep stuff in the fridge-- well, that's one of the reasons you keep stuff in the fridge is to stop it going off-- the virus will last longer. So yeah, the answer to the question, I think, is if it's exposed outside to strong sunlight, then that will help to kill it off. What it won't really do is have any effect on people that are already infected.

**RACHEL MCMULLAN:** Great. Thanks very much, Jon.

So we've had a number of questions that have come in around new stories that people have heard and questions about whether these are fake news or whether there's any truth in them. So one of the questions that's come in is "I'm reading a lot about the virus having started in a lab in Wuhan. Is this fake news?" And in relation to that, there's also somebody that has asked about-- someone's asked, "I know there have been predictions of viruses being released from the melting ice poles. Are people surprised about where the virus originated from?"

**JON GOLDING:** I mean, I don't have a response to talk about kind of the social interactions with that. But I can answer stuff about whether it was created in a lab or not, and where it actually came from in the first place. I don't know how you want to do this, Rachel.

**HARITH ALANI:** You go ahead, then I'll comment.

**JON GOLDING:** Well, regarding the lab theory, no, complete bunkum. So there was a paper in Nature Medicine, which was published on the 17th of March by Chris Anderson's team that completely debunks that theory. And there are about three or four reasons why it's not the case.

So the structure of the new coronavirus 2's spike proteins, now, those proteins most strongly resemble those that are found in a type of animal called a pangolin. So the pangolin coronaviruses, it most strongly resembles those ones. The pangolin coronaviruses have been very, very poorly studied. There's hardly anything in the literature about them. And they never normally cause illness in humans. So if you were trying to deliberately make a virus against humans, you wouldn't start from that perspective.

Another piece of evidence is that computer simulations of the receptor binding domain that interacts with that ACE-2 protein on human cells, it's not that optimal for this particular virus. It is better at attaching to ACE-2 than the SARS virus. But if you were just to do this on the basis of computer simulations, if you were designing a virus, you would choose sequences that are much better optimised to cause it to bind to ACE-2. You wouldn't choose the sequences that it currently has at the moment.

And finally, if you were trying to make a coronavirus against humans, you would probably want to insert sequences from other viruses that are known to increase infection and the level of disease. So for instance, you might want to put in some sequences from Ebola virus or something like that.

There's no evidence of any kind of such copy and paste going on here. So it really does appear that it's come via a natural route.

So where did it come from if it didn't come from some evil lab? So its overall sequence, the RNA sequence, is most closely similar to coronaviruses that circulate in bats. So that's the overall sequence.

If you look very specifically at the spike protein, that's got a sequence that is most closely related to pangolin animals. So that suggests that coronavirus 2, it came from bats via pangolins, and then to humans. So it's not some sort of mutated version of SARS.

And there's also the possibility that it could have been circulating in humans for quite some time as some sort of asymptomatic or mild disease. And then within humans, that mutated to become-- to give more serious symptoms. That's possible. Although, in general, viruses tend to evolve the other way, to give less serious symptoms, because after all, a virus that kills its host, it reduces its chance of getting passed on to anybody else.

So yeah, was it created in the lab? No.

**HARITH ALANI:** So one thing I always encourage people to try is to look for these facts. The problem is that there are new rumours and myths coming out every day. And so even if you go to the official health organisation websites that try to debunk these myths, the chances are they haven't caught up with all the new rumours that have come out that day perhaps.

So one thing I would encourage everyone to try and do is to put that claim that you're trying to check on a search engine, like Google or others, and put fact check next to it, and see what you come up with. All of these search engines now have boosted their efforts to try and tackle misinformation about COVID. And so what you will see is that many very legitimate websites will come out that will give you the right information. These are authoritative websites, not any kind of fake news websites. And there are unfortunately many of them.

So try to search for the facts for any claim that you find. And chances are you will find a lot of very clear information that will tell you whether this is something that's true or not.

Unlike the other panellists, I know nothing about viruses. So this is my strategy to verify anything that I come across, because who knows what could be true and what isn't?

**RACHEL MCMULLAN:** Brilliant. Thank you, guys.

So there's been a couple of questions coming in about the tests and the type of tests. One question has come from K. Cody, which asks, "Are there any antibody tests currently being used for other viruses around the world?" And then kind of following up from that, "Can we repurpose and reuse these tests to test for COVID-19?"

**DAVID MALE:** OK. I'll take that one, Rachel.

**RACHEL MCMULLAN:** Yeah.

**DAVID MALE:** There are plenty of tests, antibody tests, for various viruses around the world. Mostly they're used in laboratories. And many of them are based on something called an Enzyme-Linked Immunosorbent Assay, or it's called an ELISA for short. And essentially, people will have material

from the virus called an antigen, something that is recognised by the antibody, and they will use the antigen from the particular virus they're interested in to detect the specific antibody.

And there's been a paper came out about two or three weeks ago showing some very effective laboratory-based ELISA assays that could distinguish very effectively between people who have got antibodies and have been infected and people who had not got antibodies, or had got antibodies against different coronaviruses. And that's very important, to be able to distinguish having an antibody to this coronavirus to having an antibody to one previously.

Now, when it comes to the accuracy of the test, first of all, obviously, you want them to be 100% accurate, because you don't want to tell somebody that they're immune to something when they're not. But also, you want them to be specific so that they detect this particular antibody, and not possibly antibodies against a previous coronavirus that you might have had as a mild infection.

So these antibody tests for coronaviruses have been around for quite some time. But an antibody test for this coronavirus has only just been developed. And it's workable in laboratories. And it should be rolled out as a laboratory test, I would think, in reasonably short order.

The question after that is whether we can actually get home testing for this. And the kind of home tests that people use are based on things like the kind of things that look like a pregnancy test, where you-- in this case, you would have to do it on blood, because you don't get antibodies in urine, or you certainly should not.

You can get antibodies in saliva or in tears and other secretions, but they're a little less reliable. So it would have to be a test based on a finger prick from blood or something like that. The trouble with that is that they are likely to be quite a lot less reliable. For example, when pregnancy tests came in, to begin with, they were about 90% reliable, which is good enough for knowing if you're pregnant, but not good enough for knowing if you've got antibodies to coronavirus. And now they're about 99% reliable. But still, any home-based test is likely to be further down the line and to be less reliable than a laboratory test.

**RACHEL MCMULLAN:** Great. Thanks, David.

So I've got an epidemiology question now, which might possibly be one for Helen. So Andrew Wilkinson has asked, "I keep seeing reports about there being a high proportion of asymptomatic cases, but the numbers seem to vary wildly, between 5% and 80%. Could you comment on that?"

**HELEN WIMALARATHNA:** OK. Yeah. So the reason the numbers vary wildly is because we don't really know. They can make reasonable estimates. So the reason we don't know is because if you're asymptomatic, then you're not going to be tested at the moment.

We can make estimates based on closed populations. For example, you'll remember that there were several cruise ships where some infections arose, and then the people were basically quarantined within the cruise ship. And then before everyone was let off they had to be-- they all had to be tested. So situations like that, for example, where you can see who's been exposed and test the prevalence of infection, and then also do questionnaires for prevalence of disease symptoms, then you can use those data to extrapolate and estimate the prevalence of asymptomatic infection.

So you'll notice that I use the word extrapolate. So every time we extrapolate from data, we have to be really careful. So for example, a cruise ship population is not representative of the population at large. You'll see a different age structure, a different socioeconomic structure, and so on.

So the reason why estimates differ wildly is because we've got massive margins of error. And we don't really know, and we still won't know until we see lots of community testing. And that doesn't mean testing everyone, but it means, for example, sentinel surveillance, where an nice stratified random sample of people symptomatic and asymptomatic are tested. And we can start to come up with some more realistic estimates for the degree of asymptomatic infection.

**RACHEL MCMULLAN:** Thanks, Helen.

So this one's perhaps a little bit more on the fake news side of things. So we've been asked, "News reporting is always selective. So presumably, it can be just as impactful as fake news in the sense that people will be making judgement about what's important and what's not based on the news that they see." So I wondered if the panel would like to comment on that one.

**HARITH ALANI:** Well, that's a big question. Yes, of course. Misinformation also spreads virally as well.

One thing we need to always remind ourselves is, what's the motivation for creating this information in the first place? And there are various ones. So in some cases, I've seen cases during crises where people were coming up with rumours for the fun of it, because they want to see those circulating. And then they will come up with the exact same rumour whenever there is another earthquake, for example. One of those crises that tends to happen.

So here, for example, AI technology is pretty good, because it has already seen that one before. It can pick it up again.

But there are many cases where we need to dig a little bit deeper to understand why there are these fake news websites, why they are these people on YouTube who are coming up with these videos, making all sorts of conspiracy theories. And many of them are about COVID as well. It's because, unfortunately, there is a financial model to support it. The more people who come and watch you, the more people who would register to your YouTube channel, the more people who would visit your website, the more you get from advertising, the more the marketing companies will target you.

Some of these people that were on the news recently, they have about a million followers on some of these social media. So there is a very strong financial benefit for basically fabricating information.

And why do we fall for that is because, like was mentioned earlier, we're looking for easy explanations. We want to find something that will get us out of that situation. And in many cases, people have mastered the art of fabricating information, of manipulating information. So they know exactly how to attract us to that by tapping into our fear, into our worry, into our anxiety.

Yes, the media might pick up some of these things. But they are not just us, but there are quite a few research teams who are monitoring social media just to see what's spreading over there. In some cases, you are able to track the source for that, or what has amplified it. So if that was mentioned somewhere on the media or on the news, then, of course, that particular one will be amplified.

And psychologists have been looking into these things for quite a while. And they always warn that if you repeat the myth, even if you are debunking it, you're actually indirectly supporting it somehow, because people will hear it. Even if they see the debunk information to go with it, that myth will reside in their memory. So we also-- the media, as well as us, we always need to be very careful how to phrase the rumour that you're trying to debunk by making sure that it's crystal clear that you say it's false before you even describe it. So you see that sort of style now being used more often.

So there are many factors that influence these things. And unfortunately, there are no ways of very easily identifying what's for a particular rumour.

**DAVID MALE:** I just wanted to pick up on the point of selective reporting, which was in the question there. And one of the things that I've noticed is that you may have a briefing. And then the questions will go out to the political journalists and then the economic journalists who will ask questions on those. And then quite a way down the line they may ask the scientific questions. And just by having perfectly good information by the questions coming in from a particular slant, you may get a different kind of message come out from them.

**RACHEL MCMULLAN:** Thank you. Also, I've got two people who've asked a kind of a similar question. So I'm going to apologise for taking your questions and kind of shoving them together. But this question comes from Nicola and Amy. So in America, there's a much higher death rate reported for black Americans than white Americans. Is there any evidence, do you think, that there is a genetic explanation as to why people are differently affected by the virus or is it more likely to be socioeconomic?

**DAVID MALE:** Do you want me to start on that one?

**RACHEL MCMULLAN:** Yeah. Go on, David.

**DAVID MALE:** I mean, that's quite a hot potato, that one.

**RACHEL MCMULLAN**: Yeah.

**DAVID MALE**: I think there's very, very good evidence that it is correct that there is a higher incidence in black Americans than in the rest of the American population.

I do think that there are important genetic elements to it, but I think probably in America, it may be a large portion of it is socioeconomic. That is just my view. But we could certainly spend some time going into genetic variability and why some people are more susceptible to developing serious disease than others.

Jon, you wanted to say something there, I think.

**JON GOLDING**: Not really. I mean, it's just the news reports that were going around this morning on exactly that topic. And these people, they do tend to be more the front line people. They're the people driving the buses. They're the people that are nurses. They're the people that can't really afford to self-isolate, because they need the money. So they're more likely to get out and catch this virus.

So I think there's a very strong element of socioeconomics on that. But we need more data to get a look at that. And that's more sort of leaning towards the epidemiology side of stuff and the stats side of stuff.

Yes, there are differences in the genetics of people, and also age as well. And that's going to come out more and more. I mean, there are some links to-- in patient genetics there are links to something called interferon-stimulated gene response that can exacerbate the seriousness of this. There's an epidermal growth factor response that can also exacerbate the seriousness of this.

If you've got an underlying lung condition, so if you've been smoking, for instance. Even the presence of certain bacteria in the lung can make the condition much worse, stuff that normally wouldn't really

bother you. But the combination of that plus coronavirus will make it worse. And so there's genetics and socioeconomics.

I don't know if anybody else wants to comment on that.

**RACHEL MCMULLAN:** Anyone else? No? Thank you.

So these are a couple of questions. One from Bridget Harris here. And then apologises, I can't find the name of the other person. They're slightly related. So does acquired immunity to other coronaviruses in any way help when exposed to this coronavirus? And can we catch it again?

**DAVID MALE:** Right. That's an interesting question. As Jon described earlier, the part of the coronavirus that attaches on to a cell is part of a spike protein. And that is one of the most highly variable parts of the virus between different strains or different types of the virus.

It is likely that any previous immunity we've had to coronaviruses will not be effective against those outer proteins of the virus, which help prevents the virus attaching to a new cell. So in terms of the previous antibody response to another coronavirus, probably not.

The inner parts of the virus, the nucleocapsid, the N protein, the envelope, and so on mutates and are less variable between different viruses. And those parts of the viruses are presented on the outside of infected cells for sort of review and surveyance by lymphocytes, which can potentially recognise them.

Now, it is possible that the immune response against the inner parts of the virus of an older virus might still have some effectiveness against the current one. But those kind of immune responses come in slightly later down the line. So although it is possible, I think it is unlikely that there will be any really strong effective immune response from previous coronaviruses that will protect us against this one.

And then the second part of the question was how long will immunity to this one persist. Based on previous coronaviruses, I would have said it's going to be probably years, provided it doesn't mutate significantly.

**JON GOLDING:** I mean, that's when it-- it mutates. It's an RNA virus. They do mutate very fast. But within the RNA viruses, it's actually quite a slow mutating one. It's about a four times slower mutation rate than influenza, which is a related virus. So within RNA viruses, it's relatively stable.

**DAVID MALE**: There's two differences between this virus and flu in terms of the potential mutation. One is that the genome of flu comes in bits, and can therefore be sort of reassorted between different viruses more quickly. And the second thing is that it's possible that coronaviruses, because they have a long genome, have some proofreading capacity in their replication machinery, which influenza lacks. And actually, coronaviruses do develop the infection just slightly more slowly than coronaviruses and some other upper respiratory tract viruses.

So there are some differences here. And I agree with Jon that the consensus view is that this is a slightly slower mutating virus than some of the other ones that concern us.

**JON GOLDING**: I mean, the parts of the virus that's really essential for it is this binding to the ACE-2 receptor. So it probably is mutating, but you're not seeing a lot of those mutations, because if it fails to stick to a cell, then that mutation is lost. It can't get into a cell anymore.

So there is a selection pressure on the virus to get into cells. And that's why some of the drugs that are being designed at the moment-- I mean, they are targeting this spike protein.

There's one that's just being looked at at the moment. And I think it's a recombinant form of ACE-2. The idea is that you can use that to stick to the virus to swamp it, to prevent it getting into cells. It just gets coated in ACE-2 protein. And then it's no longer able to get into a human cell.

All right. Sorry.

**RACHEL MCMULLAN**: Thank you. So I think we've just got time for a couple of final questions. So hold on. I'm scrolling through. So one question we've had coming. Is there any uniformity in the way that each country records deaths, but also cases of coronavirus? And then how do you determine if somebody has died from coronavirus?

**HELEN WIMALARATHNA:** So I'll start, but maybe some more people have something else to say on this.

So is there any uniformity? Yes, there's some uniformity. But it's not completely comparing apples with apples. So if you're in the habit, which I'm trying to break, of looking at the statistics daily and comparing across countries, it's really not very helpful. I know it's not very helpful, and yet I still do it.

They have-- don't forget we have time differences around the globe. And they have different reporting times. So it's very likely that you will end up comparing sort of half of one day with a full day from another country.

There is-- you've probably heard a lot of talk over recent days about the fact that, for example, deaths within care homes, for example, are not being recorded within the official corona death statistics. There are various reasons for this, but that is fairly common across countries. So the actual death statistics that you see will be more likely an underrepresentation of the number of people who have died with coronavirus.

That with word is important, because we can't say for sure that all of these deaths, that all of these individuals have died from coronavirus. In many, many cases, these people are suffering from multiple morbidities. Sadly, many of these people were in kind of end-of-life care in any case. And what we know is that the coronavirus is likely to have been a contributory factor, and that at the point of death, they had coronavirus. But we don't know to what extent that was actually a causal factor, and whether if it had been taken in isolation would it have been a causal factor.

So in terms of the way in which they're reported as well. Remember, there can be a lag, because of the kind of bureaucratic reporting systems, and the way it takes a while for the data to go through the correct offices.

**RACHEL MCMULLAN**: OK. Thank you. So we've just got time for just one more question before we wrap our session up today. So this one comes from Margot Edwards. It's "Is there any truth that the virus can transmit itself from people to animals or is this another example of fake news?"

**JON GOLDING**: Yes, it probably can. Coronaviruses, they infect lots of different mammals-- cats, dogs, pigs, sheep, chickens. And these ones, they've still got this ACE-2 receptor. It might be slightly different, but it still an ACE-2 receptor. So there's no reason why it can't do that.

Remember where this thing probably came from. It came via bats through pangolins, and then into humans. So there's no reason why it can't go back the other way.

And there was something in the press a couple of days ago about a tiger in New York that had contracted coronavirus, too. So it can go in either way. And this is probably where it sits for most of its-- well, obviously, it's not alive-- but for most of its life, it's probably just circulating in a reservoir, such as bats. And it's only when those come into close contact with people that they can then springboard into people.

**RACHEL MCMULLAN**: Great. Thanks, Jon. So I think that brings us to the end of our session today.

Thank you to everybody who submitted a question. I'm really sorry that we didn't get time to answer all of them. And I'd just like to thank our expert panel for answering all your questions today. And thanks to you all for watching. And have a good day.

**DAVID MALE**: Cheerio.