HOUSE OF LORDS

Select Committee on Science and Technology

Corrected oral evidence: The science of Covid-19

Tuesday 19 May 2020

11 am

Watch the meeting

Members present: Lord Patel (The Chair); Baroness Blackwood of North Oxford; Lord Borwick; Lord Browne of Ladyton; Baroness Hilton of Eggardon; Lord Hollick; Lord Mair; Baroness Manningham-Buller; Viscount Ridley; Baroness Rock; Baroness Sheehan, Baroness Walmsley; Lord Winston (co-opted); Baroness Young of Old Scone.

Evidence Session No. 2 Heard in Public Questions 11 – 22

Witnesses

Dr Samantha Lycett, Group Leader in the Infection & Immunity Division of the Roslin Institute, University of Edinburgh; Professor John Edmunds OBE, Professor of Infectious Disease Modelling, London School of Hygiene and Tropical Medicine; Dr Rosalind Eggo, Assistant Professor, London School of Hygiene and Tropical Medicine.

USE OF THE TRANSCRIPT

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Examination of witnesses

Dr Samantha Lycett, Professor John Edmunds and Dr Rosalind Eggo.

The Chair: We turn to the next session. May I ask Lord Mair to start the session, please?

Q11 **Lord Mair:** My question is initially for Professor Edmunds, but I am sure that Dr Lycett and Dr Eggo would also like to comment. By what mechanisms does the virus transmit between individuals?

Professor John Edmunds: There are three routes of transmission for these sorts of viruses, including this one. There is droplet transmission when someone coughs or breathes out heavily. These are relatively large particles—obviously, they are not that large; you cannot see them—that contain a virus, and because they are quite big they settle out quite quickly. You need to be quite close to an individual to be infected in that way. That is droplet transmission.

You can get aerosol transmission, which is via much smaller finer particles. They can remain suspended in the air and virus can be contained in those.

The third route is what is called fomite transmission, which is where the surface is infected. Someone might cover their face when they cough and infect their hands. The virus gets on to their hands, and if they touch surfaces, such as a doorknob or something like that, the next person can touch that surface, pick up that virus and transfer it to their face, their nose, their mouth, their eyes, and infect themselves that way.

Those are the three main mechanisms. It is always a bit of a mystery as to exactly the role and the extent to which each of those mechanisms plays in transmission. From the evidence we have, it looks as if the two main mechanisms for transmission of this virus are fomite transmission—transmission from viral-infected surfaces—and droplet transmission—direct transmission through exhaled larger particles. That is where the 2 metre-rule comes into play, because those larger droplets would settle out much more quickly, so if you are staying more than a couple of metres away from an individual who may be breathing out or coughing, you are much less likely to be infected via the droplet route.

Lord Mair: Thank you. Dr Lycett, would you like to comment?

Dr Samantha Lycett: Yes, I agree that the mechanisms are airborne and to do with droplets. The size of the droplets and the amount of virus in each droplet can also make a difference. Also, the very small droplets might be able to go further and infect further down into the lungs. This might also be the difference between the mild cases and the severe cases, depending on where the infection occurs.

Dr Rosalind Eggo: I would add that there is some evidence of viral shed in stools, but it is not at all clear yet how important that is in transmission.

Lord Mair: May I follow that up and ask how, first, transmission

mechanisms and, secondly, transmission rates compare with other coronaviruses and with influenza?

Professor John Edmunds: They are quite similar. We do not know that much about the other coronaviruses; they have not been very well studied. The common ones cause relatively mild coughs and colds and they have just not been studied very heavily. MERS and SARS have been studied in quite some detail, and droplet transmission and transmission via fomites are again probably quite important, and there is probably some transmission via aerosols, but again it is probably less important than the other two routes of transmission for those viruses.

Flu is also relatively similar in that you have these three routes of transmission. Again, transmission seems to occur primarily through the droplet and fomite routes, which is why there is good evidence that washing your hands protects you against many of the respiratory viruses. It seems strange that hand washing would protect you, but we have pretty good evidence that it protects against respiratory viruses. This one is almost certainly no exception.

Dr Rosalind Eggo: I have nothing to add on that.

Dr Samantha Lycett: I have nothing to add apart from to say that I think you will ask us about the reproductive number later.

Lord Mair: May I ask a final question? Have any of the mutations of the virus so far affected transmission rates?

Dr Samantha Lycett: Although in the previous session you talked about there being mutations of the virus already occurring, it is too early to say whether any of them have really affected the transmission rate. This is partly because everything is confounded with where the virus is.

As for the rate of transmission from person to person, at the moment this is affected much more by the contact between people, the density of the people in the different countries, their lockdown regimes, and that kind of thing. This is completely overwhelming any effect there might be from one or two mutations. That is not to say that next year perhaps there might be a difference, but it is far too early to say at the moment.

The Chair: Do mutations always make transmission easier, or sometimes does mutation occur that makes transmission less likely?

Dr Samantha Lycett: They can occur either way. The normal way of thinking of mutations is that they are random mistakes, so for the most part they do not affect anything. There are usually one or two in certain key positions which, if they mutated, there might be some big effect from, but these ones are even rarer than normal mutual mutations.

Q12 **Baroness Manningham-Buller:** First, I need to declare an interest, which I should have done in the previous session, as chair of the Wellcome Trust.

I have a question about infectiousness. We have heard from the previous session that one of the successes of this virus is that it can spread from people who either have no symptoms or are hardly ill at all. What do we

know now about how soon from exposure to the virus people who will get it become infectious? How does that relate in most cases to them beginning to have symptoms, and of course in some cases no symptoms?

Professor John Edmunds: I did not hear you very well, but I think you asked when people become infectious post infection and when they develop symptoms.

Baroness Manningham-Buller: From what we know, how soon after exposure, given that some people never develop symptoms, or very few symptoms, do we believe people become infectious?

Professor John Edmunds: If we take the people who develop symptoms, which is a majority of adults, post infection it takes perhaps around five to six days, somewhere in that range, for them to first display symptoms. However, people are infectious probably a day or two before that, so you have this rapid increase in infectiousness, which starts from perhaps about day three onwards, which is obviously problematic from the point of view of control. Seemingly, individuals who are in what we call that pre-symptomatic phase are about as infectious as individuals who have overt symptoms. That is certainly a problem in terms of being able to control the virus.

Then there are individuals who do not develop any clear symptoms at all. As I say, there is a fair fraction of those. You would expect about 30% or 40% of adults to have no clear symptoms, perhaps no symptoms at all, or very mild symptoms that are easily passed over and missed. How infectious those individuals are is very unclear. You can identify virus from them. You can look at the levels of virus in samples taken from nasal or throat swabs, and you can compare the level of virus in those individuals versus individuals with symptoms.

The evidence is a little mixed. Some studies suggest that the levels of virus are just as high as in symptomatic individuals, whereas other studies suggest that levels of viral replication are somewhat lower in asymptomatic individuals. Overall, the evidence points to levels of virus probably being somewhat lower in asymptomatic individuals. How that pans out for infectiousness is not clear. There is no clear evidence of transmission from asymptomatic individuals, because it is hard to find those cases. There is certainly clear evidence of transmission from pre-symptomatic individuals, and there is certainly some transmission from asymptomatic individuals, but the level of infectiousness of those individuals compared with someone with overt symptoms is still not very clear.

Baroness Manningham-Buller: If they had overt symptoms but very minor ones, so they never went near a hospital, do we have any idea of the average time that those people are infectious?

Professor John Edmunds: It looks like you are most infectious around the time that you first develop symptoms, so just before you first develop symptoms and just after you develop symptoms. Again, if you look at the levels of virus that can be detected from nose or throat swabs, you can see that the levels of virus start to drop over the next few days. If you try

to grow virus out of those swabs, after about a week it is very difficult to do. You may still be what is known as PCR positive—that is the test used to detect viral RNA—and you may well continue to be PCR positive for another few days, but it is very difficult to grow virus from individuals from samples after about a week post onset. Individuals with more severe disease who end up in hospital continue to shed virus for longer than the average person.

Baroness Manningham-Buller: Thank you very much. Do Dr Lycett or Dr Eggo have anything to add to what Professor Edmunds has said?

Dr Rosalind Eggo: No, I have nothing to add.

Dr Samantha Lycett: No, nothing to add.

The Chair: May I come back, Professor Edmunds, to what you said in response to Baroness Manningham-Buller's question, to confirm it? Did I hear you correctly that as many as 30% to 40% of people who may have acquired the virus are totally asymptomatic? Secondly, the current evidence, which is lacking, suggests, according to what you said, that they might be less infective. The logic is surely that if they are infective before symptoms start, they must be as infective.

Professor John Edmunds: Yes, I did say that perhaps 30% to 40% of adults have subclinical infections—infections that are so mild that they do not record any symptoms at all, or that are mild and very easily passed over, and it is only on very intensive questioning that they would come up with certain symptoms that may be Covid related. Dr Eggo has also done a significant amount of work on this. It seems that the fraction of people who are asymptomatic may be related to age, and that children would be more likely to be asymptomatic or have very mild symptoms.

Dr Rosalind Eggo: Our work so far has found a much lower fraction of infections in those under 20 to be symptomatic. Perhaps as little as 20% of those infections show clinical symptoms, rising with age, so perhaps around 75% at least of infections in older adults over 70 show symptoms.

As Professor Edmunds said, it is very difficult to identify just how infectious people are who are subclinically infected—completely asymptomatic or with very mild symptoms. The science community is working on that from a variety of angles. Our work is using transmission models to try to understand that, but people are also looking at different viral loads in saliva and the nasopharynx of both the clinically infected and subclinically infected to try to get at this question of infectiousness and subclinical infections.

Q13 **Lord Winston:** What is our best estimate of the R0 value in Covid-19? What factors affect the value of the virus? We have covered some of this stuff in the previous session, but you will have been present, so it would be good to hear your views on this.

The Chair: The question is: what are the best estimates of the R0 value, and what factors affect the R0? Is that correct?

Lord Winston: Yes, it is indeed.

Professor John Edmunds: The basic reproduction number for this virus seems to be somewhere between 2.5 and 3, which does not sound a lot but is plenty. Each person will infect on average two or three other individuals, so very rapidly you have an exponential increase in cases. Factors usually affecting R0 for these sorts of viruses include the setting—an enclosed setting, for instance a care home, a prison, or indeed a cruise ship. There you might expect the reproduction numbers to be somewhat higher. There is certainly evidence that the reproduction number in the "Diamond Princess" was higher. In those enclosed settings, with large numbers of individuals who come into quite close contact with each other for long periods, you can get higher reproduction numbers.

Dr Rosalind Eggo: I would add that the R0, the basic reproduction number, is what you see at the very beginning of an epidemic, when there is no susceptibility, and no interventions. Once you have interventions, the effective reproduction number is always lower than the basic reproduction number. Once you have immunity, that starts to push it down as well. We measured the basic reproduction number right at the beginning of the epidemic, in the UK and elsewhere.

Dr Samantha Lycett: I would repeat that the R0 number is the number at the very start. There have been a lot of studies in different countries trying to measure the R0 number in their countries. Typically, the values for this range between 2 and 4. For SARS the range was perhaps 2 to 3. For the 2009 pandemic, it was lower than that—between 1.2 and 2.3, and about 1.5 overall. As has already been mentioned, this is the value at the start, before you have any restrictions in place.

Lord Winston: Could you add what the R number would be for the other coronaviruses, in particular for the coronavirus that causes influenza?

Dr Samantha Lycett: Yes, the R0 for SARS looked like it was between 2 and 3, and for H1N1 it was about 1.5.

Dr Rosalind Eggo: The 2009 pandemic was influenza, not coronavirus.

Dr Samantha Lycett: Sorry, yes.

Lord Winston: I think Lord Ridley has a rather interesting question about the R number, so I will leave that to him.

Q14 **Viscount Ridley:** I declare my interests as a fellow of the Academy of Medical Sciences, president of the Centre for Life in Newcastle, and a shareholder in a diagnostic testing company called QuantuMdX.

My question is about R and R0, but to some extent it has been answered. Lord Winston said that I was going to ask the interesting version of his question, and the question is: what parameters are used to estimate R0? What I am getting at here is whether R pops out of a model or we get it from empirical data.

Dr Samantha Lycett: Typically, R or R0 is calculated afterwards as a composite. It comes from the transmission rate and the recovery rate, but depending on the type of model there might be some other rates that

also go into this number. The transmission rate and recovery rate are estimated more from the data.

Viscount Ridley: How heterogenous will R be? Professor Edmunds mentioned that transmission is bound to be more effective in closed settings. If you get control of the infection in hospitals, care homes, ski resort bars and places like that, does the average R for the population come down dramatically when you are dealing merely with socially distanced people, for example?

Professor John Edmunds: Yes. It is a complicated average, but it is an average number of secondary cases that each case generates. You have to think about what a typical case is. Over the course of an epidemic, a typical case will change somewhat. Since the peak of this epidemic, for instance, we have seen a shrinking of the epidemic in the community but a continuing epidemic in certain settings—in hospitals and in care homes in particular.

The effect of the lockdown has been very effective at reducing transmission, so the reproduction number has come down, but the epidemic has shrunk in to the places where it is continuing. A typical case now is somewhat different from a typical case some weeks ago. We have seen the reproduction number go up because we are seeing a reflection of this shrinkage of the epidemic into its more hard-to-control settings, which are hospitals and nursing homes. The reproduction number was lower than it is now, but I think people have misunderstood that. It is not that widespread transmission in the community has gone up again. I do not think there is evidence of that—not yet, anyway. It is just that the epidemic has shrunk into places that are harder to control.

Viscount Ridley: What you are saying there is moderately hopeful, because the relatively high R places are the only places where it is happening, and the R is pretty low in the community at the moment because of the measures that have been taken.

Professor John Edmunds: Correct, yes, but the places where it is happening, or where the epidemic has been concentrated over the last few weeks, are of course very important for many reasons. Those are the most vulnerable individuals in our community generally, and cases occurring in those settings result, unfortunately, in large numbers of deaths. That has happened and will continue to happen until we get on top of those epidemics. That is the first thing.

Secondly, these are not completely separated epidemics, of course. They seep back out in to the community, probably largely through healthcare workers or care home workers, who are picking up the virus themselves, unfortunately, bringing it home, and potentially infecting their family members and others. The epidemic is seeping out from these harder-to-reach populations back into the community.

Q15 **Viscount Ridley:** Chair, I hope you will not mind if I ask a quick supplementary. It goes back to a previous conversation about asymptomatic and pre-symptomatic cases. To what extent should we be encouraged by the new studies coming out suggesting that some people

have an acquired immunity to cold coronaviruses, a T-cell immunity, and this might be partly protective? Would that give us an explanation as to why some people seem to be unable to pick up this virus or are asymptomatic if they do?

Professor John Edmunds: That would be good news, of course. This is all very preliminary work. There is much that needs to be understood about the role of different aspects of the immune system, whether that is T-cell immunity or B-cell mediated immunity, and the role of antibodies. All this is very unclear at the moment. Of course, this is a brand new disease. It will take some time for us to understand how the immune system works, the duration of immunity, and what the different aspects of the immune system might give to individuals. It is early days yet, really.

The Chair: Do we know whether people who developed SARS and survived have immunity to this one?

Professor John Edmunds: You can measure their antibody responses. We often measure individuals' antibody responses, as that is the easiest thing to measure. You can see from studying survivors of SARS that antibody responses decline over time. After a couple of years, their antibodies have declined quite significantly. We can also see from other coronaviruses, the ones that cause coughs and colds, that individuals seem not to have particularly long-term immunity to some of those viruses, allowing them to get reinfected later. It is potentially bad news for us that immunity may not last that long against this virus.

Dr Rosalind Eggo: It does not look like SARS antibodies are cross-reactive with SARS-Cov-2. A new study has come out very recently, and it does not look like they are protective.

The Chair: We will have to examine in a different session the interrelationship between immune challenge and this virus's ability to defend itself against that challenge. Is it different?

Dr Rosalind Eggo: There are certainly a lot of questions about cross-reaction between all these coronaviruses and the history of infection by each of them, which definitely need to be resolved, and for our work on modelling to work out how and if those are relevant for protection and transmission it is extremely important, but it is really unknown at this time.

Q16 **Baroness Rock:** We have talked a bit already about our current understanding of the role of pre-symptomatic and asymptomatic carriers in the transmission of the virus. What is our understanding of the mechanisms of pre-symptomatic and asymptomatic transmission of viruses in general? Does the science community have experience of this from previous epidemics?

Professor John Edmunds: It is a very good question. We do not know a whole lot about it. Being symptomatic and coughing is probably a very useful mechanism for getting the virus out of your respiratory system and into somebody else's. It makes sense that symptomatic individuals may

be more dangerous to others and may be more likely to transmit to others, but the evidence for it is not that clear. It makes sense, but there is no clear evidence as to whether symptomatic individuals are more infectious than, let us say, pre-symptomatic individuals.

Of course, mostly when we are going about our lives, even with a cough, we are not coughing all the time. Most of the time we are just breathing more or less normally, so transmission can occur just from normal breath, normal exhalation and inhalation, by the susceptible individual. I do not think that you need to cough to transmit, but it probably helps.

Dr Rosalind Eggo: There is evidence on other activities like singing, and there has been a lot of transmission among choirs. It is definitely clear that there are other things you can do that will generate infectious virus.

Baroness Rock: Do you have anything to add, Dr Lycett?

Dr Samantha Lycett: Not especially, apart from to say that where the virus is coming from, whether it is deep within the lungs or the top of the lungs, might have an impact.

Dr Rosalind Eggo: I would add in answer to the second part of your question that there is also pre-symptomatic transmission for influenza. This is a reasonably common feature of respiratory infections. After you show symptoms, there might be behavioural changes in response to those, because you feel ill or you take precautions that might decrease transmission. If those occur, it will proportionately push more of the percentage of transmission on to that pre-symptomatic phase, even if the infectiousness is no different. It is fairly standard that there would be pre-symptomatic transmission for respiratory infections, although we did not see that for SARS-1. We are used to trying to incorporate that into our work to understand that. Generally, it makes control of these infections more difficult.

Q17 **Baroness Rock:** May I ask a supplementary question? I would be very keen to get an understanding of what we know about the role of children as carriers. We have touched on adults, but we have not really talked about children. Perhaps, Dr Eggo, you might touch on that one.

Dr Rosalind Eggo: There are three key questions about the role of children. The first is whether they get it if challenged and whether they are as susceptible as adults; secondly, if they get it, whether they are as likely to get ill; and, thirdly, if they get it, whether they are likely to transmit.

On the first question about the susceptibility of children, the evidence is quite mixed, but from a variety of studies there are starting to be hints and evidence that children are less likely to acquire infection than adults. As I said, this is information coming from a variety of different study types and there is by no means agreement, but the current thinking on the evidence seems to be that children are slightly less likely to acquire infection. There is very good evidence that if children are infected, they are less likely to have severe outcomes, so there are more asymptomatic

infections, more mild infections and a much lower risk of death in children.

The third point about the infectiousness in children, as raised previously, is that if children are more likely to be subclinically infected—asymptomatic or with very mild symptoms—it is very difficult to determine how infectious those infections are. A bit of evidence is starting to appear that there is a possibility, as Professor Edmunds noted, of lower infection from these groups, but this is very early in our understanding. We need more studies to pin this down, because it is so important. We think so far that children are less likely to get it, but it is not certain. We are very certain that children are less likely to have severe outcomes, and there are hints that children are less infectious, but again it is not certain.

Professor John Edmunds: It is unusual that children do not seem to play much of a role in transmission, because for most respiratory viruses and bacteria they play a central role in it. For this they do not seem to. The evidence for that is that there is only one documented outbreak associated with a school, which is amazing. You would normally expect most of the outbreaks to be associated with schools, yet in the global literature at the moment there is only one documented outbreak. That was in a secondary school in France with older children, so they were all aged 14 to 17 years of age. I have to say, of course, that schools have been closed in most countries from very early on in the outbreaks, so being able to determine outbreaks associated with schools will be difficult anyway, but it is still pretty remarkable that there has only been one outbreak associated with a school so far.

Q18 **Baroness Sheehan:** My question will take us back to where we started. It is about the extent to which we understand the role of different routes of transmission. We have spoken a bit about aerosol, surface contaminants and hand shaking. For the sake of completeness, could you also address the faecal/oral route, vertical transmission between mother and foetus, and food-borne transmission?

Professor John Edmunds: On the faecal/oral route, you can identify viral fragments in faeces, but that does not mean that it is necessarily viable virus. I do not know if anybody has actually managed to culture viable virus from a faecal sample; they may have, but I am are not aware of it. When you swallow, of course, you are swallowing down mucus from your naso- pharynx, so if you have been infected you will swallow virus all the time and eventually it passes out through faecal material, so it is not unusual to be able to pick up viral fragments, PCR, or find viral RNA in faecal material, but that does not mean to say that it is necessarily replicating in the digestive tract. It might do, however. Diarrhoea is associated with this spectrum of disease, so perhaps in individuals with diarrhoea you might expect the virus to be replicating in the digestive tract, but we do not know. That is the faecal/oral route. What were the other ones you were interested in?

Baroness Sheehan: Transmission between mother and foetus.

Professor John Edmunds: Again, there is very little evidence of transmission between mother and unborn child. There have been one or two isolated instances where that might have been the case, but very few, so it looks as if that is not a major route of transmission. You mentioned food handling, and I do not think that has been implicated in any major outbreaks. There have been cases associated with restaurants, weddings and events like that, but those are seemingly mostly transmission from individuals to individuals rather than food handlers, as far as I am aware.

Clearly it could happen. Again, if you do not practise good hand hygiene and you are symptomatic, it is of course possible that you can infect plates and forks and things like that, which others might pick up, so transmission could happen, but I have seen no strong evidence of that as a significant route of transmission.

Dr Rosalind Eggo: I saw a paper this morning about finding infectious virus in stool, but that is brand new and we need to keep an eye on that. Certainly we have not had a strong signal from the transmission or the literature so far that faecal/oral is a major route of transmission. We will keep an eye on that, but it really does look like respiratory is the main one. I do not know about vertical or food-borne transmission. I have not seen that.

Q19 **Baroness Sheehan:** I have a supplementary about what we understand about the role of the environment in transmission, eg indoor/outdoor, climate, meteorology. At the same time could you address a question I am very interested in: does it survive longer at room temperature or in the fridge?

Dr Rosalind Eggo: From what Professor Edmunds said earlier about respiratory transmission and droplets and aerosols, it seems as if confined areas would have higher transmission, and they do have higher transmission. That is indoor versus outdoor. It is very unclear how much of an effect that the environment has on transmission, because right now there are ongoing epidemics in hot parts of the world, cold parts of the world, and in damp and dry parts of the world.

The major effect on transmission right now is the fact that the reproduction number is above 1. If there are climate effects that affect transmission, we might start to see those only once this becomes a seasonal virus and where the effective reproduction number is around 1. Right now, it is dominated by the fact that this is extremely transmissible and immunity is at very, very low levels in the community and the population.

Dr Samantha Lycett: I would like to add something on the environmental aspect. As has just been mentioned, you would need to have a few seasons' worth of data to be able to see the effect of the environment, but as you also heard this morning there are a lot of whole genomes being sequenced, and in fact there is a method of using this genetic data together with the environmental data to see what the effect might be on transmission, but it is far too early to tell at the moment.

The Chair: Do you mean that environmental factors would be seen in epigenetic markers of the genome?

Dr Samantha Lycett: No, I mean that you can use the changes in the genes that are able to tell you how quickly viruses move around between places to correlate with what you expect the changes in environment to be.

Q20 **Baroness Young of Old Scone:** We had some evidence from Dr Eggo on children. This is probably a bit of a leading question, but based on what we know about the rate of infection in children and the transmission in children, should we be opening our primary schools?

Dr Rosalind Eggo: That is a difficult question that requires input from a lot of different scientists, both on modelling transmission and other factors. I believe there has been a lot of evidence feeding in to SPI-M, the modelling subgroup, and SAGE, the group of experts, to help to understand the evidence. I am not sure.

Professor John Edmunds: Clearly, the decision whether to open primary schools is a political one. It is not a scientific decision. The scientists can offer some advice. It looks like the risk to children is low and the vast majority do not have significant symptoms. As Dr Eggo mentioned earlier, they may be less likely to transmit to others as well, so the risk to others may be relatively low, but overall you have to weigh up those risks with other things: risks to the community, problems with children. Clearly, we cannot keep children off school for ever, but the actual decision and weighing of all those things needs to be done by politicians.

Baroness Young of Old Scone: At the moment, as a scientist, you would say that we could not tell whether children might pass this on to teachers?

Professor John Edmunds: We will know much more in the coming weeks. Other countries—Denmark, Germany, and many others—have opened their primary schools already, so if there are risks we will see them much more clearly now in the coming weeks. I have not looked carefully at those data yet myself, but I am sure others will be doing that.

The Chair: I should have told you all to declare your interests at the beginning. I forgot.

Q21 **Lord Winston:** Perhaps I should declare that I am a fellow of the Academy of Medical Sciences.

I want to come back to fomites for a moment. Again, it is a question of public interest. A lot of people are very worried about what they can and cannot touch—we have covered food, for example. There was a paper in the *New England Journal of Medicine* about a month ago that looked at different surfaces. Could you comment on whether there are some surfaces where the virus is likely to be prolonged in its infectivity?

Professor John Edmunds: Yes. There are certain surfaces where the virus can persist for longer—for example, metal surfaces. Typically, the virus persists less well with cloth surfaces and those sorts of things.

The Chair: What is longer?

Professor John Edmunds: At room temperature inside, in an enclosed environment, the virus can persist for 24 hours or more. You can detect virus in those sorts of settings. Some studies have looked at these. They tend to be in very controlled environments and so on, but the virus can persist for some considerable period on certain surfaces. Outside is a very different matter. Outside the virus can persist for minutes.

Lord Winston: That is really helpful, thank you.

Q22 **Viscount Ridley:** Quickly coming in on the outside versus inside point, this seems to be a very important distinction. It is not only that the virus on surfaces outside tend to last less long, but that ventilation outside tends to mean that droplets are not such an issue. Does that need to be got across more, particularly when we have seen the police telling people not to sunbathe in parks and things like that?

Professor John Edmunds: If you think of the three routes of transmission—fomites, droplets and aerosols—fomites are much less likely in the outside environment, partly because the virus cannot survive very long anyway and because you are less likely to touch things that other people have touched outside if you are sitting in or sunbathing in a park.

Viscount Ridley: They do not have doorknobs in parks, for example.

Professor John Edmunds: Exactly. In children's playgrounds you have surfaces that would be touched by many other individuals, so there is a potential risk there. But generally speaking, yes, I think the fomite transmission will be much reduced outside. Aerosol transmission is probably not the major route of transmission anyway, but aerosol transmission outside would be pretty unusual, I would have thought, because of course the aerosols would be diluted so massively by fresh air. I would have thought that would be a very minor route of transmission outside.

That leaves droplet transmission. That is where transmission occurs from larger droplets in face-to-face type contacts. If you keep a social distance of two metres or more, you reduce your chances of droplet transmission. There may be some instances outside—if you are downwind from somebody who is coughing, for instance—which might increase your risk compared with inside, just because the wind might take the droplets a bit more easily, but generally speaking if you keep outside a 2-metre radius of other individuals you should reduce your risk of droplet transmission as well.

The Chair: Is there a link between the R0 value and herd immunity?

Professor John Edmunds: Yes. Herd immunity is the level of immunity required in the population to prevent onward transmission. It does not

mean that you get no cases, but chains of transmission would dwindle rather than increase. That is the level of immunity you would require in the population. If you get a high enough level of immunity in the population, which ideally you would want to do via vaccine, you can prevent these chains of transmission occurring.

There is a simple formula that you can work out of 1-1/R0. The higher the reproduction number, the larger the fraction of the population you need to immunise to prevent transmission in the community. With something like measles, which has a very high reproduction number of 15 to 20, somewhere in the region of 95% of the population need to be immune to stop outbreaks of measles from occurring. With this virus, the level of immunity required in the population to stop transmission so that chains of transmission would dwindle away rather than increase is in the region of 65% to 70%.

The Chair: But if 30% of the population infected are asymptomatic, will that number keep increasing?

Professor John Edmunds: We need to know more about immunity. We do not know yet whether infection leads to long-term immunity, or how long immunity might last. It seems likely that the individuals who have been recently infected would be immune, but we do not know that with great certainty. It is not really to do with asymptomatics, because you would also expect that asymptomatics, once they get over their infection, are immune to some extent. First, it would be a function of the level of vaccination in the community, but given that we do not have a vaccine yet, we have to leave that one out, so the level of immunity in a population would be related to how much infection has happened whether those infections were symptomatic or asymptomatic.

Lord Winston: A surprising point that has come up very recently is that in many African countries, where there is very poor health and a lot of infectious disease, the transmission of this virus seems to be a good deal less. Certainly, the infection rates are a lot lower. Would you like to comment on that, or is that outside your field of expertise?

Dr Rosalind Eggo: There are a lot of unanswered questions about the epidemic that is happening in Africa. There is certainly evidence of transmission, but a lot of countries have had lockdowns or similar types of intervention policies in place since quite early in the epidemic. There is evidence of ongoing transmission. In South Africa, for instance, there is now evidence of an increase in cases. It remains an area of study for us and for scientists in those countries to understand the epidemic dynamics and what is happening there.

The Chair: May I thank all three of you very much for helping us? Both sessions today have been most interesting. We are very grateful to you. Of course, there will be other sessions, because the pandemic is continuing. Thank you for today.